Aeterna Zentaris Inc.
Form 20-F
March 31, 2011

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

FORM 20-F

o **Registration Statement Pursuant to Section 12(b) or 12(g) of The Securities Exchange Act of 1934**

OR

ý **Annual Report Pursuant to Section 13 or 15(d) of The Securities Exchange Act of 1934 for the fiscal year ended December 31, 2010**

OR

o **Transition Report Pursuant to Section 13 or 15(d) of The Securities Exchange Act of 1934**

OR

o **Shell Company Report Pursuant to Section 13 or 15(d) of The Securities Exchange Act of 1934**

Commission file number 0-30752

AETERNA ZENTARIS INC.

(Exact Name of Registrant as Specified in its Charter)

Not Applicable
(Translation of Registrant's Name into English)

Canada
(Jurisdiction of Incorporation)

1405 du Parc-Technologique Blvd.
Quebec City, Quebec
Canada, G1P 4P5
(Address of Principal Executive Offices)

Dennis Turpin
Telephone: 418-652-8525
E-mail: dturpin@aezsinc.com
1405 du Parc-Technologique Blvd.

Quebec City, Quebec
Canada, G1P 4P5
(Name, Telephone, E-mail and Address of Company Contact Person)

Securities registered or to be registered pursuant to Section 12(b) of the Act:

Title of Each Class	Name of Each Exchange on Which Registered
Common Shares	Nasdaq Global Market Toronto Stock Exchange

Securities registered or to be registered pursuant to Section 12(g) of the Act: **NONE**

Securities for which there is a reporting obligation pursuant to Section 15(d) of the ACT: **NONE**

Indicate the number of outstanding shares of each of the issuer's classes of capital or common stock as at the close of the period covered by the annual report: 83,429,914 common shares as at December 31, 2010.

Indicate by check mark whether the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act.

Yes o No ý

If this report is an annual or transition report, indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or 15(d) of the Securities Exchange Act of 1934.

Yes o No ý

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 ý No o

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files).

Yes o No ý

Indicate by check mark whether the registrant is a large accelerated filer, an accelerated filer or, or a non-accelerated filer. See definitions of "accelerated filer" and "large accelerated filer" in Rule 12b-2 of the Exchange Act. (Check one):

Large accelerated filer o Accelerated filer ý Non-accelerated filer o

Indicate by check mark which basis of accounting the registrant has used to prepare the financial statements included in this filing:

US GAAP o International Financial Reporting Standards as issued by the International Accounting Standards Board o Other ý

If "other" has been checked in response to the previous question, indicate by check mark which financial statement item the registrant has elected to follow.

Item 17 o Item 18 ý

If this is an annual report, indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Exchange Act).

Yes o No ý

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Basis of Presentation

General

Except where the context otherwise requires, all references in this annual report on Form 20-F ("Form 20-F") to the "Company", "Aeterna Zentaris Inc.", "we", "us", "our" or similar words or phrases are to Aeterna Zentaris Inc. and its subsidiaries, taken together. In this annual report, references to "$" and "US$" are to United States dollars and references to "CAN$" are to Canadian dollars. Unless otherwise indicated, the statistical and financial data contained in this annual report are presented as at December 31, 2010.

Forward-Looking Statements

This annual report contains forward-looking statements made pursuant to the safe harbor provisions of the U.S. Securities Litigation Reform Act of 1995. Forward-looking statements involve known and unknown risks and uncertainties, which could cause the Company's actual results to differ materially from those in the forward-looking statements. Such risks and uncertainties include, among others, the availability of funds and resources to pursue R&D projects, the successful and timely completion of clinical studies, the ability of the Company to take advantage of business opportunities in the pharmaceutical industry, uncertainties related to the regulatory process and general changes in economic conditions. Investors should consult the Company's quarterly and annual filings with the Canadian and U.S. securities commissions for additional information on risks and uncertainties relating to the forward-looking statements. Investors are cautioned not to rely on these forward-looking statements. The Company does not undertake to update these forward-looking statements and we disclaim any obligation to update any such factors or to publicly announce the result of any revisions to any of the forward-looking statements contained herein to reflect future results, events or developments except if we are required to do so by a governmental authority or applicable law.

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GENERAL INFORMATION

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PART I

Item 1. Identity of Directors, Senior Management and Advisers

A. Directors and senior management

Not applicable.

B. Advisors

Not applicable.

C. Auditors

Not applicable.

Item 2. Offer Statistics and Expected Timetable

A. Offer statistics

Not applicable.

B. Method and expected timetable

Not applicable.

Item 3. Key Information

A. Selected financial data

The consolidated statement of operations data set forth in this Item 3.A with respect to the years ended December 31, 2010, 2009 and 2008, and the consolidated balance sheet data as at December 31, 2010 and 2009, have been derived from the audited consolidated financial statements listed in Item 18, which have been prepared in accordance with Canadian generally accepted accounting principles ("Canadian GAAP"), except as otherwise described therein. The consolidated statement of operations data set forth in this Item 3.A with respect to the years ended December 31, 2007 and 2006, and the consolidated balance sheet data as at December 31, 2008, 2007 and 2006, have been derived from other consolidated financial statements not included herein and have been prepared in accordance with Canadian GAAP, except as otherwise described therein. The selected financial data should be read in conjunction with our audited consolidated financial statements and the related notes included elsewhere in this annual report, and "Item 5. Operating and Financial Review and Prospects" of this annual report.

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Consolidated Statements of Operations Data

(in thousands of US dollars, except share and per share data)

Canadian GAAP

	Years Ended December 31,				
	2010	**2009**	**2008**	**2007**	**2006**
	$	**$**	**$**	**$**	**$**
Revenues	27,703	63,237	38,478	42,068	38,799
Operating expenses					
Cost of sales, excluding depreciation and amortization	18,700	16,501	19,278	12,930	11,270
Research and development costs	20,546	44,217	57,448	39,248	27,422
Research and development tax credits and grants	(687)	(403)	(343)	(2,060)	(1,564)
Selling, general and administrative expenses	11,875	16,040	17,325	20,403	16,478
Depreciation and amortization					
Property, plant and equipment	1,005	3,285	1,515	1,562	2,816
Intangible assets	1,492	7,555	5,639	4,004	6,148
Impairment of long-lived assets held for sale				735	
	52,931	87,195	100,862	76,822	62,570
Loss from operations	(25,228)	(23,958)	(62,384)	(34,754)	(23,771)
Other income (expenses)					
Unrealized gain on held-for-trading financial instrument	687				
Interest income	207	349	868	1,904	1,441
Interest expense					
Long-term debt and convertible term loans				(85)	(1,270)
Other	(26)	(5)	(118)		(163)
Foreign exchange gain (loss)	1,170	(1,110)	3,071	(1,035)	319
Loss on disposal of long-lived assets held for sale			(35)		
Loss on disposal of equipment	(28)		(44)	(28)	
Gain on disposal of long-term investment					409
	2,010	(766)	3,742	756	736
Share in the results of an affiliated company					1,575
Loss before income taxes from continuing operations	(23,218)	(24,724)	(58,642)	(33,998)	(21,460)
Income tax (expense) recovery			(1,175)	1,961	29,037
Net (loss) earnings from continuing operations	(23,218)	(24,724)	(59,817)	(32,037)	7,577
Net (loss) earnings from discontinued operations				(259)	25,813
Net (loss) earnings for the year	(23,218)	(24,724)	(59,817)	(32,296)	33,390
Net (loss) earnings per share from continuing operations					
Basic	(0.31)	(0.43)	(1.12)	(0.61)	0.14
Diluted	(0.31)	(0.43)	(1.12)	(0.61)	0.14
Net earnings per share from discontinued operations					
Basic					0.50
Diluted					0.48
Net (loss) earnings per share					

Basic	(0.31)	(0.43)	(1.12)	(0.61)	0.64
Diluted	(0.31)	(0.43)	(1.12)	(0.61)	0.62
Weighted average number of shares					
Basic	75,659,410	56,864,484	53,187,470	53,182,803	52,099,290
Diluted	75,659,410	56,864,484	53,187,470	53,182,803	52,549,260

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US GAAP

	Years Ended December 31,				
	2010	**2009**	**2008**	**2007**	**2006**
	$	**$**	**$**	**$**	**$**
Net (loss) earnings for the year	(29,165)	(16,794)	(56,070)	(37,428)	34,262
Of which:					
Net (loss) earnings from continuing operations	(29,165)	(16,794)	(56,070)	(36,415)	8,449
Net (loss) earnings from discontinued operations				(1,013)	25,813
Net (loss) earnings per share from continuing operations					
Basic	(0.39)	(0.30)	(1.05)	(0.68)	0.16
Diluted	(0.39)	(0.30)	(1.05)	(0.68)	0.16
Net (loss) earnings per share from discontinued operations					
Basic				(0.02)	0.50
Diluted				(0.02)	0.49
Net (loss) earnings per share					
Basic	(0.39)	(0.30)	(1.05)	(0.70)	0.66
Diluted	(0.39)	(0.30)	(1.05)	(0.70)	0.65
Weighted average number of shares					
Basic	75,659,410	56,864,484	53,187,470	53,182,803	52,099,290
Diluted	75,659,410 3	56,864,484	53,187,470	53,182,803	52,549,260

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Consolidated Balance Sheet Data

(in thousands of US dollars)

Canadian GAAP

	As at December 31,				
	2010	**2009**	**2008**	**2007**	**2006**
	$	**$**	**$**	**$**	**$**
Cash and cash equivalents	31,998	38,100	49,226	10,272	8,939
Short-term investments	1,934		493	31,115	51,550
Working capital	30,688	29,745	39,554	37,325	85,413
Restricted cash	827	878			
Total assets	76,574	86,262	108,342	123,363	223,491
Long-term debt and payable	90	143	172		687
Share capital	60,149	41,203	30,566	30,566	168,466
Shareholders' equity	12,439	9,226	21,475	88,591	178,879

US GAAP

	As at December 31,				
	2010	**2009**	**2008**	**2007**	**2006**
	$	**$**	**$**	**$**	**$**
Cash and cash equivalents	31,998	38,100	49,226	10,272	8,939
Short-term investments	1,934		493	31,115	51,550
Working capital	29,733	29,745	39,554	37,325	85,413
Restricted cash	827	878			
Total assets	74,853	84,116	100,001	109,182	209,143
Warrant liability, short-term	955				
Warrant liability, long-term	13,412	1,351			
Long-term debt and payable	90	143	172		687
Share capital	52,318	33,226	22,589	22,589	160,489
Shareholders' (deficiency) equity	(3,649)	5,729	13,134	74,410	169,704

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B. Capitalization and indebtedness

Not applicable.

C. Reasons for the offer and use of proceeds

Not applicable.

D. Risk factors

Risks Relating to Us and Our Business

Investments in biopharmaceutical companies are generally considered to be speculative.

The prospects for companies operating in the biopharmaceutical industry may generally be considered to be uncertain, given the very nature of the industry and, accordingly, investments in biopharmaceutical companies should be considered to be speculative.

We have a history of operating losses and we may never achieve or maintain operating profitability.

Our product candidates remain at the development stage, and we have incurred substantial expenses in our efforts to develop products. Consequently, we have incurred recurrent operating losses and, as disclosed in our audited consolidated financial statements as at December 31, 2010 and December 31, 2009 and for the years ended December 31, 2010, 2009 and 2008, we had an accumulated deficit of $150.8 million as at December 31, 2010. Our operating losses have adversely impacted, and will continue to adversely impact, our working capital, total assets and shareholders' equity. We do not expect to reach operating profitability in the immediate future, and our expenses are likely to increase as we continue to expand our research and development ("R&D") and clinical study programs and our sales and marketing activities and seek regulatory approval for our product candidates. Even if we succeed in developing new commercial products, we expect to incur additional operating losses for at least the next several years. If we do not ultimately generate sufficient revenue from commercialized products and achieve or maintain operating profitability, an investment in our securities could result in a significant or total loss.

Our clinical trials may not yield results which will enable us to obtain regulatory approval for our products, and a setback in any of our clinical trials would likely cause a drop in the price of our securities.

We will only receive regulatory approval for a product candidate if we can demonstrate in carefully designed and conducted clinical trials that the product candidate is both safe and effective. We do not know whether our pending or any future clinical trials will demonstrate sufficient safety and efficacy to obtain the requisite regulatory approvals or will result in marketable products. Unfavorable data from those studies could result in the withdrawal of marketing approval for approved products or an extension of the review period for developmental products. Clinical trials are inherently lengthy, complex, expensive and uncertain processes and have a high risk of failure. It typically takes many years to complete testing, and failure can occur at any stage of testing. Results attained in pre-clinical testing and early clinical studies, or trials, may not be indicative of results that are obtained in later studies.

None of our product candidates has to date received regulatory approval for its intended commercial sale. We cannot market a pharmaceutical product in any jurisdiction until it has completed rigorous pre-clinical testing and clinical trials and passed such jurisdiction's extensive regulatory approval process. In general, significant research and development and clinical studies are required to demonstrate the safety and efficacy of our product candidates before we can submit regulatory applications. Pre-clinical testing and clinical development are long, expensive and uncertain processes. Preparing, submitting and advancing applications for regulatory approval is complex, expensive and time-consuming and entails significant uncertainty. Data obtained from pre-clinical and clinical tests can be interpreted in different ways, which could delay, limit or prevent regulatory approval. It may take us

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many years to complete the testing of our product candidates and failure can occur at any stage of this process. In addition, we have limited experience in conducting and managing the clinical trials necessary to obtain regulatory approval in the United States, in Canada and abroad and, accordingly, may encounter unforeseen problems and delays in the approval process. Though we may engage a clinical research organization with experience in conducting regulatory trials, errors in the conduct, monitoring and/or auditing could invalidate the results from a regulatory perspective. Even if a product candidate is approved by the United States Food and Drug Administration ("FDA"), the Canadian Therapeutic Products Directorate or any other regulatory authority, we may not obtain approval for an indication whose market is large enough to recoup our investment in that product candidate. In addition, there can be no assurance that we will ever obtain all or any required regulatory approvals for any of our product candidates.

We are currently developing our product candidates based on R&D activities, pre-clinical testing and clinical trials conducted to date, and we may not be successful in developing or introducing to the market these or any other new products or technology. If we fail to develop and deploy new products successfully and on a timely basis, we may become non-competitive and unable to recoup the R&D and other expenses we incur to develop and test new products.

Interim results of pre-clinical or clinical studies do not necessarily predict their final results, and acceptable results in early studies might not be obtained in later studies. Safety signals detected during clinical studies and pre-clinical animal studies may require us to do additional studies, which could delay the development of the drug or lead to a decision to discontinue development of the drug. Product candidates in the later stages of clinical development may fail to show the desired safety and efficacy traits despite positive results in initial clinical testing. Results from earlier studies may not be indicative of results from future clinical trials and the risk remains that a pivotal program may generate efficacy data that will be insufficient for the approval of the drug, or may raise safety concerns that may prevent approval of the drug. Interpretation of the prior pre-clinical and clinical safety and efficacy data of our product candidates may be flawed and there can be no assurance that safety and/or efficacy concerns from the prior data were overlooked or misinterpreted, which in subsequent, larger studies appear and prevent approval of such product candidates.

Furthermore, we may suffer significant setbacks in advanced clinical trials, even after promising results in earlier studies. Based on results at any stage of clinical trials, we may decide to repeat or redesign a trial or discontinue development of one or more of our product candidates. Further, actual results may vary once the final and quality-controlled verification of data and analyses has been completed. If we fail to adequately demonstrate the safety and efficacy of our products under development, we will not be able to obtain the required regulatory approvals to commercialize our product candidates.

Clinical trials are subject to continuing oversight by governmental regulatory authorities and institutional review boards and:

must meet the requirements of these authorities;

must meet requirements for informed consent; and

must meet requirements for good clinical practices.

