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-tolerated in children with recurrent solid tumors and that these early signals of activity warrant further investigation in patients with advanced neuroblastoma and select brain tumors. Previously, perifosine has been shown to target activation of Akt in neuroblastoma cells and xenografts and to significantly inhibit tumor growth *in vivo* and improve the survival of mice bearing neuroblastoma tumors.

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On July 14, 2010, our partner Keryx was granted orphan-drug designation by the FDA for perifosine for the treatment of neuroblastoma, a cancer of the nervous system affecting mostly children and infants for which there are no FDA-approved therapies.

Perifosine Other indications

On March 2, 2006, our partner Keryx announced the initiation of a corporate-sponsored Phase 2 trial, multi-cancer, clinical program to evaluate perifosine as a treatment for leukemia. Dr. Frank Giles, Professor, Department of Leukemia, at the MD Anderson Cancer Center in Houston, TX, is the principal investigator. This Phase 2 trial will assess the objective response rate and evaluate the pharmacokinetics and safety and tolerability of perifosine as a single agent in relapsed or refractory acute myeloid leukemia, acute lymphocytic leukemia, CLL, high-risk myelodysplastic syndrome and chronic myeloid leukemia in the blastic phase.

In November 2006, our partner Keryx presented intermediary results of the Phase 2 study of imatinib + perifosine in patients with imatinib-resistant gastrointestinal stromal tumor ("GIST"). The primary endpoint of this study is to evaluate the efficacy and toxicity of the combination imatinib and perifosine in patients with imatinib-resistant GIST. To date, 16 patients have been enrolled in the current study. Of the 12 patients with evaluable disease, there were two partial responses by Choi criteria (17% objective response rate ("ORR")) and one partial response by RECIST criteria (8% objective response rate). Grade 3 and 4 adverse events were rare and included fatigue, myalgias, ocular toxicity and nausea/emesis. The early data from the current study suggest that the addition of perifosine to imatinib is well-tolerated and may have efficacy in the treatment of patients with imatinib-resistant GIST.

Updated results of this trial were presented in June 2009 by our partner Keryx during the ASCO meeting. Patients with Kit (+) advanced GIST who have progressed on imatinib were eligible. Patients continued their current dose of imatinib and were randomized to one of two dosing schedules of perifosine (Arm A: 100 mg p.o. qd × 28 + imatinib or Arm B: 900 mg [300 mg p.o. tid] qweekly + qd imatinib). A Bayesian approach was utilized to assess a target response rate of 20% with an unacceptable toxicity rate of 15% or less. Response was measured every 8 weeks by RECIST and Choi criteria. The primary endpoint was to determine the efficacy of perifosine with imatinib in patients with advanced GIST who progressed while receiving imatinib. 41 patients were enrolled from August 2005 to July 2008. After 1 patient exclusion and 2 cross-overs, 22 patients were in Arm A and 18 patients in Arm B. Median age was 58 (range, 32-82), 51% were male, and median ECOG performance status was 1. The most common primary site of disease and metastasis was the stomach (29%) and liver (66%), respectively. KIT genotype was available for 22 patients (54%); 5(12%) WT, 13(32%) exon 11 mutations, and 4(10%) exon 9 mutations. The median number of cycles was 2 (range, 1-24). By Choi and RECIST, 30 patients (73%) and 36 patients (87%) were available for response, respectively. No CR was identified but the PR rate was 4/36 (11%) by Choi (4 PR, 9 SD) and 0/36 (0%) by RECIST (16 SD). 4/5 (80%) of patients with WT KIT appeared to benefit (Choi: 1 PR, 3 SD; RECIST: 4 SD). Median PFS and OS for 40 patients were 2.2 months and 18.3 months. No difference in PFS was noted for the 2 schedules. Toxicity was assessed in 39 patients; 46 grade 3 events and 4 grade 4 events (ALT elevation, blurred vision, fatigue, and mood alteration) were noted. The most common grade 3 event was fatigue (20%). Three patients (7%) were removed from the study for toxicity (Arm A:1 patient, Arm B:2 patients).

On July 14, 2009, our partner Keryx announced the initiation of a Phase 1 clinical study to evaluate perifosine as a single agent treatment for recurrent solid tumors in pediatric patients. This single-center open-label study, fully funded by an external grant provided by a private organization, will be conducted at Memorial Sloan-Kettering Cancer Center in New York City. Oren Becher, MD, Instructor, Department of Pediatrics, in coordination with Eric Holland, MD, PhD, Director of the Brain Tumor group at Memorial Sloan-Kettering Cancer Center, will act as the study's Principal

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Investigator. Perifosine is being evaluated as a single-agent in pediatric patients with any solid tumor that has failed standard therapy. Patients up to 18 years of age with a performance status of greater than 40% are eligible for this study. The study was designed as a dose escalation study to determine the MTD of perifosine alone in recurrent/progressive pediatric tumors. A standard 3+3 dose escalation design will be employed with 3 to 6 patients at each dose level. All patients will receive perifosine at a loading dose on the first day, followed by a maintenance dose to start on day two until progression of disease. A minimum of 4 and a maximum of 24 patients will be required to complete the study.

On October 8, 2009, our partner Keryx announced the initiation of a Phase 2 clinical study to evaluate perifosine as a single agent treatment for relapsed or refractory CLL and Small Lymphocytic Lymphoma (SLL). This externally funded Phase 2 study was designed by Daphne Friedman, M.D., Instructor and Principal Investigator, in coordination with J. Brice Weinberg, Professor, and Mark Lanasa, Assistant Professor, Divisions of Medical Oncology and Hematology, Duke University Medical Center, and is open for enrollment at Duke University. The single-center, open-label, study entitled, "Phase 2 Trial of Perifosine in Patients with Relapsed or Refractory Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma", will enroll approximately 30 patients. Perifosine will be given orally at a dose of 50 mg twice daily, for a total of six 28-day cycles. The patients will be formally restaged upon completion of the trial. Overall Response Rate is the primary endpoint with overall survival, progression-free survival and safety as secondary endpoints. Correlative studies will also be conducted and evaluated as a secondary endpoint.

On November 17, 2010, at the EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics in Berlin, Germany, we presented an abstract on perifosine combined with antimetabolites which induces synergistic effects on cytotoxicity and apoptosis in human colon, multiple myeloma, breast, renal, and liver tumor cell lines.

On December 6, 2010 at the ASH's 52nd annual meeting in Orlando, Florida, we announced positive safety and tolerability Phase 2 data for perifosine in patients with advanced lymphoma.

In the first Phase 2 study related to CLL, 12 patients with advanced CLL began treatment with single agent perifosine at 50 mg BID. The patients on the study were heavily pre-treated having had a median of four prior lines of therapy with 75% of patients classified as Rai Stage IV. One patient achieved a partial response (5 months on treatment) and 5 additional patients achieved stable disease (median duration of 4.25 months), for an overall 50% clinical benefit rate (PR + SD). Perifosine was well tolerated with minimal dose modifications.

In the second study presented, 26 patients were enrolled in a Phase 2 study with advanced lymphoma (6 non-Hodgkin's lymphoma, 4 CLL, 1 Waldenstrom's Macroglobulinemia and 15 HL). 73% of patients were previously refractory to their prior therapy, with 85% of patients having had 4 or more prior therapies. Perifosine (50 mg BID) was started as a single agent for 28 days; after 28 days, patients achieving PR or better were continued on single agent perifosine. Patients achieving less than a PR were given the combination of perifosine (50 mg BID) + sorafenib (Nexavar®) at 400 mg BID. All of the 4 CLL patients in this study achieved a partial response on single-agent perifosine within one month of treatment and remained on perifosine single agent. Response durations for each of the 4 patients were 4, 8, 9+ and 12 months. The remaining 22 patients were administered the combination with sorafenib, where 5 of the 15 (33%) HL patients achieved a partial response with a median response duration of 9 months. An additional 6 patients receiving the combination (40%) achieved stable disease. The combination was well tolerated with no unexpected safety events. The investigators concluded that perifosine in combination with sorafenib has significant anti-lymphoma activity in relapsed/refractory HL, and that perifosine as a single agent induced prolonged responses in high-risk, heavily pretreated CLL patients.

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Perifosine Radio-enhancer

A proof-of-concept Phase 1 study of perifosine in combination with radiotherapy conducted by the National Cancer Institute of the Netherlands was completed in 2004. Results from this trial were presented at ASCO 2004. A total of 21 radiotherapy-naïve patients, of whom 17 had advanced non-small cell lung cancer ("NSCLC") and 14 had become refractory to prior chemotherapy, received oral perifosine doses ranging from 50 mg to 200 mg/day concurrently with standard doses of radiotherapy. The trial data demonstrated an acceptable safety and tolerability profile, with 150 mg/day established as the dose recommended for use in subsequent clinical trials. Also demonstrated was preliminary evidence of anti-tumor activity at all dosage levels, including complete or partial responses (complete disappearance and decreased tumor size, respectively), or stable disease, with a median follow-up for responders of eight months. Importantly, in the cohort of 10 patients who were treated with 150 mg/day, the established dose recommended for use in subsequent clinical trials, there were three complete responses, three partial responses and four patients with stable disease.

On September 22, 2005, we announced the initiation of a multi-center Phase 2 randomized, double-blind, placebo-controlled trial with perifosine in combination with radiotherapy for NSCLC. Patients received perifosine 150 mg daily for five to six weeks and were followed for at least 12 months. The primary endpoint of this trial was the extent and duration of local control, i.e., the absence of tumor recurrence or progression in the area that has been irradiated. The trial was conducted in collaboration with the Netherlands Cancer Institute. The lead investigator is Marcel Verheij, M.D., Ph.D., of the Department of Radiation Oncology / Division of Cellular Biochemistry, at the Netherlands Cancer Institute in Amsterdam. We announced completion of recruitment of 160 patients with inoperable Stage III NSCLC on November 14, 2007.

We disclosed preliminary results for this European multi-center Phase 2 trial in NSCLC in June 2009. Starting one week before the onset of a 4-week course of radiotherapy (51 Gy in 17 fractions), 177 patients with non-metastatic but inoperable NSCLC, mainly Stage III, received a 5-week course of 150 mg perifosine daily or placebo. After end of radiotherapy, patients were followed up to determine the time to tumor recurrence or progression in the area that had been irradiated, the so-called "local control". The primary endpoint of this trial was the extent and duration of local control, specifically the proportion of patients with absence of recurrence or progression 12 months after the end of treatment. The study was planned under the basic assumption that radiotherapy alone would result in a 35% local control rate, one year after end of therapy in the placebo group. It was hypothesized that the addition of perifosine would sensitize tumor cells to the tumor-killing effect of the radiotherapy, leading to a 15% higher rate of local control. Secondary efficacy parameters included the times to loco-regional or distant/systemic failure, the tumor response rate, and overall survival. Safety investigations included the monitoring of clinical laboratory, electrocardiograms, lung function, and adverse events.

In all, 22 study sites in The Netherlands, Bulgaria, Romania, Macedonia, and Belarus participated in this trial. A total of 177 patients were randomized and treated, of whom only 26 reached the milestone of one year post-treatment follow-up without disease relapse or progression, 14 of 95 patients (14.7%) in the perifosine and 12 of 82 patients (14.6%) in the placebo control group. No difference between treatment groups could be shown for local, loco-regional and overall disease control. Also, the tumor response rate, as assessed after the end of the radiotherapy, was not different between the groups.

In contrast to the lack of an observed local effect, patients in the perifosine group, particularly the subgroup of patients who entered the study without prior chemotherapy, showed a trend towards longer survival than patients of the placebo control group despite the short duration of treatment (5-week course of 150 mg perifosine daily).

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There were no safety signals that would lead to an amendment of the current safety data or risk benefit assessments of perifosine. The type and severity of side effects were in the expected range. Following these neutral results and an unchanged safety profile, we announced that we will concentrate our efforts for perifosine on the disease targets of both multiple myeloma and metastatic colon cancer.

Partners for perifosine

A Cooperative Research and Development Agreement ("CRADA") was put in place with the National Institute of Health/the National Cancer Institute in May 2000. A cooperation and license agreement was signed in September 2002 with Access Oncology, Inc. ("AOI"), for the use of perifosine as an anti-cancer agent covering the United States, Canada and Mexico. In January 2004, AOI was acquired by Keryx, which is pursuing the clinical development of perifosine under the same conditions as AOI. The agreement, in particular, provides us free access to all data from Keryx and its partner's studies, as well as milestone payments and scale-up royalties to be paid to us on future net sales of perifosine in the United States, Canada and Mexico. In April 2009 we entered into an agreement to out-license the rights of perifosine to Handok in South Korea. On March 9, 2011, we announced that we had entered into an agreement with Yakult for the development, manufacture and commercialization of perifosine in all human uses, excluding leishmaniasis in Japan. Under the terms of this agreement, Yakult made an initial up-front payment to us of €6 million ($8.3 million). Also per the agreement, we will be entitled to receive up to a total of €44 million ($60.9 million) upon achieving certain pre-established milestones, including clinical and regulatory events in Japan. Furthermore, we will be entitled to receive double-digit royalties on future net sales of perifosine in the Japanese market. We own rest of the world rights to perifosine.

AEZS-127 erucylphosphocholine

On January 6, 2005, we announced the initiation of preclinical development of erucylphosphocholine (AEZS-127), an analog of perifosine which is suitable for i.v. administration. Like perifosine, AEZS-127 belongs to a new class of compounds based on alkylphosphocholines. AEZS-127 possesses distinctive reduced haemolytic activity thus allowing for i.v. injection.

On January 6, 2005, we also licensed to Keryx certain rights to develop and market AEZS-127 in North America, South Africa, Israel, Australia and New Zealand while keeping rights for the rest of the world. According to the agreement with Keryx, the preclinical development costs of AEZS-127 are shared between Keryx (50%) and us (50%). In the fourth quarter of 2008, we repatriated all rights for AEZS-127 from Keryx.

In 2006, studies for acute toxicity and dose range finding of erucylphosphocholine were actively pursued. The 4-week toxicity studies in rats and dogs as well as the safety pharmacology package was completed in 2007. These preclinical data are a prerequisite for the performance of a Phase 1 clinical study.

Erk/PI3K inhibitors and dual kinase inhibitors

In addition to our activities with alkylphosphocholines, we are screening small molecules for activity as agonists and antagonists to lipid-protein signaling interactions, which are seen as new and potentially important therapeutic targets.

We are focusing our efforts on single and dual inhibitors of Ras-Raf-Mek-Erk and PI3K-Akt pathways. The Ras-Raf-Mek-Erk and the PI3K-Akt pathways are constitutively activated in many cancer types, and influence both tumor development and progression.

Both signaling pathways represent promising therapeutic targets for the treatment of tumors. We have now identified a new compound class with inhibitory activity against both the Erk and PI3K

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kinases. These small molecules inhibit the kinases at nanomolar concentrations in a dose-dependent manner by competing directly at the ATP binding site. In a broad kinase panel, the molecules are very selective against other kinases. In cellular experiments the compounds inhibit the activation of downstream targets Akt and Rsk1, and can stop the proliferation of various human cancer cell lines. Moreover, a new generation of aniline-substituted pyridopyrazine-urea derivative shows highly selective PI3K inhibition. We are currently performing *in vivo* studies with front-runner compounds in four mouse xenograft models (HCT116, U87, A549 and PC3) as well as pharmacokinetic studies in rodents using an oral pre-formulation. On the basis of these studies, AEZS-126 was selected as a preclinical development candidate for *in vivo* pharmacology and pharmacokinetic studies.