We may not be able to comply with these requirements in respect of one or more of our product candidates.

In addition, we rely on third parties, including Contract Research Organizations ("CROs") and outside consultants, to assist us in managing and monitoring clinical trials. Our reliance on these third parties may result in delays in completing, or in failing to complete, these trials if one or more third parties fails to perform with the speed and level of competence we expect.

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A failure in the development of any one of our programs or product candidates could have a negative impact on the development of the others. Setbacks in any phase of the clinical development of our product candidates would have an adverse financial impact (including with respect to any agreements and partnerships that may exist between us and other entities), could jeopardize regulatory approval and would likely cause a drop in the price of our securities.

If we are unable to successfully complete our clinical trial programs, or if such clinical trials take longer to complete than we project, our ability to execute our current business strategy will be adversely affected.

Whether or not and how quickly we complete clinical trials is dependent in part upon the rate at which we are able to engage clinical trial sites and, thereafter, the rate of enrollment of patients, and the rate at which we collect, clean, lock and analyze the clinical trial database. Patient enrollment is a function of many factors, including the design of the protocol, the size of the patient population, the proximity of patients to and availability of clinical sites, the eligibility criteria for the study, the perceived risks and benefits of the drug under study and of the control drug, if any, the efforts to facilitate timely enrollment in clinical trials, the patient referral practices of physicians, the existence of competitive clinical trials, and whether existing or new drugs are approved for the indication we are studying. Certain clinical trials are designed to continue until a pre-determined number of events have occurred to the patients enrolled. Trials such as this are subject to delays stemming from patient withdrawal and from lower than expected event rates and may also incur increased costs if enrollment is increased in order to achieve the desired number of events. If we experience delays in identifying and contracting with sites and/or in patient enrollment in our clinical trial programs, we may incur additional costs and delays in our development programs, and may not be able to complete our clinical trials on a cost-effective or timely basis. In addition, conducting multi-national studies adds another level of complexity and risk as we are subject to events affecting countries outside Canada. Moreover, negative or inconclusive results from the clinical trials we conduct or adverse medical events could cause us to have to repeat or terminate the clinical trials. Accordingly, we may not be able to complete the clinical trials within an acceptable time frame, if at all. If we or any third party have difficulty enrolling a sufficient number of patients to conduct our clinical trials as planned, we may need to delay or terminate ongoing clinical trials.

Additionally, we have never filed a new drug application ("NDA"), or similar application for approval in the United States or in any country for our current product candidates, which may result in a delay in, or the rejection of, our filing of an NDA or similar application. During the drug development process, regulatory agencies will typically ask questions of drug sponsors. While we endeavor to answer all such questions in a timely fashion, or in the NDA filing, some questions may not be answered by the time we file our NDA. Unless the FDA waives the requirement to answer any such unanswered questions, submission of an NDA may be delayed or rejected.

We are and will be subject to stringent ongoing government regulation for our products and our product candidates, even if we obtain regulatory approvals for the latter.

The manufacture, marketing and sale of our products and product candidates are and will be subject to strict and ongoing regulation, even if regulatory authorities approve any of the latter. Compliance with such regulation will be expensive and consume substantial financial and management resources. For example, an approval for a product may be conditioned on our agreement to conduct costly post-marketing follow-up studies to monitor the safety or efficacy of the products. In addition, as a clinical experience with a drug expands after approval because the drug is used by a greater number and more diverse group of patients than during clinical trials, side effects or other problems may be observed after approval that were not observed or anticipated during pre-approval clinical trials. In such a case, a regulatory authority could restrict the indications for which the product may be sold or revoke the product's regulatory approval.

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We and our contract manufacturers are and will be required to comply with applicable current Good Manufacturing Practice ("cGMP") regulations for the manufacture of our products. These regulations include requirements relating to quality assurance, as well as the corresponding maintenance of rigorous records and documentation. Manufacturing facilities must be approved before we can use them in the commercial manufacturing of our products and are subject to subsequent periodic inspection by regulatory authorities. In addition, material changes in the methods of manufacturing or changes in the suppliers of raw materials are subject to further regulatory review and approval.

If we, or any future marketing collaborators or contract manufacturers, fail to comply with applicable regulatory requirements, we may be subject to sanctions including fines, product recalls or seizures and related publicity requirements, injunctions, total or partial suspension of production, civil penalties, suspension or withdrawals of previously granted regulatory approvals, warning or untitled letters, refusal to approve pending applications for marketing approval of new products or of supplements to approved applications, import or export bans or restrictions, and criminal prosecution and penalties. Any of these penalties could delay or prevent the promotion, marketing or sale of our products and product candidates.

If our products do not gain market acceptance, we may be unable to generate significant revenues.

Even if our products are approved for commercialization, they may not be successful in the marketplace. Market acceptance of any of our products will depend on a number of factors including, but not limited to:

- demonstration of clinical efficacy and safety;
- the prevalence and severity of any adverse side effects;
- limitations or warnings contained in the product's approved labeling;
- availability of alternative treatments for the indications we target;
- the advantages and disadvantages of our products relative to current or alternative treatments;
- the availability of acceptable pricing and adequate third-party reimbursement; and
- the effectiveness of marketing and distribution methods for the products.

If our products do not gain market acceptance among physicians, patients, healthcare payers and others in the medical community, which may not accept or utilize our products, our ability to generate significant revenues from our products would be limited and our financial conditions will be materially adversely affected. In addition, if we fail to further penetrate our core markets and existing geographic markets or successfully expand our business into new markets, the growth in sales of our products, along with our operating results, could be negatively impacted.

Our ability to further penetrate our core markets and existing geographic markets in which we compete or to successfully expand our business into additional countries in Europe, Asia or elsewhere is subject to numerous factors, many of which are beyond our control. Our products, if successfully developed, may compete with a number of drugs and therapies currently manufactured and marketed by major pharmaceutical and other biotechnology companies. Our products may also compete with new products currently under development by others or with products which may be less expensive than our products. We cannot assure you that our efforts to increase market penetration in our core markets and existing geographic markets will be successful. Our failure to do so could have an adverse effect on our operating results and would likely cause a drop in the price of our securities.

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We may require significant additional financing, and we may not have access to sufficient capital.

We may require additional capital to pursue planned clinical trials, regulatory approvals, as well as further R&D and marketing efforts for our product candidates and potential products. Except as otherwise described in this annual report, we do not anticipate generating significant revenues from operations in the near future and we currently have no committed sources of capital.

We may attempt to raise additional funds through public or private financings, collaborations with other pharmaceutical companies or financing from other sources. Additional funding may not be available on terms which are acceptable to us. If adequate funding is not available to us on reasonable terms, we may need to delay, reduce or eliminate one or more of our product development programs or obtain funds on terms less favorable than we would otherwise accept. To the extent that additional capital is raised through the sale of equity securities or securities convertible into or exchangeable for equity securities, the issuance of those securities could result in dilution to our shareholders. Moreover, the incurrence of debt financing could result in a substantial portion of our future operating cash flow, if any, being dedicated to the payment of principal and interest on such indebtedness and could impose restrictions on our operations. This could render us more vulnerable to competitive pressures and economic downturns.

We anticipate that our existing working capital, including the proceeds from any sale and anticipated revenues, will be sufficient to fund our development programs, clinical trials and other operating expenses for the near future. However, our future capital requirements are substantial and may increase beyond our current expectations depending on many factors including:

the duration and results of our clinical trials for our various product candidates going forward;

unexpected delays or developments in seeking regulatory approvals;

the time and cost involved in preparing, filing, prosecuting, maintaining and enforcing patent claims;

other unexpected developments encountered in implementing our business development and commercialization strategies;

the outcome of litigation, if any; and

further arrangements, if any, with collaborators.

In addition, global economic and market conditions as well as future developments in the credit and capital markets may make it even more difficult for us to raise additional financing in the future.

A substantial portion of our future revenues may be dependent upon our agreements with Keryx Biopharmaceuticals, Inc. and Yakult Honsha Co. Ltd

We currently expect that a substantial portion of our future revenues may be dependent upon our strategic partnerships with Keryx Biopharmaceuticals, Inc. ("Keryx") for North America and Yakult Honsha Co. Ltd ("Yakult") for Japan. Under these strategic partnerships, Keryx and Yakult have significant development and commercialization responsibilities with respect to the development and sale of perifosine. If Keryx or Yakult were to terminate their agreements with us, fail to meet their obligations or otherwise decrease their level of efforts, allocation of resources or other commitments under their respective agreements, our future revenues and/or prospects could be negatively impacted and the development and commercialization of perifosine would be interrupted. In addition, if Keryx or Yakult do not achieve some or any of their respective development, regulatory and commercial milestones or if they do not achieve certain net sales thresholds as set forth in the agreements, we will not fully realize the expected economic benefits of these agreements. Further, the achievement of certain of the milestones under these strategic partnership agreements will depend on factors that are outside of our control and most are not expected to be achieved for several years, if at all. Any failure

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to successfully maintain our strategic partnership agreements could materially and adversely affect our ability to generate revenues.

If we are unsuccessful in increasing our revenues and/or raising additional funding, we may possibly cease to continue operating as we currently do.

Although our audited consolidated financial statements as at December 31, 2010 and December 31, 2009 and for the years ended December 31, 2010, 2009 and 2008 have been prepared on a going concern basis, which contemplates the realization of assets and liquidation of liabilities during the normal course of operations, our ability to continue as a going concern is dependent on the successful execution of our business plan, which will require an increase in revenue and/or additional funding to be provided by potential investors as well as non-traditional sources of financing. Although we stated in our audited consolidated financial statements as at December 31, 2010 and December 31, 2009 and for years ended December 31, 2010, 2009 and 2008 that management believed that the Company had, as at December 31, 2010, sufficient financial resources to fund planned expenditures and other working capital needs for at least, but not limited to, the 12-month period following such date, there can be no assurance that management will be able to reiterate such belief in our future financial statements.

We have had sustained losses, accumulated deficits and negative cash flows from operations since our inception. We expect that this will continue throughout 2011.

Additional funding may be in the form of debt or equity or a hybrid instrument depending on the needs of the investor. In light of present and future global economic and credit market conditions, we may not be able to raise additional cash resources through these traditional sources of financing. Although we are also pursuing non-traditional sources of financing, the global credit market crisis has also adversely affected the ability of potential parties to pursue such transactions. We do not believe that the ability to access capital markets or these adverse conditions are likely to improve significantly in the near future. Accordingly, as a result of the foregoing, we continue to review traditional sources of financing, such as private and public debt or various equity financing alternatives, as well as other alternatives to enhance shareholder value including, but not limited to, non-traditional sources of financing, such as alliances with strategic partners, the sale of assets or licensing of our technology or intellectual property, a combination of operating and related initiatives or a substantial reorganization of our business. If we do not raise additional capital, we do not expect our operations to generate sufficient cash flow to fund our obligations as they come due.

There can be no assurance that we will achieve profitability or positive cash flows or be able to obtain additional funding or that, if obtained, they will be sufficient, or whether any other initiatives will be successful, such that we may continue as a going concern. There are material uncertainties related to certain adverse conditions and events that could cast significant doubt on our ability to remain a going concern.

We may not achieve our projected development goals in the time-frames we announce and expect.

We set goals and make public statements regarding the timing of the accomplishment of objectives material to our success, such as the commencement, enrollment and completion of clinical trials, anticipated regulatory submission and approval dates and time of product launch. The actual timing of these events can vary dramatically due to factors such as delays or failures in our clinical trials, the uncertainties inherent in the regulatory approval process and delays in achieving manufacturing or marketing arrangements sufficient to commercialize our products. There can be no assurance that our clinical trials will be completed, that we will make regulatory submissions or receive regulatory approvals as planned or that we will be able to adhere to our current schedule for the launch of any of our products. If we fail to achieve one or more of these milestones as planned, the price of our securities would likely decline.

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If we fail to obtain acceptable prices or adequate reimbursement for our products, our ability to generate revenues will be diminished.

The ability for us and/or our partners to successfully commercialize our products will depend significantly on our ability to obtain acceptable prices and the availability of reimbursement to the patient from third-party payers, such as governmental and private insurance plans. These third-party payers frequently require companies to provide predetermined discounts from list prices, and they are increasingly challenging the prices charged for pharmaceuticals and other medical products. Our products may not be considered cost-effective, and reimbursement to the patient may not be available or sufficient to allow us or our partners to sell our products on a competitive basis. It may not be possible to negotiate favorable reimbursement rates for our products.

In addition, the continuing efforts of third-party payers to contain or reduce the costs of healthcare through various means may limit our commercial opportunity and reduce any associated revenue and profits. We expect proposals to implement similar government control to continue. In addition, increasing emphasis on managed care will continue to put pressure on the pricing of pharmaceutical and biopharmaceutical products. Cost control initiatives could decrease the price that we or any current or potential collaborators could receive for any of our products and could adversely affect our profitability. In addition, in the United States, in Canada and in many other countries, pricing and/or profitability of some or all prescription pharmaceuticals and biopharmaceuticals are subject to government control.

If we fail to obtain acceptable prices or an adequate level of reimbursement for our products, the sales of our products would be adversely affected or there may be no commercially viable market for our products.

Competition in our targeted markets is intense, and development by other companies could render our products or technologies non-competitive.

The biomedical field is highly competitive. New products developed by other companies in the industry could render our products or technologies non-competitive. Competitors are developing and testing products and technologies that would compete with the products that we are developing. Some of these products may be more effective or have an entirely different approach or means of accomplishing the desired effect than our products. We expect competition from biopharmaceutical and pharmaceutical companies and academic research institutions to increase over time. Many of our competitors and potential competitors have substantially greater product development capabilities and financial, scientific, marketing and human resources than we do. Our competitors may succeed in developing products earlier and in obtaining regulatory approvals and patent protection for such products more rapidly than we can or at a lower price.

We may not obtain adequate protection for our products through our intellectual property.

We rely heavily on our proprietary information in developing and manufacturing our product candidates. Our success depends, in large part, on our ability to protect our competitive position through patents, trade secrets, trademarks and other intellectual property rights. The patent positions of pharmaceutical and biopharmaceutical firms, including Aeterna Zentaris, are uncertain and involve complex questions of law and fact for which important legal issues remain unresolved. Applications for patents and trademarks in Canada, the United States and in other foreign territories have been filed and are being actively pursued by us. Pending patent applications may not result in the issuance of patents and we may not be able to obtain additional issued patents relating to our technology or products. Even if issued, patents to us or our licensors may be challenged, narrowed, invalidated, held to be unenforceable or circumvented, which could limit our ability to stop competitors from marketing similar products or limit the length of term of patent protection we may have for our products. Changes in either patent laws or in interpretations of patent laws in the United States and other

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countries may diminish the value of our intellectual property or narrow the scope of our patent protection. The patents issued or to be issued to us may not provide us with any competitive advantage or protect us against competitors with similar technology. In addition, it is possible that third parties with products that are very similar to ours will circumvent our patents by means of alternate designs or processes. We may have to rely on method of use and new formulation protection for our compounds in development, and any resulting products, which may not confer the same protection as claims to compounds per se.

In addition, our patents may be challenged by third parties in patent litigation, which is becoming widespread in the biopharmaceutical industry. There may be prior art of which we are not aware that may affect the validity or enforceability of a patent claim. There also may be prior art of which we are aware, but which we do not believe affects the validity or enforceability of a claim, which may, nonetheless, ultimately be found to affect the validity or enforceability of a claim. No assurance can be given that our patents would, if challenged, be held by a court to be valid or enforceable or that a competitor's technology or product would be found by a court to infringe our patents. Our granted patents could also be challenged and revoked in opposition or nullity proceedings in certain countries outside the United States. In addition, we may be required to disclaim part of the term of certain patents.