AEZS-126

The first *in vitro* and *in vivo* data for AEZS-126 were presented in April 2009 at the AACR meeting. The first poster, entitled, "AEZS-126, a new orally bioavailable PI3K inhibitor with antitumor effects", focuses on ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) and safety profiling of the compound, as well as *in vivo* pharmacokinetic experiments and mouse xenograft antitumor studies. Results indicated that AEZS-126 was identified as a potent inhibitor of class I PI3Ks in biochemical and cellular assays and demonstrated favorable properties in early *in vitro* ADMET screening including microsomal stability, plasma stability and screening against a large safety profile composed of receptors, enzymes and cardiac ion-channels. During the course of *in vivo* pharmacokinetic experiments and mouse xenograft antitumor studies, the oral bioavailability in mice was determined to be about 60%, leading to micromolar plasma levels which are well above the nanomolar IC50 values *in vitro* studies. Significant antitumor activity was observed at 30 mg/kg daily oral administration in Hct116 and A549 models. These data suggest that AEZS-126 is a promising compound for clinical intervention of the PI3K/Akt pathway in human tumors.

The 2nd poster, entitled "*In vitro* profiling of the potent and selective PI3K inhibitor, AEZS-126", outlines the key *in vitro* characteristics of this compound that led to its selection for *in vivo* development. AEZS 126 inhibits PI3Ka with an IC50 value of 10nM and proved to be a potent inhibitor of Akt phosphorylation in cellular assays. Mode-of-action studies showed that AEZS-126 acts as an ATP competitive compound. The *in vitro* antiproliferative activity against different human tumor cell lines (MDA-MB 468, U87, Hct116, PC-3, A549 and others) was determined, with EC50 values in the nanomolar range. Based on those results presenting a favorable *in vitro* pharmacologic profile for AEZS-126, further *in vivo* profiling experiment will be performed.

AEZS-129

On April 21, 2009, we presented two posters on AEZS-129, a promising compound for clinical intervention of the PI3K/ Akt pathway in human tumors, at the AACR Annual Meeting. *In vivo* and *in vitro* data showed significant antitumor activity and a favorable *in vitro* pharmacologic profile which could lead to further *in vivo* profiling.

On November 17, 2010, we presented a poster on encouraging preclinical results for AEZS-129, a novel orally active compound with anti-tumor effects, at the 22nd EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics in Berlin, Germany. AEZS-129 has been identified as a highly potent and selective inhibitor of PI3K. The compound inhibits the PI3K/Akt signaling pathway both *in vitro* and *in vivo* and leads to growth inhibition of tumor cells. The compound was well tolerated during the 4 week treatment period and showed substantial tumor growth inhibition in different mouse xenograft tumor models.

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AEZS-132

On April 20, 2010, at the AACR's annual meeting we presented data on our dual Erk/PI3K inhibitors and on our selective Erk inhibitors. Data supported further evaluation of selective Erk inhibitors as antiproliferative agents, either as monotherapy or in combination with inhibitors of the PI3K/Akt pathway. Other data resulted in the identification of AEZS-132, a unique dual inhibitor of PI3K and Erk with a favourable pharmacology and ADMET profile, for further evaluation as an antitumor agent.

On November 17, 2010, at the EORTC-NCI-AACR meeting, we presented a poster on AEZS-132, the first-in-class dual PI3K/Erk inhibitor being selected as the optimized lead compound for further development. The compound is a unique orally active low molecular weight dual PI3K/Erk inhibitor derived from Aeterna Zentaris' medicinal chemistry program. Due to its dual PI3K and Erk inhibition, a broad anti-tumor activity is expected in tumors with over-activation of both pathways. AEZS-132 demonstrated prolonged plasma exposure when given orally in mice. Significant tumor inhibition resulted from mouse xenograft models with human colon, endometrium and lung tumors.

TUMOR TARGETING CYTOTOXIC CONJUGATES AND CYTOTOXICS

Cytotoxic conjugates

In view of the non-specific toxicity of most chemotherapeutic agents against normal cells, targeting such drugs to cancerous tissue offers a potential benefit for patients with advanced or metastatic tumors. Targeted cytotoxic peptide conjugates are hybrid molecules composed of a cytotoxic moiety linked to a peptide carrier which binds to receptors on tumors. Cytotoxic conjugates are designed to achieve differential delivery, or targeting, of the cytotoxic agent to cancer vs. normal cells.

Our cytotoxic conjugates represent a novel oncological strategy to control and reduce toxicity and improve the effectiveness of cytotoxic drugs. The development strategy was to create targeted conjugates with high cytotoxic activity based on doxorubicin, an approved and commercialized product or 2-pyrrolino-doxorubicin which is 500 to 1,000 times more active than the parent compound. We are exploring several candidates in which doxorubicin or 2-pyrrolino-doxorubicin are coupled to the peptide carriers targeting LHRH (AEZS-108 & AN-207), somatostatin (AN-238 & AN-162) or bombesin (AN-215) receptors. These conjugates are less toxic and more effective *in vivo* than the respective radicals in inhibiting tumor growth in LHRH receptor positive models of human ovarian, mammary or prostatic cancer.

In AEZS-108, the most advanced of the cytotoxic conjugates, doxorubicin is chemically linked to an LHRH agonist, a modified natural hormone with affinity for the LHRH receptor. This design allows

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for the specific binding and selective uptake of the cytotoxic conjugate by LHRH receptor positive tumors. Potential benefits of this targeted approach include a more favorable safety profile with lower incidence and severity of side effects, as normal tissues are spared from toxic effects of doxorubicin. In addition, the targeted approach may enable treatment of LHRH receptor positive cancers that have become refractory to doxorubicin which has been administered in its non-targeted form.

In preclinical studies conducted to date in several animal models of LHRH receptor positive human cancer cell lines, AEZS-108 anti-tumor activity and tolerability were shown to be superior to that of doxorubicin. As would be expected, AEZS-108 was not active or was significantly less active than doxorubicin in LHRH receptor negative cancer cell lines. On January 18, 2005, we announced the initiation of a company-sponsored Phase 1 dose-ranging study with the targeted anti-cancer agent AEZS-108.

In June 2006, we announced positive Phase 1 results for AEZS-108 in patients with gynaecological and breast cancers which showed that the compound has a good safety profile and no dose-limiting toxicities. Eight patients received AEZS-108 by i.v. infusion. Infusion was well tolerated at all dosages, without supportive treatment. Pharmacokinetic analyses showed dose-dependent plasma levels of AEZS-108 and only minor (10-20%) release of doxorubicin. Stabilization of disease was observed in one out of eight patients in the ongoing Phase 1 study.

On November 27, 2006, we disclosed additional positive Phase 1 results regarding AEZS-108 in patients with gynaecological and breast cancers. Further data showed the compound's good safety profile and established the maximum tolerated dose at 267 mg/m^2, which is equimolar to a doxorubicin dose of 77 mg/m^2. This dose will be the recommended dose for a Phase 2 trial. The Phase 1 open-label, multi-center, dose-escalation, safety and pharmacokinetic study conducted in Europe included 17 patients suffering from breast, endometrial and ovarian cancers with proven LHRH receptor status. Evidence of anti-tumor activity was found at 160 mg/m^2 and 267 mg/m^2 doses of AEZS-108, where 7 out of 13 patients showed signs of tumor response, including 3 patients with complete or partial responses. The Phase 2 trials will focus on advanced or recurrent ovarian and endometrial cancers, two forms of cancer where LHRH receptors are highly expressed. Recommended dose will be 267 mg/m^2 given once every three weeks.

AEZS-108 Ovarian and Endometrial Cancer

In 2007, a Phase 2 open-label, non-comparative, multicenter two indication trial stratified with two stages Simon Design was prepared. The study involved 82 patients with up to 41 patients with either a diagnosis of platinum-resistant ovarian cancer (stratum A) or disseminated endometrial cancer (stratum B). Under coordination by Prof. Günter Emons, MD, Chairman of the Department of Obstetrics & Gynaecology at the University of Göttingen, Germany, this open-label, multi-center and multi-national Phase 2 study "AGO-GYN 5" was being conducted by the German AGO Study Group (Arbeitsgemeinschaft Gynäkologische Onkologie / Gynaecological Oncology Working Group), in cooperation with clinical sites in Europe. In patients with tumors expressing LHRH receptors an i.v. infusion of AEZS-108 (267 mg/m^2) was administered over a period of 2 hours, every Day 1 of a 21-day (3-week) cycle. The proposed duration of the study treatment was 6 courses of a 3-week cycles. The study was performed with 14 centers of the German Gynaecological Oncology Working Group, in cooperation with 3 clinical sites in Europe. The primary efficacy endpoint at the end of Stage II was defined as 5 or more patients with partial or complete tumor responses according to RECIST and/or Gynaecologic Cancer Intergroup (GCIG) guidelines. Secondary endpoints included time to progression, survival, toxicity, as well as adverse effects. On February 12, 2008, we reported that the treatment of first patients had commenced in this Phase 2 trial. In October 2008, we announced that we had entered the second stage of patient recruitment for the Phase 2 trial in platinum-resistant ovarian cancer indication. This decision was taken following the report of two partial responses among patients with a diagnosis of platinum-resistant ovarian cancer. The second stage of patient recruitment for the

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endometrial cancer indication was reached in November 2008 and was based on the report of one complete response and two partial responses among 14 patients with a diagnosis of disseminated endometrial cancer.

In November 2009, we announced preliminary positive efficacy data from this Phase 2 study in patients with platinum-resistant and taxane-pretreated ovarian cancer. In a personalized healthcare approach, the study selected patients with tumors expressing LHRH receptors, the key element in the targeting mechanism of AEZS-108. All 42 patients with LHRH-receptor positive ovarian cancer who entered study AGO-GYN 5 had completed their study treatment. A preliminary evaluation showed that the study met its primary efficacy endpoint of 5 or more responders in 41 evaluable patients. Responders, as well as patients with stable disease after completion of treatment with AEZS-108, were to be followed to assess the duration of progression-free survival and, ultimately, overall survival.

In November 24, 2009, we announced positive efficacy data from the Phase 2 study with the targeted cytotoxic peptide conjugate, AEZS-108, in patients with advanced or recurrent endometrial cancer. A preliminary evaluation showed that the study AGO-GYN 5 had met its predefined primary efficacy endpoint of 5 or more responder patients with endometrial cancer. Responders, as well as patients with stable disease after completion of treatment with AEZS-108, were to be followed to assess the duration of progression-free survival and, ultimately, overall survival.

On May 6, 2010, we announced that we had received orphan drug designation from the FDA for AEZS-108 for the treatment of ovarian cancer. Orphan drug designation is granted by the FDA's Office of Orphan Products Development 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 a 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 or efficacy versus another drug of its type previously granted the designation for the same indication.

On May 17, 2010, we announced that we had received a positive opinion for orphan medicinal product designation from the COMP of the EMA for AEZS-108 for the treatment of ovarian cancer. 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 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 European Union for AEZS-108, once approved for the treatment of ovarian cancer.

On June 7, 2010, Prof. Günter Emons, Chairman, Department of Obstetrics & Gynaecology Georg-August University Göttingen, Germany, presented positive efficacy and safety data for AEZS-108 in ovarian cancer at the ASCO Annual Meeting. The poster (abstract #5035) entitled "Phase 2 study of AEZS-108, a targeted cytotoxic LHRH analog, in patients with LHRH receptor-positive platinum resistant ovarian cancer" (G. Emons, S. Tomov, P. Harter, J. Sehouli, P. Wimberger, A. Staehle, L. C. Hanker, F. Hilpert, P. Dall and C. Gruendker, for the AGO Study Group), details the use of AEZS-108 in women with histologically confirmed taxane-pretreated platinum-resistant/refractory LHRH receptor-positive advanced (FIGO III or IV) or recurrent ovarian cancer. Patients received a recommended dose of 267 mg/m2 by intravenous infusion over 2 hours, with retreatment every 3 weeks, for up to 6 courses. Response rate (RECIST and/or GCIG criteria) was defined as the primary endpoint. Secondary endpoints were safety, time-to-progression and overall survival.

Forty-two patients with platinum-resistant ovarian cancer entered the study. Efficacy included partial response in 5 patients (11.9%) and stable disease for more than 12 weeks in 11 patients

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(26.2%). Based on those data, a CBR of 38% can be estimated. Median time to progression and overall survival were 3.5 months (104 days) and 15.6 months (475 days), respectively. Overall survival compares favourably with data from Doxil and Topotecan (8-9 months). In all, tolerability of AEZS-108 was good and commonly allowed retreatment as scheduled. Only one patient (2.4%) had a dose reduction, and overall, 25 of 170 (14.7%) courses were given with a delay, including cases in which delay was not related to toxicity. Severe (Grade 3 or 4) toxicity was mainly restricted to rapidly reversible hematologic toxicity (leukopenia / neutropenia) associated with fever in 3 cases. Good tolerability of AEZS-108 was also reflected with only a few patients with non-hematological toxicities of grade 3 (none with Grade 4), including single cases (2.4%) each of nausea, constipation, poor general condition, and an enzyme elevation. No cardiac toxicity was reported.

On November 18, 2010, Prof. Günter Emons of the Department of Obstetrics & Gynaecology Georg-August at the University of Göttingen (Germany) presented positive data for the Phase 2 of AEZS-108 in advanced endometrial cancer at the EORTC-NCI-AACR symposium in Berlin, Germany. The study showed encouraging results as AEZ-108 was used as a single agent.

Of 43 patients treated with AEZS-108 in this study, 39 were evaluable for efficacy. Responses confirmed by independent review included 2 patients with complete response (CR; 5.1%), 10 patients with partial response (PR; 25.6%), and 17 patients with stable disease (SD; 43.6%). Based on those data, an overall response rate (ORR = CR+PR) of 30.8% and a clinical benefit rate (CBR = CR+PR+SD) of 74.4% can be estimated. Responses were also achieved in patients with prior chemotherapy, 1 CR, 1 PR and 2 SDs in 8 of the patients pre-treated with platinum/taxane regimens. Median time to progression and overall survival were 7 months (30 weeks) and 14.3 months (62 weeks), respectively. Conclusions from this trial were as follows:

- AEZS-108 at a dosage of 267 mg/m2 every 3 weeks was active and well tolerated in patients with endometrial cancer;
- hematological toxicity was rapidly reversible, and non-hematological toxicities were usually not severe, causing few deviations from scheduled treatment;
- the objective response rate of 30.8% compares well with those of single agent platinum or taxane treatment; responders included patients pre-treated with platinum/taxane combination; and
- in addition, the rate of stable disease was 43.6%, resulting in a CBR of 74.4%;

The overall survival after single agent AEZS-108 is similar to that reported for modern triple combination chemotherapy, but was achieved with lower toxicity.

Competitors for AEZS-108 in Ovarian Cancer Indication

Products on the market:

Major products available on the market for the treatment of resistant or refractory ovarian cancers:

Doxil® (pegylated liposomal doxorubicin manufactured by Schering Plough) a topoisomerase II inhibitor and DNA intercalating, is approved for 2nd-line treatment in women with advanced ovarian cancer who have failed a 1st-line platinum based chemotherapy regimen.

Gemzar® (Gemcitabine manufactured by Eli Lilly) is a deoxycytidine analogue, a pyrimidine antimetabolite related to cytarabine. The drug exhibits cell phase specificity, killing cells undergoing DNA synthesis. Gemcitabine is a prodrug and is metabolized intra cellularly to the active di-phosphate and tri-phosphate nucleosides. A small increase in the share of gemcitabine and carboplatin combination second line treatment in platinum sensitive population who has suffered paclitaxel-associated neurological toxicity from 1st-line treatment is expected. A decrease in sales of gemcitabine of under $17 million in 2009 to under $9 million in 2019 is expected.