Patent applications relating to or affecting our business have been filed by a number of pharmaceutical and biopharmaceutical companies and academic institutions. A number of the technologies in these applications or patents may conflict with our technologies, patents or patent applications, and any such conflict could reduce the scope of patent protection which we could otherwise obtain. Because patent applications in the United States and many other jurisdictions are typically not published until eighteen months after their first effective filing date, or in some cases not at all, and because publications of discoveries in the scientific literature often lag behind actual discoveries, neither we nor our licensors can be certain that we or they were the first to make the inventions claimed in our or their issued patents or pending patent applications, or that we or they were the first to file for protection of the inventions set forth in these patent applications. If a third party has also filed a patent application in the United States covering our product candidates or a similar invention, we may have to participate in an adversarial proceeding, known as an interference, declared by the United States Patent and Trademark Office to determine priority of invention in the United States. The costs of these proceedings could be substantial and it is possible that our efforts could be unsuccessful, resulting in a loss of our U.S. patent position.

In addition to patents, we rely on trade secrets and proprietary know-how to protect our intellectual property. If we are unable to protect the confidentiality of our proprietary information and know-how, the value of our technology and products could be adversely affected. We seek to protect our unpatented proprietary information in part by requiring our employees, consultants, outside scientific collaborators and sponsored researchers and other advisors to enter into confidentiality agreements. These agreements provide that all confidential information developed or made known to the individual during the course of the individual's relationship with us is to be kept confidential and not disclosed to third parties except in specific circumstances. In the case of our employees, the agreements provide that all of the technology which is conceived by the individual during the course of employment is our exclusive property. These agreements may not provide meaningful protection or adequate remedies in the event of unauthorized use or disclosure of our proprietary information. In addition, it is possible that third parties could independently develop proprietary information and techniques substantially similar to ours or otherwise gain access to our trade secrets. If we are unable to protect the confidentiality of our proprietary information and know-how, competitors may be able to use this information to develop products that compete with our products and technologies, which could adversely impact our business.

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We currently have the right to use certain technology under license agreements with third parties. Our failure to comply with the requirements of material license agreements could result in the termination of such agreements, which could cause us to terminate the related development program and cause a complete loss of our investment in that program.

As a result of the foregoing factors, we may not be able to rely on our intellectual property to protect our products in the marketplace.

We may infringe the intellectual property rights of others.

Our commercial success depends significantly on our ability to operate without infringing the patents and other intellectual property rights of third parties. There could be issued patents of which we are not aware that our products or methods may be found to infringe, or patents of which we are aware and believe we do not infringe but which we may ultimately be found to infringe. Moreover, patent applications and their underlying discoveries are in some cases maintained in secrecy until patents are issued. Because patents can take many years to issue, there may be currently pending applications of which we are unaware that may later result in issued patents that our products or methods are found to infringe. Moreover, there may be published pending applications that do not currently include a claim covering our products or methods but which nonetheless provide support for a later drafted claim that, if issued, our products or methods could be found to infringe.

If we infringe or are alleged to infringe intellectual property rights of third parties, it will adversely affect our business. Our research, development and commercialization activities, as well as any product candidates or products resulting from these activities, may infringe or be accused of infringing one or more claims of an issued patent or may fall within the scope of one or more claims in a published patent application that may subsequently issue and to which we do not hold a license or other rights. Third parties may own or control these patents or patent applications in the United States and abroad. These third parties could bring claims against us or our collaborators that would cause us to incur substantial expenses and, if successful against us, could cause us to pay substantial damages. Further, if a patent infringement suit were brought against us or our collaborators, we or they could be forced to stop or delay research, development, manufacturing or sales of the product or product candidate that is the subject of the suit.

The biopharmaceutical industry has produced a proliferation of patents, and it is not always clear to industry participants, including us, which patents cover various types of products. The coverage of patents is subject to interpretation by the courts, and the interpretation is not always uniform. In the event of infringement or violation of another party's patent or other intellectual property rights, we may not be able to enter into licensing arrangements or make other arrangements at a reasonable cost. Any inability to secure licenses or alternative technology could result in delays in the introduction of our products or lead to prohibition of the manufacture or sale of products by us or our partners and collaborators.

Patent litigation is costly and time consuming and may subject us to liabilities.

Our involvement in any patent litigation, interference, opposition or other administrative proceedings will likely cause us to incur substantial expenses, and the efforts of our technical and management personnel will be significantly diverted. In addition, an adverse determination in litigation could subject us to significant liabilities.

We may not obtain trademark registrations.

We have filed applications for trademark registrations in connection with our product candidates in various jurisdictions, including the United States. We intend to file further applications for other possible trademarks for our product candidates. No assurance can be given that any of our trademark applications will be registered in the United States or elsewhere, or that the use of any registered or

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unregistered trademarks will confer a competitive advantage in the marketplace. Furthermore, even if we are successful in our trademark registrations, the FDA and regulatory authorities in other countries have their own process for drug nomenclature and their own views concerning appropriate proprietary names. The FDA and other regulatory authorities also have the power, even after granting market approval, to request a company to reconsider the name for a product because of evidence of confusion in the marketplace. No assurance can be given that the FDA or any other regulatory authority will approve of any of our trademarks or will not request reconsideration of one of our trademarks at some time in the future. The loss, abandonment, or cancellation of any of our trademarks or trademark applications could negatively affect the success of the product candidates to which they relate.

Our revenues and expenses may fluctuate significantly, and any failure to meet financial expectations may disappoint securities analysts or investors and result in a decline in the price of our securities.

We have a history of operating losses. Our revenues and expenses have fluctuated in the past and are likely to do so in the future. These fluctuations could cause our share price to decline. Some of the factors that could cause our revenues and expenses to fluctuate include but are not limited to:

- the inability to complete product development in a timely manner that results in a failure or delay in receiving the required regulatory approvals to commercialize our product candidates;
- the timing of regulatory submissions and approvals;
- the timing and willingness of any current or future collaborators to invest the resources necessary to commercialize our product candidates;
- the revenue available from royalties derived from our strategic partners;
- licensing fees revenues;
- tax credits and grants (R&D);
- the outcome of litigation, if any;
- changes in foreign currency fluctuations;
- the timing of achievement and the receipt of milestone payments from current or future collaborators; and
- failure to enter into new or the expiration or termination of current agreements with collaborators.

Due to fluctuations in our revenues and expenses, we believe that period-to-period comparisons of our results of operations are not necessarily indicative of our future performance. It is possible that in some future quarter or quarters, our revenues and expenses will be above or below the expectations of securities analysts or investors. In this case, the price of our securities could fluctuate significantly or decline.

We will not be able to successfully commercialize our product candidates if we are unable to make adequate arrangements with third parties for such purposes.

We currently have a lean sales and marketing staff. In order to commercialize our product candidates successfully, we need to make arrangements with third parties to perform some or all of these services in certain territories.

We contract with third parties for the sales and marketing of our products. Our revenues will depend upon the efforts of these third parties, whose efforts may not be successful. If we fail to establish successful marketing and sales capabilities or to make arrangements with third parties for such purposes, our business, financial condition and results of operations will be materially adversely affected.

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If we had to resort to developing a sales force internally, the cost of establishing and maintaining a sales force would be substantial and may exceed its cost effectiveness. In addition, in marketing our products, we would likely compete with many companies that currently have extensive and well-funded marketing and sales operations. Despite our marketing and sales efforts, we may be unable to compete successfully against these companies.

We are currently dependent on strategic partners and may enter into future collaborations for the research, development and commercialization of our product candidates. Our arrangements with these strategic partners may not provide us with the benefits we expect and may expose us to a number of risks.

We are dependent on, and rely upon, strategic partners to perform various functions related to our business, including, but not limited to, the research, development and commercialization of some of our product candidates. Our reliance on these relationships poses a number of risks.

We may not realize the contemplated benefits of such agreements nor can we be certain that any of these parties will fulfill their obligations in a manner which maximizes our revenue. These arrangements may also require us to transfer certain material rights or issue our equity, voting or other securities to corporate partners, licensees and others. Any license or sublicense of our commercial rights may reduce our product revenue.

These agreements also create certain risks. The occurrence of any of the following or other events may delay product development or impair commercialization of our products:

- not all of our strategic partners are contractually prohibited from developing or commercializing, either alone or with others, products and services that are similar to or competitive with our product candidates and, with respect to our strategic partnership agreements that do contain such contractual prohibitions or restrictions, prohibitions or restrictions do not always apply to our partners' affiliates and they may elect to pursue the development of any additional product candidates and pursue technologies or products either on their own or in collaboration with other parties, including our competitors, whose technologies or products may be competitive with ours;

- our strategic partners may under-fund or fail to commit sufficient resources to marketing, distribution or other development of our products;

- we may not be able to renew such agreements;

- our strategic partners may not properly maintain or defend certain intellectual property rights that may be important to the commercialization of our products;

- our strategic partners may encounter conflicts of interest, changes in business strategy or other issues which could adversely affect their willingness or ability to fulfill their obligations to us (for example, pharmaceutical companies historically have re-evaluated their priorities following mergers and consolidations, which have been common in recent years in this industry);

- delays in, or failures to achieve, scale-up to commercial quantities, or changes to current raw material suppliers or product manufacturers (whether the change is attributable to us or the supplier or manufacturer) could delay clinical studies, regulatory submissions and commercialization of our product candidates; and

- disputes may arise between us and our strategic partners that could result in the delay or termination of the development or commercialization of our product candidates, resulting in litigation or arbitration that could be time-consuming and expensive, or causing our strategic partners to act in their own self-interest and not in our interest or those of our shareholders or other stakeholders.

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In addition, our strategic partners can terminate our agreements with them for a number of reasons based on the terms of the individual agreements that we have entered into with them. If one or more of these agreements were to be terminated, we would be required to devote additional resources to developing and commercializing our product candidates, seek a new partner or abandon this product candidate which would likely cause a drop in the price of our securities.

We have entered into important strategic partnership agreements relating to certain of our product candidates for various indications. Detailed information on our research and collaboration agreements is available in our various reports and disclosure documents filed with the Canadian securities regulatory authorities and filed with or furnished to the United States Securities and Exchange Commission ("SEC"), including the documents incorporated by reference in this Annual Report on Form 20-F. See, for example, Note 25 to our audited consolidated balance sheets as at December 31, 2010 and 2009 and our audited consolidated statements of operations, changes in shareholders' equity, accumulated other comprehensive income and deficit, comprehensive loss and cash flows for each of the years in the three-year period ended December 31, 2010 included in this Annual Report on Form 20-F.

We have also entered into a variety of collaborative licensing agreements with various universities and institutes under which we are obligated to support some of the research expenses incurred by the university laboratories and pay royalties on future sales of the products. In turn, we have retained exclusive rights for the worldwide exploitation of results generated during the collaborations.

In particular, we have entered into an agreement with the Tulane Educational Fund ("Tulane"), which provides for the payment by us of single-digit royalties on future worldwide net sales of cetrorelix and including Cetrotide®. Tulane is also entitled to receive a low double-digit participation payment on any lump-sum, periodic or other cash payments received by us from sub-licensees (see Note 25 to our audited consolidated balance sheets as at December 31, 2010 and 2009 and our audited consolidated statements of operations, changes in shareholders' equity, accumulated other comprehensive income and deficit, comprehensive loss and cash flows for each of the years in the three-year period ended December 31, 2010 included in this Annual Report on Form 20-F).

We rely on third parties to conduct, supervise and monitor our clinical trials, and those third parties may not perform satisfactorily.

We rely on third parties such as CROs, medical institutions and clinical investigators to enroll qualified patients and conduct, supervise and monitor our clinical trials. Our reliance on these third parties for clinical development activities reduces our control over these activities. Our reliance on these third parties, however, does not relieve us of our regulatory responsibilities, including ensuring that our clinical trials are conducted in accordance with Good Clinical Practice guidelines and the investigational plan and protocols contained in an Investigational New Drug application, or comparable foreign regulatory submission. Furthermore, these third parties may also have relationships with other entities, some of which may be our competitors. In addition, they may not complete activities on schedule, or may not conduct our pre-clinical studies or clinical trials in accordance with regulatory requirements or our trial design. If these third parties do not successfully carry out their contractual duties or meet expected deadlines, our efforts to obtain regulatory approvals for, and commercialize, our product candidates may be delayed or prevented.

In carrying out our operations, we are dependent on a stable and consistent supply of ingredients and raw materials.

There can be no assurance that we, our contract manufacturers or our partners, will be able, in the future, to continue to purchase products from our current suppliers or any other supplier on terms similar to current terms or at all. An interruption in the availability of certain raw materials or

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ingredients, or significant increases in the prices paid by us for them, could have a material adverse effect on our business, financial condition, liquidity and operating results.

The failure to perform satisfactorily by third parties upon which we rely to manufacture and supply products may lead to supply shortfalls.

We rely on third parties to manufacture and supply marketed products. We also have certain supply obligations *vis-à-vis* our licensing partners who are responsible for the marketing of the products. To be successful, our products have to be manufactured in commercial quantities in compliance with quality controls and regulatory requirements. Even though it is our objective to minimize such risk by introducing alternative suppliers to ensure a constant supply at all times, we cannot guarantee that we will not experience supply shortfalls and, in such event, we may not be able to perform our obligations under contracts with our partners.

We are subject to intense competition for our skilled personnel, and the loss of key personnel or the inability to attract additional personnel could impair our ability to conduct our operations.

We are highly dependent on our management and our clinical, regulatory and scientific staff, the loss of whose services might adversely impact our ability to achieve our objectives. Recruiting and retaining qualified management and clinical, scientific and regulatory personnel is critical to our success. Competition for skilled personnel is intense, and our ability to attract and retain qualified personnel may be affected by such competition.

Our strategic partners' manufacturing capabilities may not be adequate to effectively commercialize our product candidates.

Our manufacturing experience to date with respect to our product candidates consists of producing drug substance for clinical studies. To be successful, these product candidates have to be manufactured in commercial quantities in compliance with regulatory requirements and at acceptable costs. Our strategic partners' current manufacturing facilities have the capacity to produce projected product requirements for the foreseeable future, but we will need to increase capacity if sales continue to grow. Our strategic partners may not be able to expand capacity or to produce additional product requirements on favorable terms. Moreover, delays associated with securing additional manufacturing capacity may reduce our revenues and adversely affect our business and financial position. There can be no assurance that we will be able to meet increased demand over time.

We are subject to the risk of product liability claims, for which we may not have or be able to obtain adequate insurance coverage.

The sale and use of our products, in particular our biopharmaceutical products, involve the risk of product liability claims and associated adverse publicity. Our risks relate to human participants in our clinical trials, who may suffer unintended consequences, as well as products on the market whereby claims might be made directly by patients, healthcare providers or pharmaceutical companies or others selling, buying or using our products. We manage our liability risks by means of insurance. We maintain liability insurance covering our liability for our pre-clinical and clinical studies and for our pharmaceutical products already marketed. However, we may not have or be able to obtain or maintain sufficient and affordable insurance coverage, including coverage for potentially very significant legal expenses, and without sufficient coverage any claim brought against us could have a materially adverse effect on our business, financial condition or results of operations.

Our business involves the use of hazardous materials which requires us to comply with environmental and occupational safety laws regulating the use of such materials. If we violate these laws, we could be subject to significant fines, liabilities or other adverse consequences.

Our discovery and development processes involve the controlled use of hazardous and radioactive materials. We are subject to federal, provincial and local laws and regulations governing the use,

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manufacture, storage, handling and disposal of such materials and certain waste products. The risk of accidental contamination or injury from these materials cannot be completely eliminated. In the event of an accident or a failure to comply with environmental or occupational safety laws, we could be held liable for any damages that result, and any such liability could exceed our resources. We may not be adequately insured against this type of liability. We may be required to incur significant costs to comply with environmental laws and regulations in the future, and our operations, business or assets may be materially adversely affected by current or future environmental laws or regulations.