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Yondelis® (Trabectedin manufactured by PharmaMar/Centocor Ortho Biotech's Yondelis) is a tris, tetrahydroisoquinoline alkaloid which binds to the major groove of DNA, generating the formation of lethal DNA strands which causes cell cycle arrest and apoptosis. Trabectedin is used in 2nd-line therapy in combination with Doxil® in relapsed platinum sensitive ovarian cancer. The drug was approved in Europe in September 2009. European sales of Trabectedin are estimated to be under $22 million in 2019 as the drug has a poor toxicity profile.

Hycamtin® (Topotecan manufactured by GlaxoSmithKline) is a topoisomerase I inhibitor used for the treatment of patients with metastatice carcinoma of the ovary after failure of 1st-line or subsequent therapy. The drug was approved by the FDA in 2007.

Products in Phase 3 development:

Farletuzumab (MORAb-003 manufactured by Esai/Morphotek) is a folate receptor inhibitor targeting folate receptor alpha which is over-expressed on a number of epithelial-derived cancers such as ovarian cancer. A Phase 3 study of MORAb-003 in subjects with platinum sensitive ovarian cancer in first relapse started in 2009 and is currently recruiting patients. MORAb-003 received orphan drug designation by the FDA. Farletuzumab is expected to be approved for 2nd-line treatment of platinum sensitive ovarian cancer in the U.S. and Europe in 2013 and Japan in 2015. Approval for platinum resistant and refractory ovarian cancer is estimated for 2016 in U.S. and Europe.

Avastin® (Bevacizumab manufactured by Roche/Genentech) is a humanized monoclonal antibody targeting vascular endothelial growth factor. Off label sales of bevacizumab in the 3rd-line treatment will remain small but almost stable over the period 2009 to 2019 increasing from $1.4 million to $1.7 million.

Market Data Ovarian Cancer

According to Decision Resources February 2011, the number of ovarian cancer drug treatable populations in the major markets (U.S., Europe G5 and Japan) was mentioned as being 117,466 for the year 2010 and the number of total incident cases was mentioned as being 57,340 for 2010.

Competitors for AEZS-108 in Endometrial Cancer Indication

At present, there is no approved drug product for the treatment of advanced and recurrent metastatic endometrial cancer in the U.S. and Europe. There is also no systemic therapy approved in the U.S. and Europe for treating advanced or recurrent endometrial cancer.

Letrozol Novartis: Letrozol is a non-steroidal aromatase inhibitor which completed Phase 2 clinical development in the treatment of advanced or recurrent hormone receptor positive endometrial cancer.

A Phase 2 study was performed in collaboration with Sanofi Aventis investigating how well carboplatin and docetaxel followed by radiation therapy works in treating patients with Stage III, Stage IV or recurrent endometrial cancer.

XL-147 Exelixis: XL-147 is a potent and highly selective inhibitor of the class I PI3K family of lipid kinases which targets the PI3K/PTEN pathway. The drug is currently in phase 2 development for treatment of advanced or recurrent endometrial cancer.

Market Data Endometrial Cancer

According to the American Cancer Society, an estimated 43,470 cases of endometrial cancer were expected to be diagnosed in 2010 and 7,950 deaths were expected during the same year.

AEZS-108 Prostate and Bladder Cancer

In May 2009, we announced at the ASCO meeting the results supporting the evaluation of AEZS-108 in prostate cancer. Expression of LHRH receptors was determined using immunohistochemistry and the intensity was graded on a scale from zero to 3. The expression was

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analyzed in three cohorts of patients: (1) 47 men with localized prostate cancer treated with radical prostatectomy with no hormone therapy, (2) 61 men with localized prostate cancer treated with neoadjuvant LHRH agonists for varying duration prior to prostatectomy, and (3) 22 men with metastatic prostate cancer who received a palliative transurethral resection of the prostate after clinical progression. In the final cohort, 15 men were treated with castration and 7 were treated with LHRH agonists. 45 of 47 hormone naïve samples (95.7%) demonstrated LHRH receptor expression. Statistical analysis revealed a correlation between strong receptor expression and higher pathologic tumor stage as well as shorter overall survival. 60 of 61 samples treated with neoadjuvant LHRH agonist therapy (98.4%) demonstrated LHRH receptor expression. All 22 samples from patients with metastatic disease demonstrated LHRH receptor expression. The majority of these samples demonstrated moderate to strong intensity. LHRH receptors are expressed on prostate cancers cells of hormone naïve and castrated patients. The expression of these receptors appears to persist despite prolonged treatment with LHRH agonists. The new results show continued expression of LHRH receptors in prostate cancer specimens after prolonged use of LHRH agonists. These data provide further support to the investigation of the drug in hormone-refractory prostate cancer, a major genitourinary cancer indication in male patients.

On May 12, 2010, we announced that the FDA had approved our Investigational New Drug application ("IND") application for AEZS-108 in LHRH receptor-positive urothelial (bladder) cancer. Following this approval from the FDA, this trial will be conducted at the Sylvester Comprehensive Cancer Center at the University of Miami's Miller School of Medicine, and will include up to 64 patients, male and female, with advanced LHRH receptor-positive urothelial (bladder) cancer. The study will be conducted in two parts: first, a dose-finding part in up to 12 patients; subsequently, a selected dose will be studied for its effect on progression-free survival.

On August 5, 2010, we announced that the NIH had awarded Dr. Jacek Pinski, Associate Professor of Medicine at the Norris Comprehensive Cancer Center of the University of Southern California, a grant of approximately $1.5 million over three years to conduct a Phase 1/2 study in refractory prostate cancer with AEZS-108. The study, entitled "A Phase I/II Trial of AN-152 [AEZS-108] in Castration- and Taxane-Resistant Prostate Cancer", will enroll up to 55 patients and will be conducted in two portions: an abbreviated dose-escalation followed by a single arm, Simon Optimum two-stage design Phase 2 study using the dose selected in the Phase 1 portion. The primary objective of the Phase 2 portion is to evaluate the clinical benefit of AEZS-108 in men with castration- and taxane-resistant metastatic prostate cancer, for which the presence of LHRH receptors has been confirmed.

On December 14, 2010, we announced the initiation of a Phase 1/2 trial in castration refractory prostate cancer conducted by Dr. Jacek Pinski at the Norris Comprehensive Cancer Center, as well as a Phase 1/2 trial in refractory bladder cancer conducted by Dr. Gustavo Fernandez at the Sylvester Comprehensive Cancer Center.

AEZS-108 Companion diagnostic Tool

On June 28, 2010, we announced that we had concluded an agreement with Almac's Diagnostics division for AEZS-108, aimed at determining LHRH receptor expression through the development of a companion diagnostic tool. Selection for treatment with AEZS-108 is determined on the basis of LHRH receptor expression, currently measured immunohistochemically. In humans, LHRH receptors are expressed in ovarian, endometrial, breast, bladder, prostate and pancreatic tumors. This state of the art companion diagnostic tool will allow us to develop improved methods of selecting the most appropriate patients to be treated with AEZS-108 in order to enhance the efficiency of our clinical trials and help us with the future development of AEZS-108 in a number of different LHRH expressing cancers.

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TUBULIN INHIBITORS / VASCULAR TARGETING AGENTS

AEZS-112 Development of a Low Molecular Weight Tubulin Inhibitor with Anti-Angiogenic Properties

Tubulin is a protein found in all cells that plays an important role during cell division, in that it helps to transmit genetic information to the daughter cells. Inhibition of this process leads to the death of the affected cell. The anti-tumor agent taxol and vincristine, which are widely used in cancer therapy, are based on this principle. Both compounds are expensive natural substances and cause severe side effects when used in humans.

We are currently identifying and developing novel tubulin inhibitors which, compared with currently used products, exhibit in animal models improved efficacy, have a more acceptable side effect profile, an incomplete or no cross-resistance and are administered orally.

AEZS-112 is a drug development candidate with an excellent tolerability profile showing excellent *in vivo* activity in various tumor models including mammary, colon, melanoma and leukemia cancers after oral administration. This compound acts through three mechanisms of action. Strong anti-cancer activity is combined with pro-apoptotic and anti-angiogenic properties. AEZS-112 inhibits the polymerization of cancer tubulin rather than bovine brain tubulin, it destroys the mitotic spindle of the cancer cells and it inhibits topoisomerase II activity. AEZS-112 arrests the cancer cells in the G2M phase at a nanomolar concentration and induced apoptosis. AEZS-112 is not cross-resistant to cisplatin, vincristine and doxorubicine in cell lines resistant to these drugs. Given orally once weekly, AEZS-112 proved to be a potent inhibitor of *in vivo* tumor growth in melanoma, mammary, colon, lung, renal as well as in leukemia cancers at acceptable and very well tolerated doses. Furthermore AEZS-112 showed favorable safety and toxicity profiles. No findings with respect to cardiotoxicity and neurotoxicology parameters could be observed during the toxicological evaluation in mice, rats and dogs. With this profile of activity, AEZS-112 is a promising candidate for further clinical development.

On January 8, 2007, we announced the initiation of a Phase 1 trial for AEZS-112 in patients with solid tumors and lymphoma. This open-label, dose-escalation, multi-center, intermittent treatment Phase 1 trial is being conducted in the United States with Daniel D. Von Hoff, MD, Senior Investigator at the Translational Genomics Research Institute in Phoenix, AZ, as the lead investigator. The trial includes up to 50 patients with advanced solid tumors and lymphoma who have either failed standard therapy or for whom no standard therapy exists. Patients will receive a once-a-week oral administration of AEZS-112 for three consecutive weeks, followed by a one-week period without treatment. The cycles will be repeated every four weeks based on tolerability and response, basically planned for up to four cycles, but allowing for continuation in case of potential benefit for the patient. The starting dose of AEZS-112 in this study is 13 mg/week, with doubling of doses in subsequent cohorts in the absence of significant toxicity. Primary endpoint of the Phase 1 trial focuses on determining the safety and tolerability of AEZS-112 as well as establishing the recommended Phase 2 dose and regimen. Secondary endpoints are aimed at establishing the pharmacokinetics and determining the efficacy based on standard response criteria.

Results of this Phase 1 study were presented in April 2009 at the AACR meeting. In part I, 22 patients (12 men / 10 women) were studied on 7 dose levels ranging from 13 to 800 mg/week. In all, 62 treatment cycles were administered. In part II, the weekly dose was split into 3 doses taken 8 hours apart. Ultimately, 22 patients (12 men / 10 women) were studied on 5 dose levels ranging from 120 to 600 (= 200 x 3) mg/week. As at April 1, 2009, 62 treatment cycles were administered (mean 3.2/patient) and treatment were ongoing in 8 patients. SD for more than 12 weeks was observed in 16 patients; 4 more patients were ongoing at less than 12 weeks. Prolonged courses of SD ranging from 20 to 39+ weeks were observed in 9 patients with the following primary cancer types: trachea (39+), tongue (30+), thyroid (29+), prostate and melanoma (28), non-small cell lung cancer (26+), pancreas and 2x colorectal (20). Except for one patient with a background of gastrointestinal problems (GI) who had dose-limiting GI reactions and electrolyte loss at a dose of 200x3mg/week, no clinically relevant

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drug-related adverse events or changes in laboratory parameters were observed. AEZS-112 was shown to be metabolically stable in human plasma. As predicted by pharmacokinetic modelling based on data from part I of the study, the split-dose scheme leads to a higher Cmax and trough values after administration of comparable doses. Those preliminary results showed that a maximum tolerated dose for weekly dosing has not been defined so far. However, prolonged courses of stable disease in both parts of the study are an encouraging observation.

Completion of this Phase 1 trial was announced on September 21, 2009. Stable disease with time to failure ranging from 20 to 60+ weeks was achieved in 12 patients with various cancer types, including melanoma and cancers of the colon/rectum, lung, pancreas, prostate, tongue, trachea and thyroid. In several of these patients, the duration of stabilization exceeded the duration of disease control on previous treatment regimens. Except for a dose-limiting gastrointestinal reaction in a patient with pre-existing GI problems, no clinically relevant drug-related adverse events or changes in laboratory safety parameters were observed.

IMMUNOTHERAPY / VACCINES

Cellular proteins expressed by oncogenes have been recognized as a major cause of tumor development. One of the central oncoproteins involved in cancer formation are the Raf proteins. Based on these proteins, new unique therapeutic strategies, new predictive animal models and new development products have been generated to efficiently combat cancer. These consist of virulence attenuated, genetically modified bacteria expressing tumor antigens, including oncoproteins or enzymes. Such bacteria are used for vaccination as well as tumor targeting and delivery of antitumoral compounds towards the tumor tissues. Therefore, this new vaccine approach exploits the ability of bacteria to induce potent immune responses as well as direct these responses against malignancies. The immunogenicity of the vaccine will be further enhanced by the capacity of bacteria to colonize tumor tissues. This property will be used to transport substances, e.g. proteins, into the tumor tissue, which are capable of converting non-toxic pro-drugs into active drugs. The use of bacterial carriers for therapeutic vaccination against tumors and the concept of bacterial tumor targeting will be further developed with the Julius-Maximilians-University of Würzburg, including the highly recognized researchers Prof. Dr. Ulf R. Rapp, who is a member of our Scientific Advisory Board, and Prof. Dr. Werner Goebel. Prof. Rapp is a known expert in the field of cell and tumor biology and Prof. Goebel is a pioneer in the field of vaccines based on recombinant bacteria.

The preclinical proof of principle has already been shown in a transgenic animal model and is supported by several patent applications that we have filed. The most advanced products are bacterial tumor vaccines which are based on the approved human vaccine strain *Salmonella typhi* Ty21a. The principle of these recombinant vaccine strains is the secretion of the tumor antigen using a so-called Type I secretion machinery derived from *Escherichia coli*. To date, two different vaccine strains have been generated up to GMP scale production a melanoma vaccine encompassing a mutated form of the oncogene B-Raf, which is present in more than 65% of melanomas, and a prostate cancer vaccine strain expressing and secreting PSA. For both vaccines, the preclinical proof of principle has been demonstrated in distinct animal models and the immunogenicity could be further enhanced compared to our already published strains (patent application filed in November 2006).

In 2007, the PSA vaccine (AEZS-120) was selected as the first preclinical development candidate of an anti-tumor vaccine. In September 2007, scientific advice from the Paul Ehrlich Institute, the German health authority for vaccines, was sought and the preclinical development program presented by us was in principle accepted.

A grant application was filed in Germany and was approved in 2008. In accordance with this grant, 50% of our preclinical development costs and 100% of those of our university partner will be reimbursed by the German Ministry of Science and Education. The preclinical development and manufacture of material for clinical trial was initiated in 2008 and is still ongoing.

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ENDOCRINOLOGY

Growth hormone secretagogue

AEZS-130/Solorel® (macimorelin) ("ghrelin agonist")

Growth hormone secretagogues ("GHS") represent a new class of pharmacological agents that directly stimulate GH secretion from the pituitary gland without the involvement of growth hormone-releasing hormone (GH-RH) or somatostatin. We believe that there is currently no GHS on the pharmaceutical market. Since GH is a potent regulator of lipid, sugar and protein metabolism, the potential clinical uses of GHS are numerous. They include growth retardation in children and treatment of cachexia in AIDS patients, which are currently the only approved uses of therapy of GH. The administration of GH, which has to be injected every day, is cumbersome. Therefore, we believe that there would be a demand for new orally active drugs like GHS.

As part of our university collaboration, we accessed new peptidomimetic compounds with GH secretagogue properties. The lead development candidate, AEZS-130, is a novel peptidomimetic GHS with potent and selective GH-releasing activity in humans. AEZS-130 underwent limited clinical pharmacology tests that demonstrated a potent stimulation of the GH secretion after oral administration in human volunteers. This product has been licensed to Ardana Bioscience Ltd. ("Ardana") (ARD-07), which initiated an open, randomized, placebo-controlled Phase 1 dose-ranging study in April 2004. Thirty-six healthy subjects were included in this study to receive either the reference hormone GH-RH by i.v. route or one of the following dose levels of AEZS-130: 0.005, 0.05 or 0.5 mg/kg by oral route. AEZS-130 at the dose of 0.5 mg/kg orally caused an increase in growth hormone release equivalent to that induced by GH-RH i.v. The compound was well tolerated and no other hormones showed a significant modification after any dose of AEZS-130.

In June 2006, Ardana presented results regarding AEZS-130 at the 2006 ENDO Convention. These results referred to the Phase 1 trial regarding the stimulating effects of AEZS-130 on growth hormone following both oral and intra-duodenal administration in healthy males. This study showed that AEZS-130 was well tolerated by the 36 volunteers enrolled and no adverse events were reported. Administration of AEZS-130 either orally or via intra-duodenal infusion results in increased levels of growth hormone in the blood. This stimulation of growth hormone appears to be selective as no other hormones/analytes that were measured (cortisol, ghrelin, prolactin, insulin, glucose and ACTH (adrenocorticotropic hormone)) were affected in a dose-dependent or statistically significant way by administration of AEZS-130 either orally or via intra-duodenal infusion.

In May 2007, Ardana gained orphan drug designation for AEZS-130 (Solorel®) as a diagnostic test for growth hormone deficiency in adults. The clinical development and toxicology programs for this indication were ongoing and Ardana announced the commencement in the United States of the planned pivotal registration study and the enrolment of the first patient in August 2007.

In June 2008, Ardana announced that the company stopped its operations and entered into voluntary administration. Consequently, the clinical study of AEZS-130 (Solorel®) as a diagnostic test for AGHD was suspended.

We announced the recovery of worldwide rights from Ardana for the compound AEZS-130 in the third quarter of 2008. In June 2009, we reported that, after regaining from Ardana the worldwide rights to the growth hormone secretagogue, AEZS-130, we had entered into an agreement with the administrators of Ardana to acquire all Ardana assets relating to AEZS-130 for $232,000. These assets include development data, inventory of compound, regulatory authorizations, including IND and orphan drug status as a diagnostic test granted in the United States, as well as a patent application protecting the use of AEZS-130 (Solorel®) for the diagnostic of growth hormone secretion deficiency.

During the same month, the first clinical data relating to the use of AEZS-130 (Solorel®) as a simple diagnostic test for AGHD were presented at the ENDO 2009 meeting by the main investigators Dr G. Merriam and Dr B.M.K. Biller. Data showed that in adult growth hormone deficient patients,

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the responses to the orally administered AEZS-130 (Solorel®) compound were comparable to currently validated agents and clearly separated patients from normal control subjects.

In October 2009, we announced that we had initiated activities intended to complete the clinical development of AEZS-130 (Solorel®) which could be the first oral diagnostic test approved for growth hormone deficiency ("GHD"). Aeterna Zentaris had already assumed the sponsorship of the IND and discussed with the FDA, the best way to complete the ongoing Phase 3 clinical trial, and subsequently file a New Drug Application for approval of AEZS-130 (Solorel®) as a diagnostic test for AGHD.

The pivotal Phase 3 trial is designed to investigate the safety and efficacy of the oral administration of AEZS-130 (Solorel®) as a growth hormone stimulation diagnostic test. It was accepted by the FDA that for the ongoing part of the study, AEZS-130 is not tested against a comparator drug, as Geref® has been removed from the market.

Oral administration of AEZS-130 (Solorel®) offers more convenience and simplicity over the current GHD tests used, requiring either i.v. or i.m. administration. Additionally, AEZS-130 (Solorel®) may demonstrate a more favorable safety profile than existing diagnostic tests, some of which may be inappropriate for certain patient populations e.g. diabetes mellitus or renal failure, and have demonstrated a variety of side effects which AEZS-130 (Solorel®) has not thus far. These factors may be limiting the use of GHD testing and may enable AEZS-130 (Solorel®) to become the diagnostic test of choice for GHD. AEZS-130 (Solorel®) has been granted Orphan Drug Designation for the diagnosis of growth hormone deficiency by the FDA, and Aeterna Zentaris is now the sponsor of this orphan designation.

On June 21, 2010, we presented positive data at the 92nd ENDO Meeting on AEZS-130 for diagnostic and therapeutic use. The preclinical data showed that AEZS-130 is a potent and safe oral synthetic GH-releasing compound with potential utility as a diagnostic test for growth hormone deficiencies. In addition to the diagnostic indication, we believe that, based on the results of Phase 1 studies, AEZS-130 (Solorel®) 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.

On July 14, 2010, we announced the presentation of a poster on AEZS-130 (Solorel®), entitled "Use of the Orally Active Ghrelin Mimetic AEZS-130 as a Simple Test for the Diagnosis of Growth Hormone (GH) Deficiency (GHD) in adults (AGHD)." Merriam G.R., Yuen K., Bonert V., Dobs A, Garcia J., Kipnes M., Molitch M., Swerdloff R., Wang C., Cook D., Altemose I. and Biller B. This poster was presented at the Seventh International Congress of Neuroendocrinology, in Rouen, France.

On October 5, 2010, we announced at the Fifth International Congress of the Growth Hormone Research Society and the Insulin-like Growth Factors Society, after the interim Phase 3 analysis, that AEZS-130 (Solorel®) demonstrated the potential to provide a simple, well tolerated and safe oral diagnostic test for AGHD.

On December 20, 2010, we announced we had reached agreement with the FDA on an SPA for AEZS-130 (Solorel®), enabling the Company to complete the ongoing registration study required to gain approval as a diagnostic test for AGHD.

Study Design

The SPA agreement has resulted in a modification to the original study, but does not alter the basic study design so that the completed portion of the study will work with the new part of the study to provide one complete Phase 3 study.

Original Study

The completed part of the study was a two-way crossover study, and included 42 patients with confirmed AGHD or multiple pituitary hormone deficiencies and a low insulin-like growth factor-I. A

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control group of 10 subjects without AGHD were matched to patients for age, gender, body mass index and (for females) estrogen status.

Each patient received two dosing regimens in random order, while fasting, at least 1 week apart. One regimen consisted of a 1 µg/kg (max. 100 µg) dose of GHRH (Geref Diagnostic®, Serono) with 30 g of ARG (ArGine®, Pfizer) administered intravenously over 30 minutes; the other regimen was a dose of 0.5 mg/kg body weight of Solorel® given in an oral solution of 0.5 mg/ml.

Completion of the study will be accomplished with the following revisions/additions to the current protocol:

- an additional 30 normal controls subjects will be enrolled to match the AGHD patients from the original cohort;
- further, an additional 20 subjects will be enrolled 10 AGHD patients and 10 matched normal control subjects;
- the above will bring the database to approximately 100 patients;
- all subjects will be receiving a dose of 0.5 mg/kg body weight of AEZS-130 (Solorel®); and
- as a secondary endpoint, the protocol will require that at least 8 of the 10 newly enrolled AGHD patients be correctly classified by a pre-specified peak GH threshold level.

Competition for AEZS-130 (Solorel®)

Competitors for AEZS-130 (Solorel®) as a diagnostic test for AGHD are principally the diagnostic tests currently performed by endocrinologists. Most commonly used diagnostics tests for GHD are:

- measurement of blood levels of Insulin Growth Factor ("IGF")-1, which is often used as the first test when GHD is suspected. However, this test is not used to definitively rule out GHD as many growth hormone deficient patients show normal IGF-1 levels;
- insulin Tolerance Test (ITT), which is considered to be the "gold standard" for GH secretion provocative tests but requires constant monitoring and is contra-indicated in patients with seizure disorders, with cardiovascular disease and in brain injured patients and elderly patients. ITT is administered i.v.;
- GHRH + Arginine test, which is an easier test to perform in an office setting and has a very good safety profile but is considered to be costly to administer compared to ITT and Glucagon. This test is contra-indicated in patients with renal failure. GHRH + Arginine is approved in the EU and has been proposed to be the best alternative to ITT, but it is not any longer available in the U.S. This test is administered i.v.; and
- glucagon test, which is simple to perform and is considered very safe by endocrinologists but is contraindicated in malnourished patients and patients who have not eaten for more than 48 hours. Since there is a suspicion that this test may cause hypoglycemia, it may not be appropriate in diabetic populations. This test is administered i.m.

Ghrelin receptor ligands

Ghrelin is a peptide predominantly produced by the stomach. Apart from a potent GH-releasing action, ghrelin has other activities including stimulation of lactotroph and corticotroph function, influence on the pituitary gonadal axis, stimulation of appetite, control of energy balance,

influence on sleep and behavior, control of gastric motility and acid secretion, and influence on pancreatic exocrine and endocrine function as well as on glucose metabolism. The recent discovery of ghrelin and its receptors opens up new opportunities for the development of drugs that will treat metabolic disorders. There is indeed a possibility that ghrelin analogs, acting as either agonists or antagonists, might have a clinical impact without affecting GH levels. The use of ghrelin antagonists as appetite suppressants or inhibitors of lipogenesis could open up new opportunities for the treatment of obesity and associated

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diseases (e.g. diabetes, cardiovascular diseases). The use of ghrelin agonists could have therapeutic benefits which are expected to offer hope for cachexic or anorexic patients.

In 2004, we signed a research and license collaboration agreement with Le Centre National de la Recherche Scientifique and University Montpellier I and II, France, acting in their own names, as well as in the name and on behalf of the Laboratoire des Aminoacides, Peptides et Protéines (LAPP) (UMR 5810), directed by Dr. Jean Martinez, for the synthesis and characterization of new chemical entities acting as ghrelin receptor ligands. According to the agreement, we have the worldwide rights to develop and exploit the new compounds for any indication. Compounds with the most potent affinity for the ghrelin receptors will be investigated further through an international network of academic investigators with expertise in the field of endocrinology in order to identify clinical development candidates.

Additionally, we also signed a research contract with the Department of Experimental and Environmental Medicine of the University of Milan, Italy, under the direction of Prof. Vittorio Locatelli, for the pharmacological characterization of potentially ghrelin receptor ligands.

In August 2005, we filed a first patent application to protect a series of new chemical entities characterized as ghrelin receptor ligands.

In May 2006, we signed a research project agreement with the University of Montreal. This research project will focus on the characterization of ghrelin receptor ligands on fat tissue. This project is led by Huy Ong, Professor at the Faculty of Pharmacy, at the University of Montreal.

In August 2006, we also initiated a research collaboration with the Centre de recherche de l'Hôpital Laval (Québec) under the direction of Dr. Denis Richard. This research collaboration will focus on the pharmacological characterization of ghrelin receptor ligands *in vivo* (e.g. the effects in diet-induced obesity models).

In October 2006, we presented for the first time our *in vivo* data on the capacity of ghrelin antagonists of selectively inhibiting food intake. This study, using a rat model, outlined the capacity of ghrelin antagonists' ability to inhibit appetite without affecting growth hormone secretion and represents evidence that ghrelin antagonist compounds can selectively inhibit food intake. It further supports the hope that ghrelin antagonist compounds have the potential to be useful for the treatment of obesity.

In 2007 and 2008, we presented at scientific meetings preclinical candidates having the interesting property to decrease body weight gain and fat accumulation in diet induced obesity models. The ongoing work will focus on the improvement of oral bioavailability.

In July 2009, new data supporting the use of AEZS-123 (JMV-2959), a ghrelin receptor antagonist, for the treatment of alcohol dependence that involved ghrelin were published. Data were published in the renowned American scientific journal, Proceedings of the National Academy of Sciences ("PNAS"). Data show that mice treated with ghrelin increase their alcohol consumption. When ghrelin's actions are blocked by administering ghrelin receptor antagonists such as AEZS-123, mice no longer show preference for an alcohol-associated environment in other words, alcohol is no longer able to produce its addictive effects that include reward searching behaviour (akin to craving in alcoholic patients). The work, coordinated by Aeterna Zentaris, emerged from an international collaboration between the research groups of Prof. Suzanne Dickson and Prof. Jörgen Engel who performed the pharmacology work at the Sahlgrenska Academy, Gothenburg, Sweden, and the research group of Prof. Jean Martinez who synthesized the tested compound AEZS-123 at the Institut des biomolécules Max Mousseron, Montpellier, France.

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LHRH ANTAGONISTS

Cetrorelix

Cetrorelix is a peptide-based active substance which was developed in cooperation with Nobel Laureate Professor Andrew Schally presently of the United States Veterans Administration-Miami, University of Miami, and formerly of Tulane University in New Orleans. This compound is a luteinising hormone releasing hormone (LHRH, also known as GnRH) antagonist that blocks the pituitary LHRH receptors resulting in a rapid decrease of sexual hormone levels. Moreover, cetrorelix allows the LHRH receptors on the pituitary gland to be blocked gradually. Conversely, the side effects usually associated with the use of agonists and resulting from total hormone withdrawal can be avoided in conditions that do not require a castrating degree of hormone withdrawal. Therefore, in contrast to treatment with agonists, LHRH antagonists permit dose-dependent hormone suppression which is of critical importance for the tolerability of hormonal therapy.

Cetrorelix In Vitro Fertilization (COS/ART)

Cetrotide®

Cetrorelix is the first LHRH antagonist which was approved for therapeutic use as part of fertilization programs in Europe and was launched on the market under the trade name Cetrotide® (cetrorelix acetate) in 1999. In women who undergo controlled ovarian stimulation for recovery of ovocytes for subsequent fertilization, Cetrotide® helps prevent premature ovulation. LHRH is a naturally occurring hormone produced by the brain to control the secretion of LH and, therefore, final egg maturation and ovulation. Cetrotide® is designed to prevent LH production by the pituitary gland and to delay the hormonal event, known as the "LH surge" which could cause eggs to be released too early in the cycle, thereby reducing the opportunity to retrieve the eggs for the assisted reproductive techniques procedure.

In comparison with LHRH agonists that require a much longer pre-treatment, the use of our LHRH antagonist, Cetrotide®, permits the physician to interfere in the hormone regulation of the women undergoing treatment much more selectively and within a shorter time.

The effectiveness of Cetrotide® has been examined in five clinical trials (two Phase 2 and three Phase 3 trials). Two dose regimens were investigated in these trials: either a single dose per treatment cycle or multiple dosing. In the Phase 2 studies, a single dose of 3 mg was established as the minimal effective dose for the inhibition of premature LH surges with a protection period of at least four days. When Cetrotide® is administered in a multi-dose regimen, 0.25 mg was established as the minimal effective dose. The extent and duration of LH suppression was found to be dose-dependent. In the Phase 3 program, efficacy of the single 3 mg dose regimen and the multiple 0.25 mg dose regimen was established separately in two controlled studies utilizing active comparators. A third non-comparative study evaluated only the multiple 0.25 mg dose regimen of Cetrotide®. In the five Phase 2 and Phase 3 trials, 184 pregnancies were reported out of a total of 732 patients (including 21 pregnancies following the replacement of frozen-thawed embryos). In these studies, drug-related side effects were limited to a low incidence of injected site reactions; however, none of them was serious such as an allergic type of reaction or required withdrawal from treatment. In addition, no drug-related allergic reactions were reported from these clinical studies.

Cetrotide® is the only LHRH antagonist that is available in two dosing regimens. With an immediate onset of action, Cetrotide® permits precise control a single dose (3 mg), which controls the LH surge for up to four days, or a daily dose (0.25 mg) given over a short period of time (usually five to seven days). The treatment with Cetrotide® can be accomplished during a one-month cycle with a simplified, more convenient and shorter treatment requiring fewer injections than LHRH agonists.

Cetrotide® is marketed in a 3 mg and a 0.25 mg subcutaneous injection as cetrorelix acetate by Merck Serono in the United States and Europe. Approval for Cetrotide® in Japan was gained in

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April 2006. In September 2006, we announced the launch of Cetrotide® in Japan for *in vitro* fertilization. Cetrotide® is marketed in Japan by our partner Shionogi. We receive revenue from the supply of Cetrotide® to our Japanese partners. The market competitor is ganirelix (Antagon /Orgalutran®) from Schering-Plough (Organon) indicated for the inhibition of premature LH surges in women undergoing controlled ovarian hyperstimulation.