Legislative actions, new accounting pronouncements and higher insurance costs are likely to impact our future financial position or results of operations.

Changes in financial accounting standards or implementation of accounting standards may cause adverse, unexpected revenue or expense fluctuations and affect our financial position or results of operations. New pronouncements and varying interpretations of pronouncements have occurred with greater frequency and are expected to occur in the future, and we may make or be required to make changes in our accounting policies in the future. Compliance with changing regulations of corporate governance and public disclosure, notably with respect to internal controls over financial reporting, may result in additional expenses. Changing laws, regulations and standards relating to corporate governance and public disclosure are creating uncertainty for companies such as ours, and insurance costs are increasing as a result of this uncertainty.

We are subject to additional reporting requirements under applicable Canadian securities laws and the Sarbanes-Oxley Act in the United States. We can provide no assurance that we will at all times in the future be able to report that our internal controls over financial reporting are effective.

As a public company, we are required to comply with Section 404 of the Sarbanes-Oxley Act ("Section 404") and National Instrument 52-109 *Certification of Disclosure in Issuers' Annual and Interim Filings,* and we are required to obtain an annual attestation from our independent auditors regarding our internal control over financial reporting. In any given year, we cannot be certain as to the time of completion of our internal control evaluation, testing and remediation actions or of their impact on our operations. Upon completion of this process, we may identify control deficiencies of varying degrees of severity under applicable SEC and Public Company Accounting Oversight Board rules and regulations. As a public company, we are required to report, among other things, control deficiencies that constitute material weaknesses or changes in internal controls that, or that are reasonably likely to, materially affect internal controls over financial reporting. A "material weakness" is a deficiency, or a combination of deficiencies, in internal control over financial reporting, such that there is a reasonable possibility that a material misstatement of the company's annual financial statements will not be prevented or detected on a timely basis. If we fail to comply with the requirements of Section 404, Canadian requirements or report a material weakness, we might be subject to regulatory sanction and investors may lose confidence in our financial statements, which may be inaccurate if we fail to remedy such material weakness.

It is possible that we may be passive foreign investment company, which could result in adverse tax consequences to U.S. investors.

Adverse U.S. federal income tax rules apply to "U.S. Holders" (as defined in "Item 10.E Taxation Certain U.S. Federal Income Tax Consideration" in this Annual Report on Form 20-F) that directly or indirectly hold common shares or warrants of a passive foreign investment company ("PFIC"). We will be classified as a PFIC for U.S. federal income tax purposes for a taxable year if (i) at least 75 percent of our gross income is "passive income" or (ii) at least 50 percent of the average value of our assets, including goodwill (based on annual quarterly average), is attributable to assets which produce passive income or are held for the production of passive income.

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We believe that we were not a PFIC for the 2010 taxable year. However, since the fair market value of our assets may be determined in large part by the market price of our common shares, which is likely to fluctuate, and the composition of our income and assets will be affected by how, and how quickly, we spend any cash that is raised in any financing transaction, no assurance can be provided that we will not be classified as a PFIC for the 2011 taxable year and for any future taxable year.

PFIC characterization could result in adverse U.S. federal income tax consequences to U.S. Holders. In particular, absent certain elections, a U.S. Holder would be subject to U.S. federal income tax at ordinary income tax rates, plus a possible interest charge, in respect of a gain derived from a disposition of our common shares, as well as certain distributions by us. If we are treated as a PFIC for any taxable year, a U.S. Holder may be able to make an election to "mark to market" common shares each taxable year and recognize ordinary income pursuant to such election based upon increases in the value of the common shares. However, a mark-to-market election is not available to be made in respect of a warrant.

Under recently enacted U.S. tax legislation and subject to future guidance, if we are a PFIC, U.S. Holders will be required to file, for returns due after March 18, 2010, an annual information return with the Internal Revenue Service relating to their ownership of our common shares. Although expected, no guidance has yet been issued about such return, including on the information required to be reported on such return, the form of the return, or the due date of the return.

For a more detailed discussion of the potential tax impact of us being a PFIC, see "Item 10.E Taxation Certain U.S. Federal Income Tax Considerations" in this Annual Report on Form 20-F.

We will report under International Financial Reporting Standards for our interim and annual consolidated financial statements for the financial year ending December 31, 2011.

Effective January 1, 2011, the Accounting Standards Board of the Canadian Institute of Chartered Accountants require that Canadian publicly accountable enterprises adopt International Financial Reporting Standards ("IFRS"), as issued by the International Accounting Standards Board. We are thus required to report under IFRS for our interim and annual consolidated financial statements for the financial year ending December 31, 2011.

IFRS uses a conceptual framework that is similar to Canadian generally accepted accounting principles; however, we have identified certain differences that will result in changes to some of our accounting policies. We are currently in the process of preparing our first interim unaudited financial statements in accordance with IFRS, and the notes to such financial statements will explain in detail the specific impact of IFRS on our financial statements. Additional information on our conversion to IFRS is provided under "Item 5. Operating and Financial Review and Prospects" included in this Annual Report on Form 20-F.

We may incur losses associated with foreign currency fluctuations.

Our operations are in many instances conducted in currencies other than the euro, our functional currency. Fluctuations in the value of currencies could cause us to incur currency exchange losses. We do not currently employ a hedging strategy against exchange rate risk. We cannot assert with any assurance that we will not suffer losses as a result of unfavorable fluctuations in the exchange rates between the United States dollar, the euro, the Canadian dollar and other currencies. For more information, see "Item 11. Quantitative and Qualitative Disclosures About Market Risk" in this Annual Report on Form 20-F.

We may not be able to successfully integrate acquired businesses.

Future acquisitions may not be successfully integrated. The failure to successfully integrate the personnel and operations of businesses which we may acquire in the future with ours could have a material adverse effect on our operations and results.

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Risks Related to our Securities

Our share price is volatile, which may result from factors outside of our control. If our common shares were to be delisted from NASDAQ or TSX, investors may have difficulty in disposing of our common shares held by them.

Our common shares are currently listed and traded only on NASDAQ and TSX. Our valuation and share price since the beginning of trading after our initial listings, first in Canada and then in the United States, have had no meaningful relationship to current or historical financial results, asset values, book value or many other criteria based on conventional measures of the value of shares.

During the year ended December 31, 2010, the closing price of our common shares ranged from $0.79 to $2.09 on NASDAQ and from C$0.80 to C$2.14 per share on TSX. Our share price may be affected by developments directly affecting our business and by developments out of our control or unrelated to us. The stock market generally, and the biopharmaceutical sector in particular, are vulnerable to abrupt changes in investor sentiment. Prices of shares and trading volume of companies in the biopharmaceutical industry can swing dramatically in ways unrelated to, or that bear a disproportionate relationship to, operating performance. Our share price and trading volume may fluctuate based on a number of factors including, but not limited to:

clinical and regulatory developments regarding our product candidates;

delays in our anticipated development or commercialization timelines;

developments regarding current or future third-party collaborators;

other announcements by us regarding technological, product development or other matters;

arrivals or departures of key personnel;

governmental or regulatory action affecting our product candidates and our competitors' products in the United States, Canada and other countries;

developments or disputes concerning patent or proprietary rights;

actual or anticipated fluctuations in our revenues or expenses;

general market conditions and fluctuations for the emerging growth and biopharmaceutical market sectors; and

economic conditions in the United States, Canada or abroad.

Our listing on both NASDAQ and TSX may increase price volatility due to various factors, including different ability to buy or sell our common shares, different market conditions in different capital markets and different trading volumes. In addition, low trading volume may increase the price volatility of our common shares. A thin trading market could cause the price of our common shares to fluctuate significantly more than the stock market as a whole.

In the past, following periods of large price declines in the public market price of a company's securities, securities class action litigation has often been initiated against that company. Litigation of this type could result in substantial costs and diversion of management's attention and resources, which would adversely affect our business. Any adverse determination in litigation could also subject us to significant liabilities.

We must meet continuing listing requirements to maintain the listing of our common shares on NASDAQ and TSX. For continued listing, NASDAQ requires, among other things, that listed securities maintain a minimum closing bid price of not less than $1.00 per share.

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If we are unsuccessful in maintaining the NASDAQ's minimum bid requirements in the future and are unable to subsequently regain compliance within the applicable grace period, our common shares will be subject to delisting from the NASDAQ Global Market. Should we receive a delisting notification, we may appeal to the Listing Qualifications Panel or apply to transfer the listing of our common shares to the NASDAQ Capital Market if we satisfy at such time all of the initial listing standards on the NASDAQ Capital Market, other than compliance with the minimum closing bid price requirement. If the application to the NASDAQ Capital Market is approved, then we will have an additional 180-day grace period in order to regain compliance with the minimum bid price requirement while listed on the NASDAQ Capital Market. There can be no assurance that we will meet the requirements for continued listing on the NASDAQ Global Market or whether our application to the NASDAQ Capital Market will be approved or that any appeal would be granted by the Listing Qualifications Panel.

We do not intend to pay dividends in the near future.

To date, we have not declared or paid any dividends on our common shares. We currently intend to retain our future earnings, if any, to finance further research and the expansion of our business. As a result, the return on an investment in our securities will, for the foreseeable future, depend upon any future appreciation in value. There is no guarantee that our securities will appreciate in value or even maintain the price at which shareholders have purchased their securities.

Item 4. Information on the Company

A. *History and development of the Company*

Aeterna Zentaris Inc. is a late-stage drug development company specialized in oncology and endocrine therapy.

We were incorporated on September 12, 1990 under the *Canada Business Corporations Act* (the "CBCA") and continue to be governed by the CBCA. Our registered office is located at 1405 du Parc-Technologique Blvd., Quebec City, Quebec, Canada G1P 4P5, our telephone number is (418) 652-8525 and our website is www.aezsinc.com. None of the documents or information found on our website shall be deemed to be included in or incorporated into this annual report.

On December 30, 2002, we acquired Zentaris AG, a biopharmaceutical company based in Frankfurt, Germany. Zentaris was a spin-off of Degussa AG and Asta Medica GmbH, a former pharmaceutical company. With this acquisition, the Company changed its risk profile and inherited an extensive and robust product pipeline with capabilities from drug discovery to commercialization with a particular focus on endocrine therapy and oncology. As part of the acquisition, we also inherited a very experienced pharmaceutical team along with a network of strategic pharmaceutical partners. The total consideration paid for the acquisition of Zentaris was $51.9 million, net of cash and cash equivalents acquired of $2.3 million, of which an amount of $26.7 million was paid in cash and the remaining amount of $25.2 million as a balance of purchase price.

In May 2004, we changed our name to Aeterna Zentaris Inc. and on May 11, 2007, Zentaris GmbH was renamed Aeterna Zentaris GmbH ("AEZS GmbH"). AEZS GmbH is our principal operating subsidiary.

On April 6, 2005, our former subsidiary Atrium Biotechnologies Inc. (now Atrium Innovations Inc.) ("Atrium"), completed its initial public offering in Canada and began trading on the TSX under the ticker symbol "ATB."

Throughout 2006, as part of a thorough, strategic planning process, our management and Board of Directors (the "Board") made the decision to spin off Atrium in two phases. On September 19, 2006,

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we initiated the first phase, a secondary offering in which we sold 3,485,000 Subordinate Voting Shares of Atrium at a price of CAN$15.80 per share. This secondary offering closed on October 18, 2006, generating net proceeds of nearly $45 million to Aeterna Zentaris. With this transaction closed, our remaining interest in Atrium was 11,052,996 Subordinate Voting Shares representing 36.1% of its issued and outstanding shares. Therefore, we no longer had a controlling interest in Atrium as at October 18, 2006.

The second phase was to distribute our remaining interest in Atrium to our shareholders concurrently with a reduction of the stated capital of our common shares.

On December 15, 2006, our shareholders approved a reduction of the stated capital of our common shares in an amount equal to the fair market value of our remaining interest in Atrium by way of a special distribution in kind to all our shareholders. This special distribution was completed on January 2, 2007. For each common share held as at the record date of December 29, 2006, our shareholders received 0.2078824 Subordinate Voting Shares of Atrium. In May 2007, we opened an office in the United States, located at 20 Independence Boulevard, Warren, New Jersey 07059-2731.

We currently have three wholly-owned direct and indirect subsidiaries, Aeterna Zentaris GmbH ("AEZS Germany"), based in Frankfurt, Germany, Zentaris IVF GmbH, a direct wholly-owned subsidiary of AEZS Germany based in Frankfurt, Germany, and Aeterna Zentaris, Inc., based in Warren, New Jersey in the United States.

From the formation of Atrium as our subsidiary in 1999 until the distribution of our remaining interest in Atrium on January 2, 2007, Atrium did not declare or pay any dividends to its shareholders. Since the disposition of our entire interest in Atrium, we have not had access to the liquidity or cash flows generated by Atrium. Our current drug development strategy focuses mainly on our late-stage compounds perifosine (Phase 3 in multiple myeloma and colorectal cancer) and our Phase 2 program in multiple cancers, AEZS-108 (we recently completed with success a Phase 2 trial in endometrial and ovarian cancer and in clinical development in bladder and prostate cancer) and AEZS-130 (Solorel®) (Phase 3 as diagnostic test for adult growth hormone deficiency), as well as on targeted earlier-stage compounds, as depicted in the chart reproduced under the heading, "Our Product Pipeline".

Our common shares are listed for trading on the TSX under the trading symbol "AEZ" and on the NASDAQ under the trading symbol "AEZS."

The Company's agent for SEC matters in the United States is its wholly-owned subsidiary, Aeterna Zentaris, Inc., located at 20 Independence Boulevard, Warren, New Jersey 07059-2731.

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There have been no public takeover offers by third parties with respect to the Company or by the Company in respect of other companies' shares during the last or current fiscal year.

B.

Business overview

We are a late-stage drug development company specialized in oncology and endocrine therapy.

Our pipeline encompasses compounds at all stages of development, from drug discovery through to marketed products. The highest priorities in oncology are our Phase 3 program with perifosine in multiple myeloma and colorectal cancer, combined with our Phase 2 program in multiple cancers, as well as the further advancement of AEZS-108, we recently completed with success a Phase 2 trial in advanced endometrial and advanced ovarian cancer. AEZS-108 is also in development in other cancer indications, including refractory bladder and castration refractory prostate cancer. In endocrinology, our lead program is our Phase 3 trial with AEZS-130 (Solorel®) as a GH stimulation test for the diagnosis of GH deficiency in adults. We are advancing this Phase 3 trial with a Special Protocol Assessment ("SPA") obtained from the FDA.

Additionally, we are advancing AEZS-112, an oral anticancer agent which involves three mechanisms of action (tubulin, topoisomerase II and angiogenesis inhibition) in Phase 1, as well as several preclinical programs with novel targeted potential development candidates.

Recent Developments

For a complete description of our recent corporate and pipeline developments, refer to "Item 5. Operating and Financial Review and Prospects Highlights".

Our Business Strategy

Our primary business strategy is to advance, with the collaboration of our strategic partners, our product development pipeline with a focus on our flagship product candidates in oncology and endocrinology. In addition, we also continue to advance certain other clinical and pre-clinical programs as described below. Our vision is to become a fully-integrated specialty biopharmaceutical company.

Oncology

Our highest priorities in oncology are our Phase 3 program with perifosine in multiple myeloma and colorectal cancer, combined with our Phase 2 program in multiple cancers, as well as the further advancement of AEZS-108, which recently completed with success a Phase 2 trial in advanced endometrial and advanced ovarian cancer. AEZS-108 is also in development in other cancer indications, including refractory bladder and castration refractory prostate cancer.

Perifosine

Perifosine is a novel, oral anticancer treatment that inhibits Akt activation in the PI3K pathway. Perifosine, in combination with chemotherapeutic agents, is currently in Phase 3 studies for the treatment of multiple myeloma, colorectal cancer and in Phase 2 studies for the treatment of other cancers, and is the most advanced anti-cancer compound of its class in late-stage development. Perifosine as monotherapy is also being explored in other indications. The FDA has granted perifosine orphan-drug designation in multiple myeloma and in neuroblastoma and Fast Track designations in both multiple myeloma and refractory advanced colorectal cancer. Additionally, an agreement was reached with the FDA to conduct the Phase 3 trials in both of these indications under an SPA. Perifosine has also been granted Orphan Medicinal Product designation from the European Medicine Agency ("EMA") in multiple myeloma, and has received positive Scientific Advice from the EMA for both the multiple myeloma and advanced colorectal cancer programs, with ongoing Phase 3 trials for these indications expected to be sufficient for registration in Europe.

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AEZS-108

AEZS-108 represents a new targeting concept in oncology leading to personalized medicine using a cytotoxic peptide conjugate which is a hybrid molecule composed of a synthetic peptide carrier and doxorubicin. The design of AEZS-108 allows for the specific binding and selective uptake of the cytotoxic conjugate by luteinizing hormone releasing hormone ("LHRH")-receptor-positive tumors. Phase 2 trials in advanced endometrial cancer and advanced ovarian cancer have been completed with success. AEZS-108 is also in development in other cancer indications, including refractory bladder and castration refractory prostate cancer. We have obtained orphan-drug status for AEZS-108 in advanced ovarian cancer from the FDA and from the Committee for Orphan Medicinal Products of the EMA.

Endocrinology

In endocrinology, aside from Cetrotide®, we reactivated the Phase 3 trial with AEZS-130 (Solorel®) as an oral growth hormone ("GH") stimulation test for the diagnosis of adult growth hormone deficiency ("AGHD").

AEZS-130/Solorel®

AEZS-130/*Solorel® (macim*orelin), a ghrelin agonist, is a novel synthetic small molecule that stimulates the secretion of growth hormone. The product is currently in Phase 3 for use as a simple oral diagnostic test for AGHD. Solorel® has been granted orphan-drug designation by the FDA. In addition to the diagnostic indication, we believe that AEZS-130, based on the results of Phase 1 studies, has potential applications for the treatment of cachexia, a condition frequently associated with severe chronic diseases such as cancer, chronic obstructive pulmonary disease and Acquired Immune Deficiency Syndrome or AIDS.

Clinical and Preclinical Programs

Additionally, we are advancing in Phase 1, AEZS-112, an oral anticancer agent which involves three mechanisms of action, (tubulin, topoisomeras II and angiogenesic inhibition), as well as several preclinical programs with targeted potential development candidates. Among the targets for which we expect to propose clinical development candidates in the coming years are: AEZS-120 (prostate cancer vaccine), AEZS-127 (erucylphosphocholine derivatives), AEZS-129, AEZS-131 and AEZS-132 (Erk and PI3K inhibitors), AEZS-115 (non-peptide LHRH antagonists) and AEZS-123 (ghrelin receptor antagonist).

We also continue to perform targeted drug discovery activities from which we are able to derive pre-clinical candidates. This drug discovery includes high throughput screening systems and a library of more than 120,000 compounds.

We are currently in a stage in which some of our products and product candidates are being further developed or marketed jointly with strategic partners.

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Our product pipeline

Pipeline table

Status of our drug pipeline as at March 24, 2011

Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Commercial
120,000 compound library	AEZS-120 Prostate cancer vaccine (oncology) AEZS-129, 131 and 132 Erk & PI3K inhibitors (oncology) AEZS-127 ErPC (oncology) AEZS-123 Ghrelin receptor antagonist (endocrinology) AEZS-115 Non-peptide LHRH antagonists (endocrinology and/or oncology)	AEZS-112 (oncology) AEZS-130 Therapeutic in cancer cachexia and other indications (endocrinology)	Perifosine Multiple cancers AEZS-108 Ovarian cancer Endometrial cancer Castration refractory prostate cancer Refractory bladder cancer	Perifosine Multiple myeloma Refractory advanced colorectal cancer AEZS-130 (Solorel®) Diagnostic in adult growth hormone deficiency (endocrinology)	Cetrotide® (*in vitro* fertilization)
Partners					
			Perifosine: **Keryx** North America **Handok** Korea **Yakult** Japan	Perifosine: **Keryx** North America **Handok** Korea **Yakult** Japan	Cetrotide®: **Merck Serono** (World except Japan) **Nippon Kayaku / Shionogi** Japan

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ONCOLOGY

SIGNAL TRANSDUCTION INHIBITORS

Perifosine

Perifosine is a novel, oral anticancer treatment that inhibits Akt activation in the PI3K pathway.

Perifosine is an alkylphosphocholine compound with structural similarity to phospholipids, which are the main constituents of cellular membranes, and it is an active ingredient with anti-tumor capacities. In tumor cells, perifosine has demonstrated interactions with vital signal transduction mechanisms and induction of programmed cell death (apoptosis).

Perifosine exerts a marked cytotoxic effect in animal and human tumor cell lines. The most sensitive cancer cell lines were larynx carcinoma, breast, small cell lung, prostate and colon. Based on the *in vitro* trials, the mode of action of perifosine appears to be fundamentally different from that of currently available cytotoxics. Pharmacodynamic data have demonstrated that perifosine possesses anti-tumor activity, including tumor models that are resistant to currently available agents for cancer therapy. This activity is based on a direct and relatively specific action on tumors.

In preclinical and clinical Phase 1 trials (solid tumors), this orally administered agent has been found to have good tolerability. Five Phase 1 trials have been conducted on perifosine, including one trial of perifosine in combination with radiotherapy.

Based on findings in various tumor models, the U.S. National Cancer Institute, along with our North American partner, Keryx, investigated additional dosage regimens of perifosine in oncology patients. A number of screening Phase 2 studies examined perifosine as a single agent or in combination in several tumor types. Encouraging results lead to further development in specific indications.

Perifosine, in combination with chemotherapeutic agents, is currently in Phase 3 studies for the treatment of multiple myeloma, colorectal cancer and in Phase 2 studies for the treatment of other cancers, and is the most advanced anti-cancer compound of its class in late-stage development.

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Perifosine as monotherapy is also being explored in other indications. The FDA has granted perifosine orphan-drug designation in multiple myeloma and in neuroblastoma and Fast Track designations in both multiple myeloma and refractory advanced colorectal cancer. Additionally, an agreement was reached with the FDA to conduct the Phase 3 trials in both of these indications under an SPA. Perifosine has also been granted Orphan Medicinal Product designation from the EMA in multiple myeloma, and has received positive Scientific Advice from the EMA for both the multiple myeloma and advanced colorectal cancer programs, with ongoing Phase 3 trials for these indications expected to be sufficient for registration in Europe. Perifosine rights have been licensed to Keryx for North America, to Handok for Korea and recently to Yakult for Japan.

Perifosine Anti-cancer agent

Perifosine Multiple myeloma ("MM")

In June and December 2007, preliminary positive Phase 1 and Phase 2 data on perifosine were presented in patients with relapsed/refractory MM. Data demonstrated clinical activity of perifosine in combination with bortezomib and dexamethasone, and with lenalidomide (Revlimid®) + dexamethasone.

In December 2008, our partner Keryx presented final results of the Phase 1 clinical trial in which patients with relapsed or refractory MM were administered a combination of perifosine + lenalidomide and dexamethasone. Four cohorts of ≥6 patients each were enrolled and perifosine dose was 50 or 100 mg (daily), lenalidomide dose was 15 or 25 mg for days 1 to 21 and dexamethasone dose was 20 mg (for days 1-4; 9-12; and 17-20 for 4 cycles, followed by 20 mg for days 1-4) in 28-day cycles. To limit dexamethasone-related toxicities, the protocol was amended to use weekly dexamethasone (40 mg), applying to cohorts 3, 4, and the Maximal Tolerated Dose ("MTD") cohort. Dose Limiting Toxicity ("DLT") was defined as grade (G) 3 non-hematologic toxicity, G4 neutropenia for 5 days and/or neutropenic fever, or platelets <25,000/mm^3 on >1 occasion despite transfusion. Response was assessed by modified EBMT criteria. To be enrolled, patients had to have received at least one but no more than four prior therapies. Patients refractory to lenalidomide/dexamethasone were excluded. 32 patients (17 men and 15 women, median age 61 years old, range 37-80) were enrolled; 6 patients in cohort 1 (perifosine 50 mg, lenalidomide 15 mg, dexamethasone 20 mg); 6 patients in cohort 2 (perifosine 50 mg, lenalidomide 25 mg, dexamethasone 20 mg); 8 patients in cohort 3 (perifosine 100 mg, lenalidomide 15 mg, dexamethasone 40mg/week); 6 patients in cohort 4 (perifosine 100 mg, lenalidomide 25 mg, dexamethasone 40 mg/week) and 6 patients at MTD (Cohort 4). Median prior lines of treatment was 2 (range 1-4). Prior therapy included dexamethasone (94%), thalidomide (83%), bortezomib (47%), and stem cell transplant (47%). 37% of patients had progressed on prior thalidomide/dexamethasone. Two patients did not complete one full cycle (non-compliance and adverse event not related to study drugs both in cohort 3) and were not included in the safety and efficacy analysis. Of the 30 patients evaluable for safety, the most common (≥10%) grade 1 / 2 events included nausea (13%); diarrhea (17%); weight loss (17%); upper respiratory infection (23%); fatigue (30%); thrombocytopenia (20%); neutropenia (20%); hypophosphatemia (23%); increased creatinine (23%); anemia (36%); hypercalcemia (47%). Grade 3 / 4 adverse events ≥5% included neutropenia (20%); hypophosphatemia (17%); thrombocytopenia (13%); anemia (10%), fatigue (7%). There was one reported DLT in cohort 3 (nausea). Lenalidomide was reduced in 8 patients, perifosine reduced in

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8 patients and dexamethasone reduced in 6 patients. All 30 patients in the analysis were evaluable for response, with best response as follows:

Response: N = 30	**N (%)**	**Duration (wks)**	**ORR (≥PR)**
Near Complete Response (nCR)	2 (7%)	79+, 15+	
Very Good Partial Response (VGPR)	3 (10%)	62+, 34, 17	15 (50%)
Partial Response (PR)	10 (33%)	26+ (range 11 - 54+)	
Minimal Response (MR)	6 (20%)	17+ (range 9 - 30+)	
Stable Disease (SD)	7 (23%)	14+ (range 8 - 19)	
Progression (PD)	2 (7%)	8, 4	

stable disease: < 25% reduction in M-protein

Patients have tolerated the treatment regimen of perifosine + lenalidomide + dexamethasone well with manageable toxicity, and with encouraging clinical activity demonstrated by an overall response rate ("ORR") (> PR) of 50%.

Updated results of this study were presented in February 2009 at the 12th International Multiple Myeloma Meeting by our partner Keryx. Results indicated that Perifosine in combination with lenalidomide (Revlimid®) + dexamethasone continues to be well tolerated, with a median progression-free survival in responding patients of 10.9 months. Median overall survival still was not reached and was at 17 months at time of analysis.

Also in December 2008 during the meeting of the American Society of Hematology, Keryx presented results of a Phase 1/2 multicenter trial of perifosine + bortezomib (Velcade®) in patients with relapsed or relapsed/refractory MM who were previously relapsed from or refractory to bortezomib ± dexamethasone. The Phase 1 stage of the study enrolled a total of 18 patients in 4 cohorts of 3 patients each with dosing of perifosine 50 mg or 100 mg (daily) and bortezomib 1.0 or 1.3 mg/m^2 (on day 1, 4, 8, 11) in 21-day cycles. The selected dose for Phase 2 was perifosine 50 mg once daily + bortezomib 1.3 mg/m^2 (on day 1, 4, 8, 11) in 21-day cycles, with a planned enrollment of 64 patients. Dexamethasone 20 mg (on day of and after each bortezomib dose) could be added in patients with progressive disease ("PD"). For the Phase 1 portion, DLT was defined as any grade (G) 3 non-hematologic toxicity, G4 neutropenia for 5 day and/or neutropenic fever, or platelets <10,000/mm^3 on more than one occasion despite transfusion. Response was assessed by modified EBMT and Uniform criteria. A total of 76 patients have been enrolled (18 patients in Phase 1 and 58 patients in Phase 2) comprised of 45 men and 31 women, median age 63 years old, (range 41-89). 84% of patients had relapsed/refractory MM, with a median of 6 lines of prior treatment (range 2-13). Prior therapy included bortezomib (100%), dexamethasone (95%), thalidomide (79%), lenalidomide (71%) and stem cell transplant (57%). 63 patients have completed at least one cycle and were evaluable for safety (13 patients are currently not evaluable; 3 were removed in cycle 1 and 10 are too early in their treatment). Most common (>10%) grade 1 / 2 events were nausea, diarrhea, fatigue and myelosuppression, which were manageable with supportive care and growth factors. Grade 3 / 4 adverse events >5% included thrombocytopenia (40%); lymphopenia (36%); neutropenia (21%); anemia (14%); hyponatremia (13%); leukopenia (11%); proteinuria (8%), and upper respiratory infection (6%). No deep vein thrombosis has been seen, and only one worsening peripheral neuropathy from grade 1 to 3 has been reported to date. Two patients had perifosine reduced to 50 mg (nausea, fatigue) in the Phase 1 cohort, and 7 patients had bortezomib dose reductions primarily due to hematologic

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toxicity. 57 patients had completed at least 2 cycles and were evaluable for response, with best response to perifosine + bortezomib (+/ - dexamethasone) as follows:

		CR		PR		MR		ORR		SD	
All Patients: Best Response	**N=57**	**2**	**4%**	**7**	**12%**	**14**	**25%**	**23**	**40%**	**23**	**40%**
Perifosine + bortezomib	57	1	2%	5	9%	8	14%	14	24%	17	30%
With dexamethasone added*	31	1	2%	2	3%	6	11%	9	16%	6	11%

(* as a subset of the evaluable population)

9 of 76 patients (12%) rapidly progressed without response or stable disease ("SD"), including 6 patients in whom dexamethasone was also added. As at August 2008, the median time to progression ("TTP") for patients achieving ≥PR is 34 weeks, and for all patients achieving ≥MR is 33 weeks. Perifosine in combination with bortezomib (+/- dexamethasone) was generally well tolerated and is active in a heavily pre-treated bortezomib-exposed patient population, with an ORR of 40%, including an ORR of 37% and a median TTP of 9.25 months in responding but previously bortezomib-refractory patients.

Updated data for the effect of perifosine in combination with bortezomib (Velcade®) +/- dexamethosone were reported at the 12th International Multiple Myeloma Meeting in February 2009 by our partner Keryx. Eighty-four patients were enrolled in a combined Phase I/II study (18 patients in the Phase I component and 66 patients in the Phase II component). At the time of this analysis, 73 patients were evaluable for response. Median prior lines of therapy was 5 (range 1 - 13), including bortezomib (100%; 50% of the patients were previously treated with at least 2 bortezomib-based therapies and 81% were previously treated with bortezomib + dexamethasone); dexamethasone (98%); lenalidomide (Revlimid®) and/or thalidomide (Thalomid) (99%); and prior stem cell transplant (57%). No unexpected adverse events have been seen. Toxicities were manageable with supportive care and/or dose reductions as required.

Best response (MR or better) and stable disease (no progression for 3 months) to either perifosine + bortezomib (+/- dexamethasone) for patients previously relapsed from or refractory to prior bortezomib (Velcade®) treatment was as follows:

Evaluable Patients	**CR**		**PR**		**MR**		**ORR**		**SD> 3 mos**	
Bortezomib relapsed (n=20)	2	10%	6	30%	3	15%	11	55%	9	45%
Bortezomib refractory (n=53)	1	2%	6	11%	10	19%	17	32%	24	45%
All evaluable patients (n=73)	3	4%	12	16%	13	18%	28	38%	33	45%

Patients who had previously relapsed on a bortezomib-based treatment had a median TTP of 8.5 months. The median TTP for all 73 evaluable study patients (both bortezomib relapsed and refractory) was 6.4 months. As stated in Keryx's February 26, 2009 press release, there were 16 patients who remained at the time on active treatment.