Partners for Cetrotide®

In August 2000, we entered into a commercialization agreement with Merck Serono for Cetrotide®. Under the terms of this agreement, we granted an exclusive license to Merck Serono to commercialize Cetrotide® for IVF/COS/ART worldwide ex-Japan and we are entitled to receive fixed and sales royalties from Merck Serono. The Japanese rights for this indication are held by Shionogi whereby, according to a commercialization agreement, we received transfer pricing from Shionogi.

In December 2008, we sold our rights to royalties on future sales of Cetrotide® covered by our license agreement with Merck Serono for $52.5 million to Cowen Healthcare Royalty Partners ("CHRP") less transaction costs of $1.0 million, resulting in initial net proceeds to us of $51.5 million. In addition, upon net sales of Cetrotide® having reached a specified level in 2010, we received an additional payment of $2.5 million from CHRP in February 2011. Furthermore, under the terms of the agreement, we agreed to make a one-time cash payment to CHRP in an amount ranging from $5 million up to a maximum of $15 million in the event cetrorelix is approved for sale by the European regulatory authorities in an indication other than *in vitro* fertilization. The amount which would be due to CHRP will be higher the earlier the product receives European regulatory approval. Since cetrorelix development has been terminated, we do not expect to make this one-time cash payment to CHRP.

Clinical Development Overview of Cetrorelix in Benign Prostatic Hyperplasia ("BPH"), Endometriosis and Uterine Myoma

Cetrorelix in BPH

BPH is a hormone-driven enlargement of the male prostate gland. The prostate is located directly at the vesicle outlet in the male surrounding the first part of the urethra. The enlargement puts pressure on the urethra, causing difficulty in urinating. BPH is classified into three stages according to symptoms: 1) the irritant phase, where the patient suffers dysuria (pain when urinating) and nocturia (the urge to urinate during the night); 2) residual urine occurring in the bladder thus increasing problems during urinating; and 3) overflow of the bladder. These can result in formation of bladder stones, congestion of urine and engorged kidneys which can in turn lead to life-threatening kidney damage.

BPH clinical trials

On August 17, 2009, we reported Phase 3 results for our North American efficacy trial Z-033 (including certain sites in Europe) and safety trial Z-041 in BPH, with cetrorelix. The study Z-033 failed to achieve its primary endpoint, being an improvement in International Prostate Symptom Score ("IPSS") as compared to placebo, and it demonstrated no clear differences in overall efficacy with all 3 groups showing an improvement in IPSS of approximately 4 points that was maintained throughout the 52 weeks. There was a slight advantage in favor of the main active treatment arm (Arm A) up to Week 46 of the follow-up, which was no longer demonstrated at Week 52. These differences did not achieve statistical significance. Furthermore, a statistically significant effect on the IPSS, as compared to placebo, was seen in a sub-group of patients with large prostate glands (greater than 50 cm^3) on entry to the study. Tolerability of cetrorelix in study Z-033 was very good, as evidenced by the absence of major differences to placebo with regard to both clinical adverse events and changes in laboratory parameters.

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On December 7, 2009, we reported the Phase 3 results for cetrorelix from the European efficacy trial Z-036, involving 420 patients. Study Z-036 did not reach its primary endpoint. There were no clear differences in overall efficacy, with all 3 groups (including placebo) showing an improvement in IPSS of approximately 6 points that was maintained throughout the 52 weeks. There was observation of an improvement in uroflow, both maximum and mean, and in residual volume in all treatment groups. These favorable changes are reflected in an overall improvement in Quality of Life measures. Furthermore, a favorable trend on the IPSS, as compared to placebo, was seen in a sub-group of patients with large prostate glands (greater than 50 cm^3) on entry to the study. Cetrorelix was well tolerated, there were no relevant differences to placebo with regard to both clinical adverse events or changes in laboratory parameters with the exception of the anticipated hormonal changes.

On December 18, 2009, following the unsuccessful results of our Phase 3 program in BPH with cetrorelix, we announced the termination of our agreement with sanofi dated March 5, 2009, for the development, commercialization and licensing of cetrorelix in BPH for the U.S. market. Termination of the agreement took effect as at January 9, 2010.

Cetrorelix in endometriosis and uterine myoma

There is no active program ongoing at present.

Partners for Cetrorelix

We previously licensed cetrorelix to Solvay worldwide (ex-Japan) for all indications with the exception of IVF/COS/ART, which rights belong to Merck Serono and Japanese rights are held by Shionogi. In the BPH indication, for which we regained exclusive worldwide (ex-Japan) rights, Japanese rights are held by Shionogi. On May 8, 2007, we and Solvay announced the termination of the license and cooperation agreement for cetrorelix for all remaining indications, including endometriosis, effective on that date, as a result of which we regained exclusive worldwide (ex-Japan) rights for cetrorelix in all indications without any financial compensation payable to Solvay.

On March 22, 2007, we announced that Nippon Kayaku had terminated its development agreement pertaining to cetrorelix pamoate to focus solely in oncology.

We signed a license and cooperation agreement for the commercialization of cetrorelix (BPH indication) with Handok for the Korean market during the third quarter of 2008.

On March 5, 2009, we entered into a development, commercialization and license agreement with sanofi-aventis for the development, registration and marketing of cetrorelix in BPH for the U.S. market. Under the terms of the agreement, sanofi-aventis made an initial upfront payment to us of $30.0 million. Following the announcement of the negative results for the efficacy trial in North America (study Z-033) and in Europe (study Z-036), we announced the termination of our agreement with sanofi-aventis dated March 5, 2009 for the development, commercialization and licensing of cetrorelix in BPH for the U.S. market. Termination of the agreement was effective January 9, 2010.

Following the negative Phase 3 results for cetrorelix in BPH, our Japanese partner, Shionogi, also agreed with the Company to cease the development of cetrorelix in this indication.

Ozarelix

Ozarelix is a modified LHRH antagonist which is a linear decapeptide sequence. Ozarelix is a fourth-generation LHRH antagonist aiming at extended suppression of testosterone levels that does not require a sophisticated depot formulation for long-lasting activity.

On August 12, 2004, we entered into a licensing and collaboration agreement with Spectrum Pharmaceuticals, Inc. ("Spectrum"), for ozarelix and its potential to treat hormone-dependent cancers as well as benign proliferative disorders, such as BPH and endometriosis for all potential indications in North America (including Canada and Mexico) and India while keeping the rights for the rest of the

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world. In addition, Spectrum is entitled to receive 50% of upfront and milestone payments and royalties received from our Japanese partner, Nippon Kayaku, that are generated in the Japanese market for oncological indications. In November 2010, this agreement with Spectrum was amended. Under the terms of the amended agreement, Spectrum is entitled to use the Company's patent rights and know-how to develop, use, make, have made, sell, offer for sale, have sold, import and export, commercialize ozarelix in all worldwide territories except Japan, Korea, Indonesia, Malaysia, the Philippines and Singapore. Under the terms of the amended agreement, Spectrum granted, as further consideration, 326,956 shares of its common stock, with an equivalent fair value of approximately $1,263,000, as an upfront nonrefundable license fee payment to the Company. Also per the amended agreement, the Company will be entitled to receive a total of approximately $22,765,000 in cash payments, as well as approximately $670,000 of Spectrum's common stock, upon achieving certain regulatory milestones in various markets. Furthermore, the Company will be entitled to receive royalties (scale-up royalties from high single to low double-digit) on future net sales of ozarelix products in the named territories.

During the third quarter of 2008, we entered into a commercialization agreement with Handok for ozarelix (BPH indication) for the Korean market.

On January 27, 2010, Spectrum announced that it had terminated its development program with ozarelix in BPH. Consequently, an impairment loss of approximately $1,422,000 was recorded as part of amortization expense, and all corresponding unamortized deferred revenues related to the use of ozarelix, totalling approximately $1,606,000, were fully recognized in the 2009 consolidated statement of operations.

BPH Clinical Trials

In October 2006, we announced positive and highly statistically significant Phase 2 results for ozarelix in BPH. The primary efficacy endpoint of improving clinical symptoms of BPH at week 12, as measured by significant changes in IPSS, was achieved at all dosage regimens. Secondary efficacy parameters such as uroflow, residual urinary volume, quality of life and circulating testosterone levels were also measured and showed good results. The outcome of the trial demonstrated an excellent safety profile with ozarelix as patients had no serious side effects. The erectile function was also not affected at any dose regimens.

On May 23, 2007 and September 5, 2007, Spectrum disclosed detailed Phase 2 results for ozarelix in BPH at two medical conferences. Results indicate that ozarelix was well tolerated and demonstrated statistically significant as well as clinically meaningful efficacy in the treatment of LUTS secondary to BPH.

On January 3, 2007, Spectrum announced the FDA's acceptance of an IND for ozarelix in BPH. Spectrum initiated a Phase 2b study in January 2007. On April 22, 2008, our partner Spectrum released the nine-month Phase 2b results for ozarelix. Spectrum indicated that ozarelix demonstrated sufficient clinical activity to justify its continued development in BPH. Based on these results, Spectrum initiated in September 2008 the recruitment of 860 patients for a new BPH study. In January 2010, Spectrum Pharmaceuticals announced the discontinuation of ozarelix development in BPH, stating that the mixed results of their Phase 2b study and the announced negative results of our Phase 3 registrational trial of cetrorelix in BPH does not support continued development of ozarelix in this indication.

Prostate Cancer Clinical Trials

In August 2006, we announced positive Phase 2 results for ozarelix in hormone-dependent inoperable prostate cancer. This open-label, randomized-controlled dose-finding trial enrolled 64 patients receiving different IM dosage regimens of ozarelix to assess its safety and efficacy. The study achieved its primary endpoint of defining a tolerable dosage regimen of ozarelix that would ensure continuous suppression of testosterone at castration level for a three-month test period. A

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secondary efficacy endpoint aimed at assessing tumor response as determined by a 50% or greater reduction of serum PSA level, compared to baseline, was also achieved. The best results regarding the primary endpoint of continuous suppression were obtained with a dose of 130 mg per cycle where all patients remained suppressed to castration until at least day 85. In patients with continuous testosterone suppression below castration level, tumor response as measured by PSA levels was 97%. Following these results, we, in collaboration with Spectrum, initiated an additional Phase 2 study in European centers to verify and optimize the findings derived from the cohort of patients having received 130 mg of ozarelix per cycle.

On August 3, 2006, we announced a licensing and collaboration agreement with Nippon Kayaku for ozarelix. Under the terms of the agreement, we granted Nippon Kayaku an exclusive license to develop and market ozarelix for all potential oncological indications in Japan. In return, we received an upfront payment upon signature and are eligible to receive payments upon achievement of certain development and regulatory milestones, in addition to low double-digit royalties on potential net sales. Spectrum is entitled to receive 50% of the upfront, milestone payments and royalties received from Nippon Kayaku.

Non-Peptide LHRH Antagonists

As outlined above, the LHRH receptor plays an important role in a number of benign and malignant tumors. Our drug discovery unit searches for small, non-peptide molecules which have the same effect on the receptor. Their advantage lies in the potential for oral administration.

AEZS-115 is a new orally bioavailable LHRH antagonist with LHRH-receptor binding affinity in the nanomolar range which is developed for hormone therapy of endocrinological disorder and of benign and malignant tumors. The compound demonstrates excellent selectivity to LHRH-receptor and has advanced to a preclinical stage where the *in vivo* activity has been confirmed. Major advantages are the dose-dependent reduction of sexual hormones without flare-up effect whereas no decrease down to castration level is necessary and therefore side effects are reduced.

In January 2006, we regained the exclusive worldwide rights to develop and commercialize AEZS-115 from Solvay. Attractive *in vivo* activity of this orally available peptidomimetic LHRH-antagonist was demonstrated with a single, oral administration (20mg/kg) in rats which led to efficient and revocable suppression of plasma testosterone levels for up to 12 hours. Furthermore, a repeat of the dosing of AEZS-115 increased the suppression time without accumulation in the plasma.

In 2007, an oral formulation was selected and pharmacokinetic data were obtained.

First preclinical results were presented at the 2008 San Antonio Breast Cancer Symposium on December 12, 2008 and showed substantial anti-tumor activity of AEZS-115 in human ovarian and breast cancer cell lines, as evidenced by exposure of human cell lines SKOV3, Ovcar 3 (human ovarian cancer cell lines) and MDA-MB 468 (human breast cancer cell line) to increasing concentrations of AEZS-115, peptidic GnRH-antagonist cetrorelix and GnRH-agonist Triptorelin (1, 10, and 100 µM) for 48 days. The number of viable cells was determined by crystal violet staining as well as by ATP-dependent luminometric assays. Results showed that both GnRH-antagonists dose-dependently inhibited growth of all three cell lines, while GnRH-agonist Triptorelin showed marginal growth inhibition. Cell growth was inhibited by 40-60% following exposure to a concentration of 10 µM of AEZS-115 and by 60-80% when cells were exposed to 100 µM. Inhibition with cetrorelix at 100 µM ranged from 20-40%, while only minor effects on cell growth were seen at 10µM. Optimization is ongoing.

RAW MATERIALS

Raw materials and supplies are generally available in quantities adequate to meet the needs of our business. We are dependent on third-party manufacturers for the pharmaceutical products that we market. An interruption in the availability of certain raw materials or ingredients, or significant

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

DISTRIBUTION

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. 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.

REGULATORY COMPLIANCE

Governmental authorities in Canada, the United States, Europe and other countries extensively regulate the preclinical and clinical testing, manufacturing, labeling, storage, record keeping, advertising, promotion, export, marketing and distribution, among other things, of our product candidates. Under the laws of the United States, the countries of the European Union, and other countries, we and the institutions where we sponsor research are subject to obligations to ensure that our clinical trials are conducted in accordance with GCP guidelines and the investigational plan and protocols contained in an Investigational New Drug application, or comparable foreign regulatory submission. The Japanese regulatory process for approval of new drugs is similar to the FDA approval process described below except that Japanese regulatory authorities request bridging studies to verify that foreign clinical data are applicable to Japanese patients and also require the tests to determine appropriate dosages for Japanese patients to be conducted on Japanese patient volunteers. Due to these requirements, delays of two to three years in introducing a drug developed outside of Japan to the Japanese market are possible. Set forth below is a brief summary of the material government regulations affecting the Company in the major markets in which we intend to market our products.

Canada

In Canada, the Canadian Therapeutic Products Directorate is the Canadian federal authority that regulates pharmaceutical drugs and medical devices for human use. Prior to being given market authorization, a manufacturer must present substantive scientific evidence of a product's safety, efficacy and quality as required by the *Food and Drugs Act* and other legislation and regulations. The requirements for the development and sale of pharmaceutical drugs in Canada are substantially similar to those in the United States, which are described below.

United States

In the United States, the FDA under the Federal Food, Drug, and Cosmetic Act, the Public Health Service Act and other federal statutes and regulations, subject pharmaceutical products to rigorous review.

In order to obtain approval of a new product from the FDA, we must, among other requirements, submit proof of safety and efficacy as well as detailed information on the manufacture and composition of the product. In most cases, this proof entails extensive preclinical, clinical, and laboratory tests. Before approving a new drug or marketing application, the FDA also typically conducts pre-approval inspections of the company, its contract research organizations and/or its clinical trial sites to ensure that clinical, safety, quality control, and other regulated activities are compliant with Good Clinical Practices, or GCP, or Good Laboratory Practices, or GLP, for specific non-clinical toxicology studies. Manufacturing facilities used to produce a product are also subject to ongoing inspection by the FDA. The FDA may also require confirmatory trials, post-marketing testing, and extra surveillance to monitor the effects of approved products, or place conditions on any approvals that could restrict the

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commercial applications of these products. Once approved, the labeling, advertising, promotion, marketing, and distribution of a drug or biologic product must be in compliance with FDA regulatory requirements.