Updated efficacy and safety data as well as new survival data on the clinical activity of perifosine in combination with bortezomib (Velcade®) +/- dexamethasone in patients with relapsed/refractory multiple myeloma were presented by our partner Keryx during the American Society of Hematology ("ASH") meeting in December 2009. Of the 73 evaluable patients, 53 patients (73%) were previously refractory to bortezomib (defined as progression on or within 60 days of treatment to a bortezomib-based regimen), including 44 patients who were refractory to the combination of

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bortezomib + dexamethasone. Twenty evaluable patients (27%) were relapsed to a prior bortezomib-based regimen. Best response for all 73 evaluable patients was as follows:

Evaluable Patients	**CR/nCR***		**PR**		**MR**		**ORR**		**SD****	
All Evaluable Patients (n=73)	3	4%	13	18%	14	19%	30	41%	30	41%
Bortezomib relapsed (n=20)	2	10%	7	35%	4	20%	13	65%	7	35%
Bortezomib refractory (n=53)	1	2%	6	11%	10	19%	17	32%	23	43%

* nCR = Near Complete Response is defined as meeting the criteria for CR (non-detectable monoclonal protein by serum and urine), except with detectable monoclonal protein by immunofixation.

** SD = Stable Disease for a minimum of 3 months.

Approximately 60% (45 / 73) of patients demonstrated progression (or SD for 4 cycles) at some point in their treatment and received 20 mg dexamethasone, four times per week, in addition to perifosine + bortezomib. Responses occurred both with patients taking perifosine in combination with bortezomib and with patients receiving the combination + dexamethasone.

Best response for each group was as follows:

Best Response	**CR/nCR**		**PR**		**MR**		**ORR**		**SD**	
Perifosine + bortezomib (n=73)	2	3%	10	14%	6	8%	18	25%	19	26%
Dexamethasone added (n=45)	1	2%	6	13%	10	23%	17	38%	14	31%

Five patients achieved an initial response on perifosine + bortezomib alone, and subsequently responded again with the addition of dexamethasone. Three additional patients achieved stable disease on perifosine + bortezomib alone, and subsequently achieved stable disease again with the addition of dexamethasone.

Reported for the first time was median Progression-Free Survival ("PFS") and Overall Survival ("OS") data for all evaluable patients, as follows:

Evaluable Patients	**Median PFS***	**Median OS****
All Evaluable Patients (n=73)	6.4 months 95% CI (5.3, 7.1)	25 months 95% CI (15.5, NR)

NR = Not Reached

* Median PFS and median TTP were identical, as no patient deaths occurred prior to progression.

** Kaplan Meier methodology was used to determine overall survival figures.

Of particular interest was the comparison of evaluable patients who were previously refractory and the patients who were relapsed to a bortezomib-based regimen. Median PFS and OS for bortezomib relapsed vs. refractory was as follows:

Bortezomib Relapsed vs. Refractory	**Median PFS***	**Median OS****
Bortezomib relapsed (n=20)	8.8 months 95% CI (6.3, 11.2)	Not reached at 38+ months 95% CI (25, NR)
Bortezomib refractory (n=53)	5.7 months 95% CI (4.3, 6.4)	22.5 months 95% CI (12.3, NR)

* Median PFS and median TTP were identical, as no patient deaths occurred prior to progression.

** Kaplan Meier methodology was used to determine overall survival figures.

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No unexpected adverse events have been observed. Toxicities were manageable with supportive care.

In August 2009, we announced that our partner Keryx reported that it had reached an agreement with the FDA regarding an SPA on the design of a Phase 3 trial for perifosine, in relapsed or relapsed/refractory multiple myeloma patients previously treated with bortezomib (Velcade®). The SPA provided agreement that the Phase 3 study design adequately addresses objectives in support of a regulatory submission.

In September 2009, we announced that our partner Keryx reported that it had received orphan-drug designation for perifosine from the FDA for the treatment of multiple myeloma. Orphan-drug designation is granted by the FDA Office of Orphan Drug Products to novel drugs or biologics that treat a rare disease or condition affecting fewer than 200,000 patients in the U.S. The designation provides the drug developer with a seven-year period of U.S. marketing exclusivity if the drug is the first of its type approved for the specified indication or if it demonstrates superior safety, efficacy or a major contribution to patient care versus another drug of its type previously granted the designation for the same indication.

On December 2, 2009, we announced that the FDA had granted Fast Track designation for perifosine for the treatment of relapsed/refractory multiple myeloma. The Fast Track program of the FDA is designed to facilitate the development and expedite the review of new drugs that are intended to treat serious or life-threatening conditions and that demonstrate the potential to address unmet medical needs. Fast Track designated drugs ordinarily qualify for priority review, thereby expediting the FDA review process.

On December 16, 2009, we announced that our partner Keryx initiated a Phase 3 trial for perifosine entitled, "A Phase 3 Randomized Study to Assess the Efficacy and Safety of Perifosine Added to the Combination of Bortezomib (Velcade®) and Dexamethasone in Multiple Myeloma Patients Previously Treated with Bortezomib". The randomized (1:1), double-blind trial powered at 90%, will enroll approximately 400 patients with relapsed or relapsed/refractory multiple myeloma (patients can be relapsed from and refractory to all non-bortezomib based therapies, however, patients can only be relapsed (progressed > 60 days after discontinuing therapy) from prior bortezomib-based therapies. Patients must have been previously treated with both bortezomib (Velcade®) and an immunomodulatory agent (Revlimid® or Thalidomid®) and previously treated with one to four prior lines of therapy. Enrolled patients are randomized to bortezomib (Velcade®) at 1.3 mg/m^2 days 1, 4, 8 and 11 every 21 days in combination with dexamethasone 20 mg on the day of and day after bortezomib (Velcade®) treatment, and either perifosine 50 mg daily or placebo. The primary endpoint is progression-free survival and secondary endpoints include overall response rate, overall survival and safety.

As stated by our partner Keryx, it is expected that the study will be completed during the second half of 2012. Approximately 265 events (defined as disease progression or death) will trigger the un-blinding of the data.

In March 2010, we announced that we had received a positive opinion for orphan medicinal product designation for perifosine from the Committee for Orphan Medicinal Products ("COMP") of the European Medicines Agency, for the treatment of multiple myeloma. Orphan medicinal product designation is granted by the European Commission, following a positive opinion from the COMP, to a medicinal product that is intended for the diagnosis, prevention or treatment of a life-threatening or a chronically debilitating condition affecting not more than five in 10,000 persons in the European Community when the application for designation is submitted.

Orphan medicinal product designation provides the sponsor with access to the Centralized Procedure for the application for marketing authorization, protocol assistance, up to a 100% reduction in fees related to a marketing authorization application, pre-authorization inspection and post-authorization activities, and could provide ten years of market exclusivity in the EU, once approved for the treatment of multiple myeloma.

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On April 15, 2010, we received Positive Scientific Advice from the EMA for the Phase 3 registration trial with perifosine in multiple myeloma, therefore indicating that the data from the ongoing trial are expected to be sufficient for product registration in Europe.

On December 6, 2010 at the ASH's 52nd annual meeting in Orlando, Florida, we announced updated positive Phase 1 results of perifosine in combination with lenalidomide (Revlimid®) + dexamethasone in patients with relapsed or refractory multiple myeloma. The final data showed a 73% objective response rate (minimal response or better) with a 50% PR or better, a median Progression-Free Survival of 10.8 months, and a median duration for Overall Survival of 30.6 months. The myeloma investigators concluded that perifosine in combination with lenalidomide (Revlimid®) + dexamethasone was well tolerated even at the highest doses used, and demonstrated encouraging clinical activity and survival.

Competitors for Perifosine in Multiple Myeloma Indication

Products on the market

Major products available on the market for the treatment of multiple myeloma are the following:

Velcade® (bortezomib manufactured by Millenium: The Takeda Oncology Company), a proteasome inhibitor approved in combination with melphalan (Alkeran® Manufactured by Celgene) and prednisone as a 1st-line treatment and as a monotherapy for 2nd-line treatment in both the U.S. and the EU Millennium reported, according to Takeda's 2010 Annual Report, approximately $0.5 billion in global Velcade® estimated sales in 2009 (Velcade® is co-developed by Millennium Pharmaceuticals, Inc. and Johnson & Johnson Pharmaceutical Research & Development, L.L.C. Millennium is responsible for commercialization of Velcade® in the U.S., Janssen-Cilag is responsible for commercialization in Europe and the rest of the world. Janssen Pharmaceutical K.K. is responsible for commercialization in Japan).

Caelyx®/Doxil® (pegylated liposomal doxorubicin Manufactured by Schering Plough), a topoisomerase II inhibitor and DNA intercalating agent, is approved as a 2nd-line treatment in combination with Velcade® in patients with advanced multiple myeloma.

Thalomid® (thalidomide Manufactured by Celgene), an antiangiogenic compound has been approved by the FDA for use in combination with dexamethasone for the treatment of patients with newly diagnosed multiple myeloma. The Australian Therapeutic Goods Administration (TGA) approved a supplemental filing granting Thalomid® marketing approval for use in combination with melphalan and prednisone for patients with untreated multiple myeloma or ineligible for high-dose chemotherapy, and also granted Thalomid® marketing approval in combination with dexamethasone for induction therapy prior to high-dose chemotherapy with autologous stem cell rescue, for the treatment of patients with untreated multiple myeloma. In addition, Thalomid® was granted full marketing authorization by the European Commission ("EC") for use in combination with melphalan and prednisone as a treatment for patients with newly diagnosed multiple myeloma. Internationally, Thalomid® is also distributed under mandatory risk-management distribution programs tailored to meet local competent authorities' specifications to help ensure the safe and appropriate distribution and use of Thalomid®. According to Celgene's 2009 Annual Report, Thalomid® sales were down 13.4% to approximately $436.9 million in 2009.

Revlimid® (lenalidomide Manufactured by Celgene): Revlimid® is an oral immunomodulatory drug approved by the FDA and a number of other regulatory agencies in Europe, Latin America, Middle East and Asia/Pacific for treatment in combination with dexamethasone for multiple myeloma patients who have received at least one prior therapy and in Australia and New Zealand in combination with dexamethasone for the treatment of patients whose disease has progressed after one therapy. Revlimid® is distributed internationally under mandatory risk-management distribution programs tailored to meet local competent authorities' specifications to help ensure the safe and appropriate distribution and use of Revlimid®.

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Revlimid® continues to be evaluated in numerous clinical trials worldwide either alone or in combination with one or more other therapies in the treatment of a broad range of hematological malignancies, including multiple myeloma, myelodysplastic syndromes ("MDS"), non-Hodgkin's lymphoma ("non-HL"), chronic lymphocytic leukemia ("CLL"), other cancers and other diseases. According to Celgene's 2009 Annual Report, Revlimid® sales were up 28.8% to approximately $1.7 billion in 2009.

Products in Phase 3 development:

Panobinostat (LBH5893) Novartis: Panobinostat is a highly potent pan-deacetlyase inhibitor (pan-DACi) developed by Novartis. Panobistat's mechanism of action involves disrupting aggresome function, promoting accumulation of cytotoxic misfolded protein aggregates and triggering of myeloma cell death. Combination of pan-DAC and protease inhibition by co-treatment with panobinostat and bortezomib as demonstrated synergistic cytotoxicity *in vitro* and *in vivo* in preclinical experiments. Clinical experience in advanced multiple myeloma patients treated by oral panobinostat and i.v. bortezomib +/- dexamethasone showed efficacy and manageable toxicity profile. Panobinostat is currently in Phase 3 trial in patients with relapsed multiple myeloma in combination with bortezomib.

Idamycin (Idarubicin) Pfizer: Idarubicin is an oral anthracyclines and an analogue of daunorubicin (but 5 to 6 times more potent than daunorubicin) developed by Pfizer. The mechanism of action of anthracyclines is poorly understood and cytotoxicity is generally attributed to intercalation of the drug into DNA and inhibition of DNA topoisomerase II activity resulting in double and single strand DNA breaks. Idarubicin is already approved in Canada for Acute lymphocytic leukemia in adults and children as a second-line treatment and in Acute non-lyphocytic leukemia in adults as a front-line treatment or for refractory/relapsed disease. Idarubicin is currently in Phase 3 clinical trial for patients with Stage I or Stage II multiple myeloma in combination with dexamethasone.

Zolinza (vorinostat MK0683) Merck: Zolanza is an oral histone deacetylase (HDAC) inhibitor developed by Merck. Zolinza works by inhibiting the enzymatic activity of HDAC1, HDAC2, HDAC3 (Class 1) and HDAC6 (Class II). Inhibition of HDAC may result in anti-cancer effects since HDAC inhibitors, like zolinza, have the ability to induce antiproliferative effects including cyto-differentiation, cell cycle growth arrest or apoptosis in various cancer cell lines. The exact mechanism of the anticancer effect of Zolinza has not been fully characterized. Phase 1 results showed early anti-tumor activity in patients with releaspsed and/or refractory multiple myeloma when zolenza was administered in combination with bortezomib, including in patients previously treated with and no longer responding to bortezomib. A Phase 3 randomized, double-blind, placebo-controlled trial of zolinza in combination with bortezomib in patients with relapsed and/or refractory multiple myeloma is currently enrolling patients. Pulmonary embolism and deep vein thrombosis have been reported as adverse reactions following treatment with zolinza.

Carfilzomib Onyx Pharmaceuticals: Carfilzomib is the first in a new class of selective, irreversible proteasome inhibitors being developed by Proteolix (now part of Onyx Pharmaceuticals) for the treatment of hematologic malignancies and solid tumors. Carfilzomib produces specific and sustained inhibition of the proteasome, leading to apoptosis in cancer cells with minimal off-target effects. In Phase 1 and Phase 2 clinical trials, carfilzomib has demonstrated single-agent activity in hematologic malignancies and solid tumors, including multiple myeloma, Waldenstrom's macroglobulinemia, mantle cell lymphoma and renal cell carcinoma. Carfilzomib was generally well tolerated and toxicities were manageable. A Phase 3 international randomized trial evaluating the efficacy of carfilzomib in combination with lenalidomide and dexamethasone versus lenalidomide and dexamethasone as a potential treatment option for patients with relapsed multiple myeloma was started in March 2010. Orphan Drug designation was granted by EMA in June 2008 for the treatment of multiple myeloma.

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Market Data Multiple Myeloma

Multiple myeloma is the second most common blood cancer in United States and constitutes approximately 1% of all cancers.

According to Decision Resources January 2011, about 131,190 diagnosed prevalent cases (men and women) occurred in multiple myeloma in 2010 in the major markets comprising the U.S., Europe (G5) and Japan. The number of diagnosed incident cases was estimated at 44,780 in 2010 for the total major markets.

Perifosine Colon Cancer

In June 2009, results of a randomized Phase 2 study of perifosine in combination with capecitabine versus capecitabine alone in patients with second- or third-line metastatic colon cancer were presented during the American Society of Clinical Oncology ("ASCO") meeting.

This randomized, double-blind, placebo-controlled study was conducted at 11 centers across the United States. Patients with 2nd or 3rd line metastatic colon cancer were randomized to receive capecitabine (Xeloda®), an approved drug for metastatic colon cancer, at a dose of 825 mg/m^2 BID (total daily dose of 1650 mg/m^2) on days 1 - 14 every 21 days, plus either perifosine or placebo at 50 mg daily. Treatment was continued until progression. The study enrolled a total of 38 patients, 34 of which were third-line or greater. Of the 38 patients enrolled, 35 were evaluable for response (20 patients on the perifosine + capecitabine arm and 15 patients on the placebo + capecitabine arm). Of the three patients on the placebo + capecitabine arm not evaluable for response, 2 patients were not evaluable due to toxicity (days 14, 46) and 1 patient was not evaluable due to a new malignancy on day 6. All patients in the perifosine + capecitabine arm were evaluable for response.