The first stage required for ultimate FDA approval of a new biologic or drug involves completion of preclinical studies and the submission of the results of these studies to the FDA. This, together with proposed clinical protocols, manufacturing information, analytical data, and other information in an IND, must become effective before human clinical trials may commence. Preclinical studies involve laboratory evaluation of product characteristics and animal studies to assess the efficacy and safety of the product. The FDA regulates preclinical studies under a series of regulations called the current GLP regulations. If the sponsor violates these regulations, the FDA may require that the sponsor replicate those studies.

After the IND becomes effective, a sponsor may commence human clinical trials. The sponsor typically conducts human clinical trials in three sequential phases, but the phases may overlap. In Phase 1 trials, the sponsor tests the product in a small number of patients or healthy volunteers, primarily for safety at one or more doses. Phase 1 trials in cancer are often conducted with patients who have end-stage or metastatic cancer. In Phase 2, in addition to safety, the sponsor evaluates the efficacy of the product in a patient population somewhat larger than Phase 1 trials. Phase 3 trials typically involve additional testing for safety and clinical efficacy in an expanded population at geographically dispersed test sites. The sponsor must submit to the FDA a clinical plan, or "protocol," accompanied by the approval of the institutions participating in the trials, prior to commencement of each clinical trial. The FDA may order the temporary or permanent discontinuation of a clinical trial at any time. In the case of product candidates for cancer, the initial human testing may be done in patients with the disease rather than in healthy volunteers. Because these patients are already afflicted with the target disease, such studies may provide results traditionally obtained in Phase 2 studies. Accordingly, these studies are often referred to as "Phase 1/2" studies. Even if patients participate in initial human testing and a Phase 1/2 study is carried out, the sponsor is still responsible for obtaining all the data usually obtained in both Phase 1 and Phase 2 studies.

The sponsor must submit to the FDA the results of the preclinical and clinical testing, together with, among other things, detailed information on the manufacture and composition of the product, in the form of a new drug application or, in the case of a biologic, a BLA. In a process that can take a year or more, the FDA reviews this application and, when and if it decides that adequate data are available to show that the new compound is both safe and effective for a particular indication and that other applicable requirements have been met, approves the drug or biologic for marketing. The amount of time taken for this approval process is a function of a number of variables, including the quality of the submission and studies presented and the potential contribution that the compound will make in improving the treatment of the disease in question.

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. We hold orphan drug designations for perifosine in multiple myeloma and for the treatment of neuroblastoma and for AEZS-108 for the treatment of advanced ovarian cancer as well as for AEZS-130 (Solorel) for the diagnosis of growth hormone deficiency.

Under the Hatch-Waxman Act, newly-approved drugs and indications may benefit from a statutory period of non-patent data exclusivity. The Hatch-Waxman Act provides five-year data exclusivity to the first applicant to gain approval of an NDA for a new chemical entity, or NCE, meaning that the FDA has not previously approved any other drug containing the same active pharmaceutical ingredient, or active moiety. Although protection under the Hatch-Waxman Act will not prevent the submission or

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approval of another full NDA, such an NDA applicant would be required to conduct its own preclinical and adequate, well-controlled clinical trials to demonstrate safety and effectiveness.

The Hatch-Waxman Act also provides three years of data exclusivity for the approval of new and supplemental NDAs, including Section 505(b)(2) applications, for, among other things, new indications, dosage forms, routes of administration, or strengths of an existing drug, or for a new use, if new clinical investigations that were conducted or sponsored by the applicant are determined by the FDA to be essential to the approval of the application. This exclusivity, which is sometimes referred to as clinical investigation exclusivity, would not prevent the approval of another application if the applicant has conducted its own adequate, well-controlled clinical trials demonstrating safety and efficacy, nor would it prevent approval of a generic product that did not incorporate the exclusivity-protected changes of the approved drug product.

The labeling, advertising, promotion, marketing, and distribution of a drug or biologic product must be in compliance with FDA regulatory requirements. Failure to comply with applicable requirements can lead to the FDA demanding that production and shipment cease and, in some cases, that the manufacturer recall products, or to enforcement actions that can include seizures, injunctions, and criminal prosecution. These failures can also lead to FDA withdrawal of approval to market a product.

European Union

Medicines can be authorized in the European Union by using either the centralized authorization procedure or national authorization procedures.

Centralized procedure

The European Union has implemented a centralized procedure coordinated by the EMA for the approval of human medicines, which results in a single marketing authorization issued by the European Commission that is valid across the European Union, as well as Iceland, Liechtenstein and Norway. The centralized procedure is compulsory for human medicines that are derived from biotechnology processes, such as genetic engineering, contain a new active substance indicated for the treatment of certain diseases, such as HIV/AIDS, cancer, diabetes, neurodegenerative disorders or autoimmune diseases and other immune dysfunctions, and designated orphan medicines. For medicines that do not fall within these categories, an applicant has the option of submitting an application for a centralized marketing authorization to the EMA, as long as the medicine concerned is a significant therapeutic, scientific or technical innovation, or if its authorization would be in the interest of public health.

National authorization procedures

There are also two other possible routes to authorize medicinal products in several European Union countries, which are available for investigational drug products that fall outside the scope of the centralized procedure:

Decentralized procedure. Using the decentralized procedure, an applicant may apply for simultaneous authorization in more than one European Union country of medicinal products that have not yet been authorized in any European Union country and that do not fall within the mandatory scope of the centralized procedure.

The application will be reviewed by a selected Reference Member State ("RMS"). The Marketing Authorization granted by the RMS will then be recognized by the other Member States involved in this procedure.

Mutual recognition procedure. In the mutual recognition procedure, a medicine is first authorized in one European Union Member State, in accordance with the national procedures of that country. Following this, further marketing authorizations can be sought from other European Union

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countries in a procedure whereby the countries concerned agree to recognize the validity of the original, national marketing authorization.

For more information about the regulatory risks associated with the Company's business operations, see "Item 3. Key Information Risk Factors".

DRUG DISCOVERY

There is an increasing demand on the world market for active substances. Our internal drug discovery unit provides an important prerequisite for the provision of new patented active substances, which can then be developed further or licensed to third parties.

Our drug discovery unit concentrates on the search for active substances for innovative targets which open the door to the introduction of new therapeutic approaches. Further, this unit searches for new active substances having improved properties for clinically validated targets for which drugs are already being used in humans and which produce inadequate effects, cause severe side effects, are not economical or are not available in a patient-friendly form.

To this end, we possess an original substance library for the discovery of active compounds with a comprehensive range of promising natural substances which can serve as models for the construction of synthetic molecules. The initial tests involve 120,000 samples from our internal substance library in the form of high-throughput screening. The hits, i.e. the first active compounds found in the library, are tested further and built up specifically into potential lead structures. Based on two to three lead structures, they are then optimized in a further step to potential development candidates.

INTELLECTUAL PROPERTY PATENTS

We believe that we have a solid intellectual property portfolio that covers compounds, manufacturing processes, compositions and methods of medical use for our lead drugs and drug candidates. Our patent portfolio consists of about 50 owned and in licensed patent families (issued, granted or pending in the United States, Europe and other jurisdictions). Independent from the original patent expiry date additional exclusivity is possible in the United States, Europe and several other countries by data protection for new chemical entities, by orphan drug designation, or by patent term extension respective supplementary protection certificate.

In the United States, the patent term of a patent that covers an FDA-approved drug may also be eligible for patent term extension, which permits patent term restoration as compensation for the patent term lost during the FDA regulatory review process. The Drug Price Competition and Patent Term Restoration Act of 1984, or the Hatch-Waxman Act, permits a patent term extension of up to five years beyond the expiration of the patent. The length of the patent term extension is related to the length of time the drug is under regulatory review. Patent extension cannot extend the remaining term of a patent beyond a total of 14 years from the date of product approval and only one patent applicable to an approved drug may be extended. Similar provisions are available in Europe and other foreign jurisdictions to extend the term of a patent that covers an approved drug. In the future, if and when our pharmaceutical products receive FDA approval, we expect to apply for patent term extensions on patents covering those products. While we anticipate that any such applications for patent term extensions will likely be granted, we cannot predict the precise length of the time for which such patent terms would be extended in the United States, Europe or other jurisdictions. If we are not able to secure patent term extensions on patents covering our products for meaningful periods of additional time, we may not achieve or sustain profitability, which would adversely affect our business.

Of the issued or granted patents, the protective rights described below form the core of our patent portfolio with regard to our lead drugs and drug candidates.

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Perifosine:

U.S. patent 6,172,050 provides protection in the United States for the compound perifosine and other related alkyl phospholipid derivatives, pharmaceutical compositions comprising the compounds as well as their medical use for the treatment of tumors. This U.S. patent expires in July 2013. A patent term extension of up to five years may be possible.

European patent 0 579 939 provides protection in European countries for the compound perifosine and other related alkyl phospholipid derivatives, pharmaceutical compositions comprising the compounds as well as their medical use for the treatment of tumors. This European patent expires in June 2013. A patent term extension of up to five years by Supplementary Protection Certificates ("SPC") may be possible.

Japanese patent 3 311 431 provides protection in Japan for the compound perifosine and other related alkyl phospholipid derivatives. This Japanese patent expires in July 2013. A patent term extension of up to five years may be possible.

U.S., European and Japanese patent applications have been filed, comprising the combination of perifosine with an antimetabolite for treating tumour diseases.

AEZS-108:

U.S. patent 5,843,903 provides protection in the United States for the compound AEZS-108 and other related targeted cytotoxic anthracycline analogs, pharmaceutical compositions comprising the compounds as well as their medical use for the treatment of cancer. This U.S. patent expires in November 2015. A patent term extension of up to five years may be possible.

European patent 0 863 917 B1 provides protection in Europe for the compound AEZS-108 and other related targeted cytotoxic anthracycline analogs, pharmaceutical compositions comprising the compounds as well as their medical use for the treatment of tumors. This European patent expires in November 2016. A patent term extension of up to five years may be possible.

Japanese patent 3 987 575 provides protection in Japan for the compound AEZS-108 and other related targeted cytotoxic anthracycline analogs, pharmaceutical compositions comprising the compounds as well as their medical use for the treatment of tumors. This Japanese patent expires in November 2016. A patent term extension of up to five years may be possible.

AEZS-130:

U.S. patent 6,861,409 protects the compound AEZS-130 and U.S. patent 7,297,681 protects other related growth hormone secretagogue compounds, each also protecting pharmaceutical compositions comprising the compounds as well as their medical use for elevating the plasma level of growth hormone. This U.S. patent 6,861,409 expires in August 2022. A patent term extension of up to five years may be possible.

European patent 1 289 951 protects the compound AEZS-130 and European patent 1 344 773 protects other related growth hormone secretagogue compounds, pharmaceutical compositions comprising the compounds as well as their medical use for elevating the plasma level of growth hormone. This European patent 1 289 951 expires in June 2021. A patent term extension of up to five years by SPC may be possible.

Japanese patent 3 522 265 protects the compound AEZS-130 and pharmaceutical compositions comprising the compounds as well as their medical use for elevating the plasma level of growth hormone. This Japanese patent expires in June 2021. A

patent term extension of up to five years may be possible.

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Cetrotide®:

European patent 0 299 402 provides protection in European countries for the compound cetrorelix and other LHRH antagonists. This patent will expire in July 2013 pursuant to granted requests for SPC.

Japanese patent 2 944 669 provides protection in Japan for the compound cetrorelix and other LHRH antagonists. This patent will expire in July 2013 pursuant to granted requests for patent term extension.

U.S. patent 6,828,415 protects a method for preparing sterile lyophilizate formulations of cetrorelix. It specifically protects the lyophilization process used to manufacture Cetrotide®. This U.S. patent will expire in December 2021.

European patent 0 611 572 protects a method for preparing sterile lyophilizate formulations of cetrorelix. It specifically protects the lyophilization process used to manufacture Cetrotide®. This patent will expire in February 2014.

Japanese patent 4 033 919 protects a method for preparing sterile lyophilizate formulations of cetrorelix. It specifically protects the lyophilization process used to manufacture Cetrotide®. This patent will expire in February 2014.

U.S. patent 7,790,686 protects an aqueous injectable solution of the compound cetrorelix or other LHRH antagonists in an organic, pharmaceutically acceptable acid. This patent will expire in October 2023.

European patent 1 448 221 protects an aqueous injectable solution of the compound cetrorelix or other LHRH antagonists in an organic, pharmaceutically acceptable acid. This patent will expire in November 2022.

AEZS-112:

U.S. patent 7,365,081 provides protection in the United States for the compound AEZS-112 and other related indole derivatives processes for preparing, and medicaments comprising them, and their medical use for treating cancer. This U.S. patent will expire in September 2017. A patent term extension of up to five years may be possible.

European patent 1 309 585 provides protection in Europe for the compound AEZS-112 and other related indole derivatives, and medicaments comprising them. This European patent will expire in July 2021. A SPC of up to five years may be possible.

Ozarelix:

U.S. patent 6,627,609 provides protection in the United States for the compound ozarelix and related third-generation LHRH antagonists and pharmaceutical compositions comprising them. This U.S. patent will expire in March 2020. A patent term extension of up to five years may be possible.

European patent 1 163 264 provides protection in Europe for the compound ozarelix and related third-generation LHRH antagonists and pharmaceutical compositions comprising them. This European patent will expire in March 2020. A SPC of up to five years may be possible.

Japanese patent 3 801 867 provides protection in Japan for the compound ozarelix and related third-generation LHRH antagonists and pharmaceutical compositions comprising them. This Japanese patent will expire in March 2020. A patent

term extension of up to five years may be possible.

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The table below lists some of our issued or granted patents in the United States and Europe:

Patent No.	Title	Country	Expiry Date*
Perifosine			
U.S. 6,172,050	Phospholipid derivatives	United States	2013-07-07*
EP 0 579 939	Phospholipid derivatives	Germany, United Kingdom, France, Switzerland and others	2013-06-03*
JP 3 311 431	Phospholipid derivatives	Japan	2013-07-08*
AEZS-108			
U.S. 5,843,903	Targeted cytotoxic anthracycline analogs	United States	2015-11-27*
EP 0 863 917	Targeted cytotoxic anthracycline analogs	Europe	2016-11-14*
JP 3 987 575	Targeted cytotoxic anthracycline analogs	Japan	2016-11-14*
AEZS-130			
U.S. 6,861,409	Growth hormone secretagogues	United States	2022-08-01*
EP 1 289 951	Growth hormone secretagogues	Germany, United Kingdom, France, Switzerland and others	2021-06-13*
JP 3 522 265	Growth hormone secretagogues	Japan	2021-06-13*
Cetrotide®			
EP 0 299 402	LHRH antagonists	Germany, United Kingdom, France, Switzerland and others	2013-07-10
EP 0 611 572	Process to prepare a cetrorelix lyophilised composition	Germany, United Kingdom, France, Switzerland and others	2014-02-04*
U.S. 6,828,415	Oliogopeptide lyophilisate, their preparation and use	United States	2021-12-07*
U.S. 6,716,817	Method of treatment of female infertility	United States	2014-02-22*
U.S. 6,863,891	Oligopeptide lyophilisate, their preparation and use	United States	2014-02-22*
U.S. 6,867,191	Preparation and use of oligopeptide lyophilisate for gonad protection	United States	2014-02-22*
U.S. 7,790,686	Injection solution of an LHRH antagonist	United States	2022-10-28*
AEZS-112			
U.S. 7,365,081	Indole derivatives and their use as medicaments	United States	2017-09-18*
EP 1 309 585	Indole derivatives and their use as medicaments	Germany, United Kingdom, France, Switzerland and others	2021-07-26*
Ozarelix			
U.S. 6,627,609	LHRH antagonists having improved solubility properties	United States	2020-03-14*
EP 1 163 264	LHRH antagonists having improved solubility properties	Germany, United Kingdom, France, Switzerland and others	2020-03-11*
JP 3 801 867	LHRH antagonists having improved solubility properties	Japan	2020-03-11*

* Excluding any Patent Term Extension

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C. Organizational structure

The following chart presents our corporate structure, the jurisdiction of incorporation of our direct and indirect subsidiaries and the percentage of shares that we held in those subsidiaries as at December 31, 2010.

D. Property, plants and equipment

Our corporate head office and facilities are located in Quebec City, Province of Quebec, Canada. The following table sets forth information with respect to our main facilities as at March 24, 2011.

Location	Use of space	Square Footage	Type of interest
1405 du Parc Technologique Blvd. Quebec City (Quebec), Canada	Fully occupied for management, R&D and administration	4,400	Leased
20 Independence Blvd Warren, New Jersey, United States	Partially occupied for management, R&D and business development	10,741[(1)]	Leased
Weismüllerstr. 50 Frankfurt-am-Main, Germany D-60314	Fully occupied for management, R&D, business development and administration	46,465	Leased

(1) Aeterna Zentaris, Inc. sub-lets out to a sub-tenant approximately 7,500 square feet of adjacent premises.

Item 4A. Unresolved Staff Comments

None.

Item 5. Operating and Financial Review and Prospects

Highlights

Perifosine

January 25, 2010: Updated results of a Phase 2 study related to the use of perifosine in the treatment of advanced metastatic colon cancer showing a statistically significant benefit in survival.

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January 29, 2010: Publication in the February 2010 issue of the *Journal of Clinical Cancer Research* of positive Phase 2 results for perifosine as a single agent for the treatment of advanced Waldenstrom's macroglobulinemia.

February 3, 2010: Special Protocol Assessment ("SPA") granted by the United States Food and Drug Administration ("FDA") for the Phase 3 trial of perifosine in combination with capecitabine (Xeloda®) in refractory advanced colorectal cancer ("X-PECT"). The trial is to be conducted and sponsored by our partner, Keryx Biopharmaceuticals, Inc. ("Keryx").

March 1, 2010: Disclosure that the Committee for Orphan Medicinal Products of the European Medicines Agency ("EMA") had issued a positive opinion for orphan medicinal product designation for perifosine for the treatment of multiple myeloma.

April 5, 2010: Perifosine receives FDA Fast Track Designation for the Phase 3 X-PECT (**X**eloda® + **P**erifosine **E**valuation in **C**olorectal cancer **T**reatment) registration trial.

April 8, 2010: Initiation of the X-PECT Phase 3 registration trial by Keryx.

April 15, 2010: 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.

April 20, 2010: Presentations at the annual meeting of the American Association for Cancer Research ("AACR"), in Washington, D.C., of preclinical data on extracellular signal-regulated kinases ("Erk") inhibitor, AEZS-131, and on Erk/phosphoinositide 3-kinase ("PI3K") dual inhibitor, AEZS-132, as well as preclinical data from a study sponsored by the National Institutes of Health ("NIH") with perifosine in oncology.

May 17, 2010: 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.

June 7, 2010: Presentation at the American Society of Clinical Oncology's ("ASCO") annual meeting of Phase 1 data on single agent perifosine in the treatment of recurrent pediatric solid tumors, including patients with advanced brain tumors and neuroblastoma.

June 8, 2010: Report at the ASCO annual meeting of final Phase 2 results, confirming a statistically significant improvement in both time to tumor progression and overall survival with perifosine, in combination with capecitabine (Xeloda®), in the treatment of advanced metastatic colorectal cancer.

June 29, 2010: EMA issues positive Scientific Advice for Phase 3 trial with perifosine in colorectal cancer, therefore indicating that the data from the ongoing X-PECT trial are expected to be sufficient for product registration in Europe.

July 14, 2010: Perifosine receives orphan-drug designation by the FDA for the treatment of neuroblastoma, a cancer of the nervous system affecting mostly children and infants.

December, 6, 2010: Presentation at the 52nd Annual Meeting of the American Society of Hematology ("ASH") in Orlando, of the Phase 2 data on perifosine as a treatment for advanced chronic lymphocytic leukemia ("CLL") and Hodgkin's lymphoma ("HL"), as well as of Phase 1 results of perifosine in combination with Revlimid® and dexamethasone in multiple myeloma.

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AEZS-108

May 6, 2010: Received orphan drug designation from the FDA for AEZS-108, our luteinizing hormone-releasing hormone ("LHRH") receptor conjugate, for the treatment of ovarian cancer.

May 12, 2010: Received approval from the FDA for our Investigational New Drug ("IND") application for AEZS-108 in LHRH receptor-positive urothelial (bladder) cancer.

May 17, 2010: Received positive opinion for orphan medicinal product designation from the Committee for Orphan Medicinal Products ("COMP") of the EMA, for AEZS-108 for the treatment of ovarian cancer.

June 7, 2010: Presentation at the annual meeting of the ASCO of positive Phase 2 efficacy and safety data for AEZS-108 in ovarian cancer.

June 28, 2010: Announcement of a collaboration with Almac Group Ltd.'s ("Almac") Diagnostic division to develop a companion diagnostic for AEZS-108 in cancer.

August 5, 2010: Announcement that a clinical investigator, Dr. Jacek Pinski, of the Norris Comprehensive Cancer Center of the University of Southern California, had been awarded a grant of $1.5 million (payable over the next three years) from the NIH for a Phase 1/2 study in advanced refractory prostate cancer with AEZS-108.

November, 18, 2010: Presentation at the EORTC-NCI-AACR symposium in Germany of positive Phase 2 results of AEZS-108 in advanced endometrial cancer.

December 14, 2010: Announcement of the initiation of Phase 1/2 trials with AEZS-108 in castration refractory prostate cancer and refractory bladder cancer.

AEZS-130/Solorel®

June 21, 2010: Presentation at the 92nd Annual Endocrine Society ("ENDO") Meeting and Expo of positive data on Solorel®, a ghrelin agonist for diagnostic and therapeutic use.

July 14, 2010: Presentation at the Seventh International Congress of Neuroendocrinology in Rouen, France, of an abstract on Solorel®, an oral synthetic ghrelin receptor agonist, as a diagnostic test for Adult Growth Hormone ("GH") Deficiency ("AGHD").

October 5, 2010: Presentation at the Fifth International Congress of the Growth Hormone Research Society and the Insulin-like Growth Factors Society in New York City, of interim Phase 3 data on Solorel® demonstrating the potential to provide a simple, well tolerated and safe oral diagnostic test for AGHD.

December 20, 2010: Agreement with the FDA on an SPA for Solorel® to complete our Phase 3 study for the diagnosis of AGHD.

Corporate developments

April 20, 2010: Completion of a US$15.0 million registered direct offering with certain institutional investors.

June 21, 2010: Completion of a US$12.1 million registered direct offering with certain institutional investors.

On February 22, 2011, we entered into an "At-the-Market" ("ATM") sales agreement, under which we may, at our discretion, from time to time during the 24-month term of the agreement, sell up to a maximum of 12.5 million of our common shares through ATM issuances on the Nasdaq for aggregate gross proceeds not to exceed $19.8 million. On March 10, 2011, we issued

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approximately 1.7 million shares under this agreement for gross proceeds of approximately $3.2 million.

On February 28, 2011, we announced that we had received a net sales royalty milestone of $2.5 million from Cowen Healthcare Royalty Partners L.P. ("Cowen"). This milestone was payable pursuant to the sale, in December 2008, to Cowen of our rights to royalties on future net sales of Cetrotide®.

On March 9, 2011, we announced that we had entered into an agreement with Yakult Honsha Co. Ltd. ("Yakult") for the development, manufacture and commercialization of perifosine in all human uses, excluding leishmaniasis, in Japan.

Introduction

This Management's Discussion and Analysis ("MD&A") provides a review of the results of operations, financial condition and cash flows of Aeterna Zentaris Inc. for the year ended December 31, 2010. In this MD&A, "Aeterna Zentaris", the "Company", "we", "us", "our" and the "Group" mean Aeterna Zentaris Inc. and its subsidiaries. This discussion should be read in conjunction with the information contained in the Company's consolidated financial statements and related notes as at December 31, 2010 and December 31, 2009 and for the years ended December 31, 2010, 2009 and 2008. Our consolidated financial statements have been prepared in accordance with Canadian generally accepted accounting principles ("Canadian GAAP") for financial information, which differ in certain respects from United States generally accepted accounting principles ("US GAAP").The recognition, measurements and disclosure differences as they relate to the company are described in note 25 to our 2010 consolidated financial statements included elsewhere in this annual report.

About Forward-Looking Statements

This document contains forward-looking statements, which reflect our current expectations regarding future events. Forward-looking statements may include words such as "anticipate", "believe", "could", "expect", "foresee", "goal", "guidance", "intend", "may", "objective", "outlook", "plan", "seek", "should", "strive", "target" and "will".

Forward-looking statements involve risks and uncertainties, many of which are discussed in this MD&A. Results or performance may differ significantly from expectations. For example, the results of current clinical trials cannot be foreseen, nor can changes in policy or actions taken by regulatory authorities such as the FDA, the EMA, the Therapeutic Products Directorate of Health Canada or any other organization responsible for enforcing regulations in the pharmaceutical industry. Additionally, expected adjustments related to our conversion to International Financial Reporting Standards ("IFRS"), discussed below, that likely will impact various components of our future earnings and are referred to in our discussion of future expectations are unaudited, may not be complete and are subject to further review.

Given these uncertainties and risk factors, readers are cautioned not to place undue reliance on any forward-looking statements. We disclaim any obligation to update any such factors or to publicly announce any revisions to any of the forward-looking statements contained herein to reflect future results, events or developments, unless required to do so by a governmental authority or by applicable law.

About Material Information

This MD&A includes information that we believe to be material to investors after considering all circumstances, including potential market sensitivity. We consider information and disclosures to be material if they result in, or reasonably would be expected to result in, a significant change in the market price or value of our securities, or where it is likely that a reasonable investor would consider the information and disclosures to be important in making an investment decision.

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The Company is a reporting issuer under the securities legislation of all of the provinces of Canada, and its securities are registered with the United States Securities and Exchange Commission. The Company is therefore required to file or provide continuous disclosure information such as interim and annual financial statements, MD&As, proxy circulars, annual reports on Form 20-F, material change reports and press releases with the appropriate securities regulatory authorities. Copies of these documents may be obtained free of charge upon request from the Company's Investor Relations department or on the Internet at the following addresses: www.aezsinc.com, www.sedar.com and www.sec.gov.

Company Overview

Aeterna Zentaris Inc. (Nasdaq: AEZS and TSX: AEZ) is 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, 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. 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 an 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.

We are currently at 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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Key Developments for the year ended December 31, 2010

Drug Development

Status of our drug pipeline as at March 22, 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 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

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 also is 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 and to Handok for Korea.

On January 25, 2010, we announced that Keryx, our partner and licensee in North America, had reported a statistically significant benefit in survival from updated results of a Phase 2 study of perifosine in the treatment of advanced metastatic colorectal cancer. Results showed improvement in

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both time to tumor progression and overall survival in the perifosine + capecitabine arm, versus the placebo + capecitabine arm. Of notable interest, and for the first time presented, were data showing a statistically significant benefit in median overall survival (15.3 months vs. 6.8 months p=0.0088) and time to progression (18 weeks vs. 10 weeks p=0.0004) for the subset of patients who were refractory to a 5-FU (Fluorouracil) chemotherapy-based treatment regimen.

On January 29, 2010, we announced the publication in the February 2010 issue of the *Journal of Clinical Cancer Research* of positive Phase 2 results for perifosine as a single agent for the treatment of advanced Waldenstrom's macroglobulinemia. The data demonstrated a 35% overall response rate with a median progression-free survival of 12.6 months in patients with relapsed or relapsed/refractory Waldenstrom's macroglobulinemia.

On February 3, 2010, we announced that Keryx had reached an agreement with the FDA on an SPA for the Phase 3 X-PECT trial of perifosine in refractory advanced colorectal cancer, in addition to the earlier SPA agreement for the Phase 3 trial in multiple myeloma.

On March 1, 2010, we disclosed that the Committee for Orphan Medicinal Products of the EMA had issued a positive opinion for orphan medicinal product designation for perifosine for the treatment of multiple myeloma.

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, our partner, Keryx announced the initiation of a Phase 3 X-PECT registration trial with perifosine in refractory advanced colorectal cancer. The Phase 3 trial is being conducted pursuant to a SPA with the FDA. Approximately 40 to 50 U.S. sites will participate in the study.

On April 15, 2010, we received Positive Scientific Advice from the EMA for the Phase 3 program 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 April 20, 2010, at the AACR's annual meeting we presented data on our dual Erk/PI3K inhibitors and on our selective Erk inhibitors. Data supported further evaluation of selective Erk inhibitors as antiproliferative agents, either as monotherapy or in combination with inhibitors of the PI3K/Akt pathway. Other data resulted in the identification of AEZS-132, a unique dual inhibitor of PI3K and Erk with a favourable pharmacology and ADMET (Absorption, Distribution, Metabolism, Excretion, and Toxicity) profile for further evaluation as an antitumor agent. At that same meeting, preclinical data in neuroblastoma for perifosine were also presented. Data 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 dose limiting toxicity ("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 were treated thus far: high-grade glioma (5),

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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 4 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 tolerated in children with recurrent solid tumors and that these early signals of activity warrant further investigation in patients with advanced neuroblastoma and select brain tumors. Previously, perifosine has been shown to target activation of Akt in neuroblastoma cells and xenografts and to significantly inhibit tumor growth *in vivo* and improve the survival of mice bearing neuroblastoma tumors.

On June 8, 2010, we reported Phase 2 results 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 (Xeloda®), in the treatment of advanced metastatic colorectal cancer.

In this randomized, double-blind, placebo-controlled study, conducted at 11 centers across the United States, heavily pre-treated patients with second- or third-line metastatic colorectal cancer were randomized to receive capecitabine (Xeloda®) at 825 mg/m2 BID (total daily dose of 1650 mg/m2) on days 1 - 14, every 21 days, plus either perifosine or placebo at 50 mg daily. Of the 38 patients enrolled, 35 patients were evaluable for response (20 patients on the perifosine + capecitabine arm and 15 patients on the placebo + capecitabine arm). 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.

On July 14, 2010, our partner, Keryx, was granted orphan-drug designation by the FDA for perifosine for the treatment of neuroblastoma, a cancer of the nervous system affecting mostly children and infants for which there are no FDA-approved therapies.

On November 17, 2010, at the EORTC-NCI-AACR Symposium on Molecular Targets and Cancer Therapeutics in Berlin, Germany, we presented an abstract on perifosine combined with antimetabolites which induces synergistic effects on cytotoxicity and apoptosis in human colon, multiple myeloma, breast, renal, and liver tumor cell lines.

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On December 6, 2010 at the ASH's 52nd annual meeting in Orlando, we announced positive safety and tolerability Phase 2 data for perifosine in patients with advanced CLL and HL, as well as positive Phase 1 results of perifosine in combination with lenalidomide (Revlimid®) + dexamethasone in patients with relapsed or refractory multiple myeloma.

In the first Phase 2 study related to CLL, 12 patients with advanced CLL began treatment with single agent perifosine at 50 mg BID. Patients on study were heavily pre-treated having had a median of four prior lines of therapy with 75% of patients classified as Rai stage IV. One patient achieved a partial response (5 months on treatment) and 5 additional patients achieved stable disease (median duration of 4.25 months), for an overall 50% clinical benefit rate (PR + SD). Perifosine was well tolerated with minimal dose modifications.