The patients in the study were heavily pre-treated, with the arms well-balanced in terms of prior treatment regimens. The median number of prior treatment regimens for all 38 patients was two (range 1-5), with prior treatment regimens as follows: 91% of the patients received FOLFIRI (Irinotecan + 5FU + Leucovorin); 74% FOLFOX (Oxaliplatin + 5FU + Leucovorin); 63% were previously treated with both FOLFIRI and FOLFOX; 77% received Avastin; and 43% Erbitux®. Prior treatment with single agent capecitabine was excluded.

The primary endpoints of this study were to measure 1) TTP, 2) ORR, defined as the percentage of patients achieving a Complete Response ("CR") or Partial Response ("PR") by Response Evaluation Criteria in Solid Tumors ("RECIST"), and 3) Clinical Benefit Rate ("CBR") defined as the percentage of patients on treatment for greater than three months with at least SD. Safety of perifosine + capecitabine vs. placebo + capecitabine in this patient population was evaluated as a secondary endpoint. Perifosine in combination with capecitabine was well tolerated with hand/foot syndrome (14%) and anemia (11%) as the highest reported grade $^3/_4$ adverse events.

Best response and median time to progression of perifosine + capecitabine vs. placebo + capecitabine were as follows:

Group	N	CR N(%)	PR N(%)	ORR N(%)	SD > 12 wks N(%)	CBR N(%)	Median TTP (wks)
Perifosine + capecitabine	20	1 (5%)	3 (15%)	4 (20%)	11 (55%)	15 (75%)	28.9 weeks {95% CI (13, 48.1)}
Placebo + capecitabine	15	0	1 (7%)	1 (7%)	5 (33%)	6 (40%)	11 weeks {95% CI (9, 15.9)}

Perifosine + capecitabine more than doubled time to progression vs. placebo + capecitabine with a statistically significant p-value = 0.0006. In addition, perifosine + capecitabine more than doubled the ORR and almost doubled the CBR vs. placebo + capecitabine.

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Although not a primary endpoint in the study, overall survival was analyzed with results as follows:

Group	Median Overall Survival*(months)	% change
Perifosine + capecitabine	22 {95% CI (12.1, NR)}	26% Increase**
Placebo + capecitabine	16.3 {95% CI (5.3, 17.1)}	

* Survival calculated from date of randomization until date of death from any cause, whether or not additional therapies were received after removal from treatment.

** As at May 2009, median overall survival in the perifosine + capecitabine patient group is ongoing with 10 of the 20 patients in this arm still alive.

Updated results of this Phase 2 study were presented in January 2010 during the ASCO Gastrointestinal Cancers symposium. The primary endpoint of this study was to measure TTP. ORR, defined as CR+PR by RECIST, and OS were measured as a secondary endpoint. Updated results demonstrated a statistically significant advantage in the combination arm of perifosine + capecitabine for TTP and OS, as well as for the percentage of patients achieving SD lasting 12 or more weeks or better, as compared to the capecitabine arm. The perifosine + capecitabine arm demonstrated a greater than 60% improvement in OS, a more than doubling of median TTP, and almost a doubling of the percentage of patients achieving SD or better. In addition, the ORR was 20% (including one CR, and durable responses) in the perifosine + capecitabine arm vs 7% in the capecitabine arm. The updated efficacy results for all evaluable patients are as follows:

Group	N	ORR % CR / PR (Duration of Response)	> SD (min 12 wks) N (%) p=0.036	Median TTP Weeks p=0.0012	Median OS* Months p=0.0136
Perifosine + capecitabine	20	20% 1 CR (34 mos - ongoing) 3 PR (21, 19, 11 mos)	15 (75%)	28 [95% CI (12-48)]	18 [95% CI (10.8-25.7)]
Capecitabine	15	7% 1 PR (7 mos)	6 (40%)	11 [95% CI (9-15.9)]	11 [95% CI (5.3-16.9)]

* Survival calculated from date of randomization until date of death from any cause, whether or not additional therapies were received after removal from treatment.

Of notable interest, and for the first time presented, were data showing a highly statistically significant benefit in median OS (more than doubling) and TTP for the subset of patients who were refractory to a 5-FU (Fluorouracil) chemotherapy-based treatment regimen. 5-FU is a core component of the standard of care FOLFIRI and FOLFOX regimens, and capecitabine is a 5-FU pro-drug. These results are shown below:

Group	5-FU Ref* N (%)	> SD (min 12 wks) N (%) p=0.066	Median TTP Weeks p=0.0004	Median OS Months p=0.0088
Perifosine + capecitabine	14 (70%)	1 PR / 8 SD (64%)	18 [95% CI (12-36)]	15.3 [95% CI (8.4-26)]
Capecitabine	11 (73%)	0 PR / 3 SD (27%)	10 [95% CI (6.6-11)]	6.8 [95% CI (4.8-11.7)]

*

Ref= refractory

All 38 patients were evaluable for safety. The perifosine + capecitabine combination was well-tolerated with Grade 3 and Grade 4 adverse events of > 10% incidence for perifosine + capecitabine arm versus capecitabine arm as follows: anemia (15% vs. 0%), fatigue (0% vs. 11%), abdominal pain (5% vs. 11%) and hand-foot syndrome (30% vs. 0%). Of note, incidence of Grade 1 and Grade 2 hand-foot syndrome was similar in both the perifosine + capecitabine and capecitabine arms (25% vs. 22%, respectively). Hand-foot syndrome is a reported adverse event with capecitabine monotherapy. Patients who remained on treatment longer in the Phase 2 study had a greater chance to develop hand-foot syndrome as illustrated by a median time to onset of Grade 3 and Grade 4 hand-foot syndrome in the perifosine + capecitabine arm of 19 weeks.

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On February 3, 2010, we announced that our partner Keryx had reached an agreement with the FDA on an SPA for the Phase 3 X-PECT trial for perifosine in patients with refractory metastatic colorectal cancer.

On April 5, 2010, our partner Keryx was granted Fast Track designation by the FDA for the Phase 3 X-PECT registration trial.

On April 8, 2010, we announced that our partner Keryx initiated a randomized (1:1), double-blind Phase 3 X-PECT trial comparing the efficacy and safety of perifosine + capecitabine (Xeloda®) vs. placebo + capecitabine in approximately 430 patients with refractory metastatic colorectal cancer. Patients must have failed available therapy including 5-fluorouracil, oxaliplatin (Eloxatin®), irinotecan, bevacizumab (Avastin®) and, if K-Ras wild-type, failed therapy with prior cetuximab (Erbitux®) or panitumumab (Vectibix®). For oxaliplatin-based therapy, failure of therapy also includes patients who discontinued due to toxicity. The primary endpoint is overall survival, with secondary endpoints including overall response rate (complete responses + partial responses), progression-free survival and safety. Approximately 70 U.S. sites are participating in the study. Enrollment is expected to take approximately 12 months, with study completion expected by the end of 2011. Dr. Johanna Bendell, Director of GI Oncology Research for the Sarah Cannon Research Institute, Nashville, Tennessee, leads the Phase 3 investigational team.

On June 8, 2010, Phase 2 results were reported at the ASCO annual meeting, confirming a statistically significant improvement in both time to tumor progression and overall survival with perifosine, in combination with capecitabine in the treatment of advanced metastatic colorectal cancer. The perifosine + capecitabine arm demonstrated a greater than 60% improvement in overall survival, a more than doubling of median time to progression, and almost a doubling of the percentage of patients achieving stable disease or better. In addition, the overall response rate was 20% (including one complete response, and durable responses) in the perifosine + capecitabine arm versus 7% in the capecitabine arm. Of notable interest were the patients who were previously refractory to a 5-FU based regimen. The perifosine + capecitabine arm again demonstrated a statistically significant increase in both time to progression and overall survival, as compared to the capecitabine arm. As for safety, the perifosine + capecitabine arm was well tolerated.

On June 29, 2010, we announced that we had received positive Scientific Advice from the EMA regarding the Phase 3 X-PECT trial for the development of perifosine in refractory advanced colorectal cancer. The Scientific Advice from the EMA indicates that the ongoing study, in conjunction with safety data generated from other clinical studies with perifosine, is considered sufficient to provide all data necessary to support a marketing authorization of perifosine in advanced colorectal cancer. We do not intend to initiate any additional studies with perifosine for this indication. Therefore, for the development of perifosine in both multiple myeloma and colorectal cancer, we believe that the planned North American clinical program, sponsored by our partner Keryx, is now sufficient for approval in Europe and in many countries in the rest of the world, where we hold rights for our compound.

Competitors for Perifosine in colon cancer indication

Products on the market:

Standard 1st-line therapies for treatment of colon cancer are usually the FOLFOX (5-fluorouracil; leucovorin; oxaliplatin) or the FOLFIRI (5-fluorouracil; leucovorin; irinotecan) combination.

Current therapies also include:

Xeloda® (Capecitabine Manufactured by Roche) is an oral fluoropyrimidine which generates fluorouracil preferentially in tumor tissues by enzymatic cascade and is used in 1st or 2nd-line setting for treatment of metastatic colorectal or colon cancer in monotherapy and also in combination with any chemotherapy in all lines with or without Avastin. According to Roche's 2010 Annual Report, sales of

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Xeloda for colorectal, stomach and breast cancer increased 17% to approximately 1.4 billion Swiss francs in 2010.

Avastin® (Bevacizumab, a humanized monoclonal antibody targeting vascular endothelial growth factor manufactured by Genentech/Roche) is also used in 1st or 2nd-line treatment of metastatic colorectal cancer combined with available Standard therapy FOLFOX. According to Roche's 2010 Annual Report, sales of Avastin® for advanced colorectal, breast, lung and kidney cancer, and for relapsed glioblastoma (a type of brain tumour), rose 9% to approximately 6.5 billion Swiss francs in 2010.

Erbitux® (Cetuximab) is a chimeric monoclonal antibody that specifically blocks the epidermal growth factor receptor (EGFR). Cetuximab is indicated for the treatment of patients with EGFR-expressing, KRAS wild-type metastatic colorectal cancer in combination with Standard chemotherapy FOLFIRI, and in patients who have failed oxaliplatin- and irinotecan-based therapy. Erbitux® is manufactured and distributed in North America by ImClone and Bristol-Myers Squibb, while in the rest of the world distribution is by Merck KGaA. According to Merck's 2009 Annual Report, sales of Erbitux® increased by 23% to €697 million, or approximately $968 million, compared to 2008. On March 29, 2010, Merck Serono, a division of Merck KGaA announced that Erbitux® granted extended use in Japan for first-line-treatment for mCRC patients with KRAS wild-type tumors. In the United Kingdom, the National Institute for Health and Clinical Excellence (NICE) recommended in June the use of Erbitux® in combination with chemotherapy as a first-line treatment for patients with metastatic colorectal cancer who have met specific additional criteria improving the possibility of potentially curative surgery.

Vectibix® (Panitumumab) is a recombinant, human IgG2 kappa monoclonal antibody manufactured by Amgen that binds specifically to the human epidermal growth factor receptor (EGFR). Vectibix® is indicated as a single agent for the treatment of EGFR-expressing, metastatic colorectal carcinoma with disease progression on or following fluoropyrimidine-, oxaliplatin-, and irinotecan-containing chemotherapy regimens. FDA approval was achieved in September 2006. There are 2 boxed warnings for Vectibix®: dermatologic toxicity and infusion reactions. According to Amgen's 2009 Annual Report Vectibix® worldwide sales for the year 2009 were $233 million. In 2009, Amgen announced that primary endpoint of extending progression-free survival was met in Phase 3 studies evaluating Vectibix® in combination with FOLFOX or FOLFIRI. Based on these study results, Amgen is planning to file for regulatory approval in the United States and Europe for first- and second-line treatment in patients with KRAS wild-type metastatic colorectal cancer.

Product in Phase 3 development:

Aflibercept Sanofi + Regeneron: Aflibercept is an anti-angiogenesis inhibitor with a unique mechanism of action being developed by Sanofi and Regeneron. This fusion protein binds all forms of Vascular Endothelial Growth Factor-A ("VEGF-A"), as well as VEGF-B and placental growth factor ("PIGF"), additional angiogenic growth factors that appear to play a role in tumor angiogenesis and inflammation. Aflibercept has been shown to bind VEGF-A, VEGF-B, and PlGF with higher affinity than their natural receptors. The following clinical studies are currently ongoing and are fully enrolled:

VELOUR study: 2nd-line metastatic colorectal cancer in combination with fluorouracil, leucovorin, and irinotecan (FOLFIRI) Final results anticipated in the first half of 2011;

VITAL study: 2nd-line non-small cell lung cancer in combination with docetaxel Final results anticipated in the first half of 2011;

VENICE study: 1st-line hormone-refractory metastatic prostate cancer in combination with docetaxel and prednisone Final results anticipated in the second half of 2012;

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AFFIRM study: 1st-line treatment for metastatic colorectal cancer in combination with FOLFOX	Initial data anticipated in the second half of 2011.

Aptocine Light Sciences Oncology: Aptocine is a water-soluble drug targeted by a single-use, disposable drug activator included with the drug. Aptocine has three mechanisms of action: direct tumor cytotoxicity, apoptosis caused by vascular shutdown and potential anti-tumor immune stimulation. Enrollment of a Phase 3 trial for aptocine in metastatic colorectal cancer is nearly completed. This Phase 3 trial is a 450-patient trial, conducted primarily at sites in Europe and India, to assess the progression-free survival and overall survival of patients treated with Aptocine + chemotherapy versus chemotherapy alone.

Brivanib Bristol-Myers Squibb: Brivanib, developed by Bristol-Myers Squibb, is an oral prodrug of BMS-540215, a dual tyrosine kinase inhibitor of VEGFR and FGFR signalling. Brivanib strongly binds to and inhibits VEGFR2, a tyrosine kinase receptor expressed almost exclusively on vascular endothelial cells. The inhibition of VEGFR2 may result in inhibition of tumor angiogenesis, inhibition of tumor cell growth, and tumor regression. Brivanib is currently in Phase 3 randomized trial investigating Brivanib Alaninate in combination with cetuximab (Erbitux®) vs. placebo in combination with cetuximab (Erbitux®) in patients with K-RAS tumors previously treated with combination chemotherapy for metastatic colorectal carcinoma. It is not yet known whether giving brivanib together with cetuximab is more effective than cetuximab alone in treating patients with metastatic colorectal cancer.

OncoVax® Vaccinogen: OncoVax® is an autologous tumour cell vaccine and prepared for each patient using the patient's own surgically removed tumor. The active specific immunotherapy falls within the classification of Advanced Therapeutic Medicinal Product (ATMP). The patient received the first of four vaccinations several weeks after surgery. The vaccine consists of a portion of the tumor cells that has been thawed and combined with a proprietary formulation of BCG that serves as an immunogenic enhancer. This formulation is also used for the 2nd inoculation. The 3rd and the final booster inoculations are prepared the same way but without the addition of BCG. Phase 3a results demonstrated efficacy of Oncovax® in Stage II colon cancer patients with a statistically significant increased 5-year overall survival rate and increased recurrence-free survival by log-rank analysis. OncoVax® currently has a marketing authorization from Swissmedic, Switzerland's medical authority, in the category of "procedes therapeutiques". A pre-submission meeting to request Scientific Advice from the EMA for submission of a Conditional Marketing Autorization was done in December 2009.

Ramucirumab Eli Lilly + ImClone: Ramucirumab is an anti-VEGFR2 antibody blocking the binding of VEGF to its receptor. Ramucirumab is currently being tested for 2nd-line treatment in metastatic colorectal cancer in combination with FOLFIRI.

Market Data Colon Cancer

According to the American Cancer Society, colorectal cancer is the third most common form of cancer diagnosed in the United States, excluding skin cancers. It is estimated that over 142,570 people were diagnosed with some form of colorectal cancer with over 51,370 patients dying from colorectal cancer in 2010. Surgery is often the main treatment for early stage colorectal cancer. When colorectal cancer metastasizes (spreads to other parts of the body such as the liver), chemotherapy is commonly used. Treatment of patients with recurrent or advanced colorectal cancer depends on the location of the disease. Chemotherapy regimens (i.e. FOLFOX or FOLFIRI either with or without bevacizumab) have been shown to increase survival rates in patients with metastatic/advanced colorectal cancer. Currently, there are seven approved drugs for patients with metastatic colorectal cancer: 5-fluorouracil (5-FU), capecitabine (Xeloda®), irinotecan (Camptosar®), oxaliplatin (Eloxatin®), bevacizumab (Avastin®), cetuximab (Erbitux®), and panitumumab (Vectibix®). Depending on the stage of the cancer, two or more of these types of treatment may be combined at the same time, such as FOLFOX and FOLFIRI, or used after one another. Bevacizumab, a VEGF monoclonal antibody, is commonly

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administered with chemotherapy. Typically, patients who fail 5-FU, oxaliplatin, irinotecan, and bevacizumab-containing therapies, and who have wild-type KRAS status receive EGFR monoclonal antibody therapy with either cetuximab or panitumumab. Once patients progress on these agents, there are no further standard treatment options.

Perifosine Waldenstrom's Macroglobulinemia ("WM")

Results of a Phase 2 study on perifosine in patients with WM were presented in June 2008 at ASCO and in December 2008 during the ASH meeting. Thirty-six patients were evaluable for response. Perifosine showed clinical activity as a single agent in patients with relapsed/refractory WM, with an ORR (partial response ["PR"] + minimal response ["MR"]) of thirteen patients (36%). PR occurred in 2 patients (6%), with a median duration of response of 9+ and 18+ months, MR occurred in 11 patients (30%), with a median duration of response of 7 months (2-21+ months). SD occurred in 21 patients (58%) and progressive disease ["PD"] in 2 patients (6%) at 2 and 4 months. The most common adverse events were GI toxicities (nausea, vomiting and diarrhea) with grade 1 and 2 in 36% of the patients. Grade 3 and 4 events included anemia (9%) and leucopenia (9%). Grade 3 arthritis occurred in 9% of the patients; was considered likely related to therapy, (especially in rapidly responding patients), and reversed with symptomatic treatment as well as dose reduction. Dose reductions to 100 mg occurred in a total of 36% of the patients and were otherwise due to GI toxicity or cytopenias. Perifosine monotherapy induces a prolonged time to progression in relapsed or refractory WM, with a promising response rate of 36%, stabilization of disease in 58% of patients, and manageable toxicity, as well as the convenience of oral administration. Future clinical trials in combination with rituximab are planned.

In January 2010, we announced that an article entitled "*Clinical and Translational Studies of a Phase II Trial of the Novel Oral Akt Inhibitor Perifosine in Relapsed or Relapsed/Refractory Waldenstrom's Macroglobulinemia,*" reporting Phase 2 data demonstrating the single agent activity of perifosine for the treatment of advanced Waldenstrom's Macroglobulinemia, appeared in the February 1, 2010 issue of the Journal of Clinical Cancer Research. Dr. Irene Ghobrial, Assistant Professor of Medicine, Bing Center for Waldenstrom's Macroglobulinemia at Dana-Farber Cancer Institute, led the Phase 2 study, in which 37 patients were treated with perifosine 150 mg daily for 6 cycles. In this study, 41% of the patients had 3 or more lines of prior therapy and 78% had 2 or more prior lines of therapy. Such prior therapies include nucleoside analogues, bortezomib, alkylating agents and rituximab, which are not approved for, but are often used in the treatment of Waldenstrom's. Stable or responding patients were allowed to continue therapy until progression. Of the 37 patients, 4 achieved a partial response (11%), 9 achieved a minimal response (24%), and 20 showed stable disease (54%). Overall, 89% (33/37) of patients treated with single agent perifosine were reported to have stable disease or better, while 11% (4 patients) demonstrated progression. The median progression-free survival in the study was 12.6 months (90% C.I. (10.2, 22.7)), with a median overall survival of 26 months (90% C.I. (26 upper limit not reached)). Perifosine was generally well tolerated with gastrointestinal symptoms and fatigue reported as the most common adverse events related to therapy.

Perifosine Renal Cell Carcinoma ("RCC")

In June 2006, we announced positive data of perifosine in patients with advanced RCC. Keryx disclosed results from an interim analysis performed at the end of the first year of accrual, from a Phase 2, multi-center trial of perifosine that included multiple types of tumor and the results of the RCC group met protocol requirements for expansion of this cohort. Of the 13 patients with RCC, seven were evaluable for response. Three of them (43%) had a partial response and an additional two patients (29%) achieved long-term stable disease. Two patients (29%) had progressive disease. Results of a Phase 1 multicenter trial of perifosine in combination with sorafenib for patients with advanced cancers including RCC were disclosed by Keryx in June 2007 during the ASCO meeting and in November 2007. The trial was designed to accrue 3-6 patients in each of four cohorts. Response by

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RECIST criteria was a secondary endpoint. Perifosine was escalated from 50 mg once per day to 50 mg three times per day; sorafenib dose was escalated from 400 mg once per day to 400 mg twice per day; and sunitinib dose was escalated from 25 mg to 50 mg once per day for 4 weeks of treatment out of 6. DLT was defined as grade (G) 3 non-hematologic or G4 hematologic toxicity. MTD was the dose below that at which 2 out of 6 patients experienced a DLT.

For the combination perifosine + sorafenib, 20 patients were enrolled (12 males / 8 females, median age 64 (range 44-87)) with a median number of 2 prior therapies (range 1-4). Three patients were not evaluable due to rapid disease progression. Diagnosis was as follows; RCC (11 pts), sarcoma (5), colorectal (2), hepatocellular (1) and neuroendocrine (1). 17 patients were evaluable for toxicity: no drug-related Grade 4 adverse events (AE) were seen. Suspected DLT of hand-foot syndrome was seen in cohort 4 and additional patients were enrolled. There was no increase in hand-foot syndrome compared to sorafenib alone. Of interest, 6/9 evaluable RCC patients (67%) had SD >12 weeks (median 26 weeks, range 12-62+). One hepatocellular patient had SD for 36 weeks. The combination of perifosine + sorafenib was well tolerated with no increased hand-foot syndrome compared to sorafenib alone. Six out of 9 RCC patients (67%) achieved SD up to 62+ weeks.

For the combination perifosine + sunitinib, 14 patients (8 males / 6 females; media range 62 years old, range 28-81) were enrolled. Disease type was as follows: RCC (3), Sarcoma (3), Other (8). Six patients were evaluable for response. After 2 treatment cycles, one patient had a PR, 3 patients showed a SD and 2 patients had disease progression (PD). In the sub-group RCC, three out of three patients were evaluable for response: one patient had a PR, 1 patient showed a SD and 1 patient had a PD. Results indicated that patients to date have tolerated well the treatment combination of perifosine + sunitinib with no unexpected toxicities and clinical activity has been noted within the first 3 cohorts with 4 of 6 (67%) evaluable patients with advanced cancer achieving at least SD for more than 6 months.

Results from a Phase 2 trial of perifosine in patients with advanced RCC who have failed tyrosine kinase inhibitors (TKI) were also presented at the ASCO meeting in June 2009 by our partner Keryx. The goal of this multi-center Phase 2 trial was to determine the safety and efficacy of perifosine in patients with advanced RCC refractory to VEGFR TKI.

The study enrolled a total of 50 patients, of which 46 patients were evaluable for response. Evaluable patients were defined as those who had greater than 7 days of treatment. The primary endpoint of this study was clinical benefit, defined as response rate (RECIST), and PFS in RCC patients who failed a prior VEGF receptor inhibitor (sunitinib or sorafenib). Safety of perifosine in this patient population was evaluated as a secondary endpoint. The best response to single-agent perifosine was as follows:

Group	N	PR N (%)	SD > 12 wks N (%)	CBR* N (%)	Median PFS (SD or >)
All Pts	46	5 (11%)	16 (35%)	21 (46%)	33 weeks [95% CI (24, 60)]

* CBR: Clinical Benefit Rate defined as patients with Stable Disease or Partial Response

The median PFS for all 46 patients was 12.5 weeks [95% CI (11.9, 19)]. The median overall survival has not been reached with 33 of 46 patients (72%) still alive.

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Also of interest was the patient subgroup who had failed both a VEGF receptor inhibitor (sunitinib or sorafenib) and an mTOR inhibitor (either everolimus or temsirolimus). For this group, the best response and median PFS to single agent perifosine was as follows:

Group	N	PR N (%)	SD > 12 wks N (%)	CBR N (%)	Median PFS
VEGF + mTOR	16	1 (6%)	7 (44%)	8 (50%)	16 weeks [95% CI (11.7, 33.6)]

Three patients out of the group of patients previously treated with and failed both a VEGF and an mTOR inhibitor remain on active treatment, now out 5, 9 and 17 months.

Updated clinical results of this Phase II study of perifosine as a single-agent treatment for advanced metastatic RCC were presented in September 2009 at the 8th International Kidney Cancer Symposium. Those updated data included results from a subgroup of patients who failed both a VEGF receptor inhibitor (sunitinib or sorafenib) and an mTOR inhibitor (temsirolimus or everolimus). Evaluable patients (n=16) were defined as those who had greater than 7 days of treatment (2 additional patients withdrew consent within 7 days). Patients received 100 mg of perifosine daily until progression or unacceptable toxicity. The primary endpoint of this study was clinical benefit, defined as response rate (CR / PR by RECIST) or percent of patients progression-free for at least 3 months. Median PFS and overall survival were also analyzed for efficacy. Safety was a secondary endpoint. Perifosine was well-tolerated with the most common adverse events being gastrointestinal discomfort and fatigue. Best response to single agent perifosine was as follows:

N	PR N (%)	SD > 12 wks N (%)	PD 12 wks N (%)	Median PFS	Overall Survival
16	1 (6%)	7 (44%)	8 (50%)	16 wks [95% CI (11.7, 28)]	Not Reached (14/16 alive)
	Median PFS for patients SD or >			33 wks [95% CI (19, NR)]	at 22+ months

Perifosine Sarcoma

In June 2007, our partner Keryx presented results of Phase 1 and 2 studies for the treatment of patients with advanced sarcoma at the ASCO meeting. The dose schedules in the Phase 1 trials were weekly 100-800 mg or loading dose 300-1,800 mg on Day 1 followed by 50-150 mg daily for Days 2-21 every 28 days or loading dose 400-900 mg and daily 50-100 mg continuously. In the Phase 2 trial, doses were loading dose 900 mg on Day 1 and 150 mg daily for days 2-21 every 28 days; loading dose 900 mg and 100 mg daily continuously; 50 mg daily continuously without a loading dose; and 900-1,500 mg weekly. 145 patients with sarcoma were entered into studies and were assessed for CBR. Partial responses were seen, in one patient each, with chondrosarcoma, extra-skeletal myxoid chondrosarcoma, leiomyosarcoma and a desmoid tumor. At lower doses with 52 patients fully evaluable for CBR, the CBR was 52% with four partial responses and 23 stable diseases at ≥ 4 months. At higher doses with 30 patients fully evaluable for CBR, CBR was 53% with 16 stable diseases at ≥ 4 months. Toxicities were mainly gastrointestinal and/or fatigue. The percentage of patients with grade 0 nausea, vomiting, diarrhea and fatigue for lower dose perifosine (76 patients) was 46%, 49%, 38% and 55%, respectively, compared to 26%, 32%, 20%, and 58% for higher dose perifosine (69 patients). The proportion of patients with grade 2+ nausea, vomiting, diarrhea and fatigue was 20%, 13%, 15%, and 21% for lower dose perifosine and 49%, 35%, 42%, and 25% for higher dose perifosine.

In November 2007, our partner Keryx announced positive preliminary Phase 2 data of perifosine in patients with chemo-insensitive sarcoma. Data demonstrated the tolerability and clinical activity of perifosine as a single agent with an overall clinical benefit of 40% (stable disease > 3 months) in

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patients with refractory rare sarcomas. Perifosine was well tolerated with the most common grade 1 & 2 adverse events reported as nausea, vomiting, diarrhea and fatigue.

Perifosine Gliomas

In November 2007, our partner Keryx announced early results of a Phase 2 trial of perifosine as a single agent for the treatment of recurrent malignant gliomas (malignant glioblastoma and malignant anaplastic gliomas). Twenty-five patients with advanced malignant gliomas were treated with a loading dose of 600 mg (150 mg x4) followed by a 100 mg daily dose of perifosine. The median progression free survival and overall survival in the anaplastic glioma group was nine weeks (range 2-50 weeks) and 49 weeks, respectively. Toxicity was minimal with the following reported events: one grade 1 nausea, one grade 1 diarrhea, one grade 2 pain, and one grade 4 gout exacerbation. The study was designed to enroll at least 12 evaluable malignant glioblastoma patients and at least 10 evaluable malignant anaplastic gliomas patients. If at least one patient achieves six month progression free survival, the study would continue to enroll an additional subset of patients. Therefore, the malignant glioblastoma arm has been halted and the malignant anaplastic gliomas arm will continue to enroll.

Perifosine Neuroblastoma

On April 20, 2010 at the American Association for Cancer Research's ("AACR") annual meeting, we presented preclinical data that demonstrated that single agent perifosine targets activation of Akt in neuroblastoma cells and xenografts, significantly inhibited tumor growth *in vivo* and improved the survival of mice bearing neuroblastoma tumors.

On May 17, 2010, we announced the publication of an article in the May 12, 2010 issue of the *Journal of the National Cancer Institute* entitled "*In Vitro* and *In Vivo* Inhibition of Neuroblastoma Tumor Cell Growth by AKT Inhibitor Perifosine", demonstrating the single agent activity of perifosine in neuroblastoma tumor preclinical models.

On June 7, 2010, we announced that Phase 1 data for perifosine in recurrent pediatric solid tumors had been presented in the pediatric solid tumor poster discussion session held at the 46th annual ASCO meeting in Chicago. This study, conducted by the Memorial Sloan-Kettering Cancer Center pediatric group, marks the first time that perifosine has been administered in a pediatric patient setting.

This Phase 1 study of perifosine for recurrent pediatric solid tumors is a single center, open-label, dose-escalating study to assess safety, tolerability, pharmacokinetics ("PK"), and to identify any DLT of single agent perifosine in pediatric patients with any solid tumor that has failed standard therapy. Eleven patients (4 males, 7 females), at a median age of 13 years (5-18) were treated in this study to date. The following tumor types have been treated thus far: high-grade glioma (5), medulloblastoma (2), neuroblastoma (3), and ependymoma (1). Most patients were heavily pretreated with a median of three prior lines of therapy. Cohorts of three patients were treated at three dose levels of perifosine after a loading dose on day 1, and taking into account the drug's long half-life (t1/2 100 hours). No DLTs were observed at any of the three dose levels; dose level 4 is currently open for accrual. PK data thus far suggest similar drug absorption by pediatric patients relative to adult patients treated with single agent perifosine.

Of particular interest are the early signs of clinical activity observed in two of the three patients with Stage IV refractory neuroblastoma. Both patients were refractory to prior treatments upon entering the study and achieved stable disease for 48 weeks and 55+ weeks (ongoing). The investigators concluded that perifosine is well-tolerate