In the second study presented, 26 patients were enrolled in a Phase 2 study with advanced lymphoma (6 non-HL, 4 CLL, 1 Waldenstrom's Macroglobulinemia and 15 HL). 73% of patients were previously refractory to their prior therapy, with 85% of patients having had 4 or more prior therapies. Perifosine (50 mg BID) was started as a single agent for 28 days; after 28 days, patients achieving partial response (PR) or better were continued on single agent perifosine. Patients achieving less than a PR were given the combination of perifosine (50 mg BID) plus sorafenib (Nexavar®) at 400 mg BID. All of the 4 CLL patients in this study achieved a partial response on single-agent perifosine within one month of treatment and remained on perifosine single agent. Response durations for each of the 4 patients were 4, 8, 9+ and 12 months. The remaining 22 patients were administered the combination with sorafenib, where 5 of the 15 (33%) HL patients achieved a partial response with a median response duration of 9 months. An additional 6 patients receiving the combination (40%) achieved stable disease.

The combination was well tolerated with no unexpected safety events.

The investigators concluded that perifosine in combination with sorafenib has significant anti-lymphoma activity in relapsed/refractory HL, and that perifosine as a single agent induced prolonged responses in high-risk, heavily pretreated CLL patients.

With regard to multiple myeloma, the final data set from the Phase 1 study of perifosine + lenalidomide (Revlimid®) + dexamethasone were also presented during the ASH meeting. 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 + dexamethasone was well tolerated even at the highest doses used, and demonstrated encouraging clinical activity and survival.

AEZS-108

AEZS-108 has been studied in gynecological cancers and has been shown to be effective and well tolerated in advanced endometrial and ovarian cancers. Positive Phase 2 results for ovarian cancer were disclosed in June 2010 at the annual ASCO meeting, while positive Phase 2 results for endometrial cancer were presented at the EORTC-NCI-AACR International Symposium on Molecular Targets and Cancer Therapeutics in November 2010. In addition to the ongoing Phase 1/2 studies in refractory bladder cancer and castration refractory prostate cancer, we intend to initiate discussions with the FDA and the EMA in an effort to reach an agreement on a protocol for a registration trial in endometrial cancer.

On May 6, 2010, we announced that we had received orphan drug designation from the FDA for AEZS-108 for the treatment of ovarian cancer. Orphan drug designation is granted by the FDA's Office of Orphan Products Development to novel drugs or biologics that treat a rare disease or condition affecting fewer than 200,000 patients in the US. The designation provides a drug developer with a

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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 or efficacy versus another drug of its type previously granted the designation for the same indication.

On May 12, 2010, we announced that the FDA had approved our IND application for AEZS-108 in LHRH receptor-positive urothelial (bladder) cancer. Following this approval from the FDA, this trial will be conducted at the Sylvester Comprehensive Cancer Center at the University of Miami's Miller School of Medicine, and will include up to 64 patients, male and female, with advanced LHRH receptor-positive urothelial (bladder) cancer. The study will be conducted in two parts: first, a dose-finding part in up to 12 patients; subsequently, a selected dose will be studied for its effect on progression-free survival.

On May 17, 2010, we announced that we had received a positive opinion for orphan medicinal product designation from the COMP of the EMA, for AEZS-108 for the treatment of ovarian cancer. 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 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 European Union for AEZS-108, once approved for the treatment of ovarian cancer.

On June 7, 2010, Prof. Günter Emons, Chairman, Department of Obstetrics & Gynaecology Georg-August University Göttingen, Germany, presented positive efficacy and safety data for AEZS-108 in ovarian cancer at the ASCO Annual Meeting. The poster (abstract #5035), entitled "Phase 2 study of AEZS-108, a targeted cytotoxic LHRH analog, in patients with LHRH receptor-positive platinum resistant ovarian cancer" (G. Emons, S. Tomov, P. Harter, J. Sehouli, P. Wimberger, A. Staehle, L. C. Hanker, F. Hilpert, P. Dall and C. Gruendker, for the AGO Study Group), details the use of AEZS-108 in women with histologically confirmed taxane-pretreated platinum-resistant/refractory LHRH receptor-positive advanced (FIGO III or IV) or recurrent ovarian cancer. Patients received a recommended dose of 267 mg/m^2 by intravenous infusion over 2 hours, with retreatment every 3 weeks, for up to 6 courses. Response rate (RECIST and/or GCIG criteria) was defined as the primary endpoint. Secondary endpoints were safety, time-to-progression and overall survival.

42 patients with platinum-resistant ovarian cancer entered the study. Efficacy included partial response in 5 patients (11.9%) and stable disease for more than 12 weeks in 11 patients (26.2%). Based on those data, a Clinical Benefit Rate of 38% can be estimated. Median time to progression and overall survival were 3.5 months (104 days) and 15.6 months (475 days), respectively. Overall survival compares favourably with data from Doxil and Topotecan (8-9 months). In all, tolerability of AEZS-108 was good and commonly allowed retreatment as scheduled. Only one patient (2.4%) had a dose reduction, and overall, 25 of 170 (14.7%) courses were given with a delay, including also cases in which delay was not related to toxicity. Severe (Grade 3 or 4) toxicity was mainly restricted to rapidly reversible hematologic toxicity (leukopenia / neutropenia) associated with fever in 3 cases. Good tolerability of AEZS-108 was also reflected with only a few patients with non-hematological toxicities of grade 3 (none with Grade 4), including single cases (2.4%) each of nausea, constipation, poor general condition, and an enzyme elevation. No cardiac toxicity was reported.

On June 28, 2010, we announced that we had concluded an agreement with Almac's Diagnostics division for AEZS-108, aimed at determining LHRH receptor expression through the development of a companion diagnostic tool. Selection for treatment with AEZS-108 is determined on the basis of LHRH receptor expression, currently measured immunohistochemically. In humans, LHRH receptors

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are expressed in ovarian, endometrial, breast, bladder, prostate and pancreatic tumors. This state of the art companion diagnostic tool will allow us to develop improved methods of selecting the most appropriate patients to be treated with AEZS-108 in order to enhance the efficiency of our clinical trials and help us with the future successful development of AEZS-108 in a number of different LHRH expressing cancers.

On August 5, 2010, we announced that the NIH had awarded Dr. Jacek Pinski, Associate Professor of Medicine at the Norris Comprehensive Cancer Center of the University of Southern California, a grant of approximately $1.5 million over three years to conduct a Phase 1/2 study in refractory prostate cancer with AEZS-108. The study, entitled *A Phase I/II Trial of AN-152 [AEZS-108] in Castration- and Taxane-Resistant Prostate Cancer*, will enrol up to 55 patients and will be conducted in two portions: an abbreviated dose-escalation followed by a single arm, Simon Optimum two-stage design Phase 2 study using the dose selected in the Phase 1 portion. The primary objective of the Phase 2 portion is to evaluate the clinical benefit of AEZS-108 in men with castration- and taxane-resistant metastatic prostate cancer, for which the presence of LHRH receptors has been confirmed.

On November 18, 2010, Prof. Günter Emons of the Department of Obstetrics & Gynaecology Georg-August at the University of Göttingen (Germany) presented positive data for the Phase 2 of AEZS-108 in Advanced Endometrial Cancer at the EORTC-NCI-AACR symposium in Berlin, Germany. The study showed encouraging results as AEZ-108 was used as a single agent.

Of 43 patients treated with AEZS-108 in this study, 39 were evaluable for efficacy. Responses confirmed by independent review included 2 patients with complete response (CR; 5.1%), 10 patients with partial response (PR; 25.6%), and 17 patients with stable disease (SD; 43.6%). Based on those data, an overall response rate (ORR = CR+PR) of 30.8% and a clinical benefit rate (CBR = CR+PR+SD) of 74.4% can be estimated. Responses were also achieved in patients with prior chemotherapy, 1 CR, 1 PR and 2 SDs in 8 of the patients pre-treated with platinum/taxane regimens. Median time to progression and overall survival were 7 months (30 weeks) and 14.3 months (62 weeks), respectively. Conclusions from this trial were as follows:

AEZS-108 at a dosage of 267 mg/m2 every 3 weeks was active and well tolerated in patients with endometrial cancer;

Hematological toxicity was rapidly reversible, and non-hematological toxicities were usually not severe, causing few deviations from scheduled treatment;

The objective response rate of 30.8% compares well with those of single agent platinum or taxane treatment; responders included patients pre-treated with platinum/taxane combination; and

The rate of stable disease was 43.6%, resulting in a Clinical Benefit Rate of 74.4%.

The overall survival after single agent AEZS-108 is similar to that reported for modern triple combination chemotherapy, but was achieved with lower toxicity.

On December 14, 2010, we announced the initiation of a Phase 1/2 trial in castration refractory prostate cancer conducted by Dr. Jacek Pinski at the Norris Comprehensive Cancer Center, as well as a Phase 1/2 trial in refractory bladder cancer conducted by Dr. Gustavo Fernandez at the Sylvester Comprehensive Cancer Center.

AEZS-130/Solorel®

AEZS-130/Solorel® (macimorelin), 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.

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On June 21, 2010, we presented positive data at the 92nd ENDO Meeting and Expo on AEZS-130 for diagnostic and therapeutic use. The preclinical data showed that AEZS-130 is a potent and safe oral synthetic GH-releasing compound with potential utility as a diagnostic test for growth hormone deficiencies. In addition to the diagnostic indication, we believe that, based on the results of Phase 1 studies, AEZS-130 (Solorel®) 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.

On July 14, 2010, we announced the presentation of a poster on Solorel®, entitled *Use of the Orally Active Ghrelin Mimetic AEZS-130 as a Simple Test for the Diagnosis of Growth Hormone (GH) Deficiency (GHD) in adults (AGHD)*, Merriam G.R., Yuen K., Bonert V., Dobs A, Garcia J., Kipnes M., Molitch M., Swerdloff R., Wang C., Cook D., Altemose I. and Biller B. This poster was presented at the Seventh International Congress of Neuroendocrinology, in Rouen, France.

On October 5, 2010, we announced at the Fifth International Congress of the Growth Hormone Research Society and the Insulin-like Growth Factors Society, after the interim Phase 3 analysis of the orphan drug AEZS-130, that it demonstrated the potential to provide a simple, well tolerated and safe oral diagnostic test for AGHD. Solorel® has been granted orphan drug designation by the FDA as a diagnostic test.

Corporate developments

On April 20, 2010, we completed a registered direct offering of 11,111,111 units, with each unit consisting of one common share and a warrant to purchase 0.40 of a common share, at a price of $1.35 per unit (the "April 2010 Offering"). Total proceeds raised upon completion of the April 2010 Offering amounted to $15.0 million less cash transaction costs of approximately $1.3 million. The securities described above were offered by us pursuant to a shelf prospectus dated March 12, 2010 and a prospectus supplement dated April 15, 2010.

We granted warrants (the "April 2010 Investor Warrants") to the investors who participated in the April 2010 Offering. Each April 2010 Investor Warrant entitles the holder to purchase 0.40 of a common share at an exercise price of $1.50 per share. The April 2010 Investor Warrants are exercisable between October 20, 2010 and October 20, 2015, and, upon complete exercise, would result in the issuance of an aggregate of 4,444,444 common shares.

We estimated the fair value attributable to the April 2010 Investor Warrants of $3.6 million as at the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 2.56%, expected volatility of 87.3%, an expected term of 5 years, a dividend yield of 0.0% and an issue-date market share price of $1.24. Transaction costs allocated to the April 2010 Investor Warrants amounted to approximately $0.3 million.

On June 21, 2010, we completed a registered direct offering of 8,805,964 units, with each unit consisting of one common share and a warrant to purchase 0.50 of a common share, at a price of $1.3725 per unit (the "June 2010 Offering"). Total proceeds raised upon completion of the June 2010 Offering amounted to $12.1 million, less cash transaction costs of approximately $0.8 million. The securities described above were offered by us pursuant to a shelf prospectus dated March 12, 2010 and a prospectus supplement dated June 15, 2010.

We granted warrants (the "June 2010 Investor Warrants") to the investors who participated in the June 2010 Offering. Each June 2010 Investor Warrant entitles the holder to purchase a common share at an exercise price of $1.3725 per share. The June 2010 Investor Warrants are exercisable between June 21, 2010 and June 21, 2015, and, upon complete exercise, would result in the issuance of an aggregate of 4,402,982 common shares.

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We estimated the fair value attributable to the June 2010 Investor Warrants of $3.5 million as at the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 2.05%, expected volatility of 89.3%, an expected term of 5 years, a dividend yield of 0.0% and an issue-date market share price of $1.18. Transaction costs allocated to the June 2010 Investor Warrants amounted to approximately $0.2 million.

We also granted warrants (the "June 2010 Compensation Warrants") to the sole placement agent (and to certain of its designated representatives) engaged in connection with the June 2010 Offering. Each June 2010 Compensation Warrant entitles the holder to purchase a common share at an exercise price of $1.7156 per share. The June 2010 Compensation Warrants are exercisable between June 15, 2010 and June 15, 2015, and, upon complete exercise, would result in the issuance of 264,178 common shares.

We estimated the fair value attributable to the June 2010 Compensation Warrants of $0.2 million as at the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 2.04%, expected volatility of 89.4%, an expected term of 5 years, a dividend yield of 0.0% and an issue-date market share price of $1.18. The initial fair value of the June 2010 Compensation Warrants has been accounted for as additional transaction costs, since the instruments were granted to the sole placement agent as part of the terms of the underlying engagement and in recognition of the efforts made in connection with the June 2010 Offering.

On June 23, 2009, we completed a registered direct offering of 5,319,149 units, with each unit consisting of one common share and a warrant to purchase 0.35 of a common share at a price of $1.88 per unit (the "June 2009 Offering"). Total proceeds raised through the June 2009 Offering amounted to $10.0 million, less cash and non-cash transaction costs of $1.6 million. The purchasers in this offering were comprised of institutional investors, and the securities described above were offered by us pursuant to a shelf prospectus dated September 27, 2007 and a prospectus supplement dated June 18, 2009.

We granted a total of 5,319,149 warrants (the "June 2009 Investor Warrants") to the institutional investors who participated in the June 2009 Offering. Each June 2009 Investor Warrant entitles the holder to purchase 0.35 of a common share at an exercise price of $2.06 per share. The June 2009 Investor Warrants are exercisable between September 23, 2009 and December 23, 2011, and, upon complete exercise, would result in the issuance of an aggregate of 1,861,702 common shares of the Company.

We estimated the fair value attributable to the June 2009 Investor Warrants of $1.6 million as at the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 1.74%, expected volatility of 90.6%, an expected term of 2.5 years, dividend yield of 0.0% and an issue-date market share price of $1.75. Transaction costs allocated to the June 2009 Investor Warrants amounted to approximately $0.2 million.

We granted a total of 820,668 warrants (the "June 2009 Compensation Warrants") to the sole placement agent and its designated representatives engaged in connection with the June 2009 Offering. Each June 2009 Compensation Warrant entitles the holder to purchase 0.35 of a common share at an exercise price of $2.35 per share. The June 2009 Compensation Warrants are exercisable between December 23, 2009 and December 23, 2011, and, upon complete exercise, would result in the issuance of 287,234 common shares of the Company.

We estimated the fair value attributable to the June 2009 Compensation Warrants of $0.2 million as at the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 1.74%, expected volatility of 90.6%, an expected term of 2.5 years, an expected dividend yield of 0.0% and an issue-date market share price of

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$1.75. The initial fair value of the June 2009 Compensation Warrants has been accounted for as additional transaction costs, since the instruments were granted to the sole placement agent as part of the terms of the underlying engagement and in recognition of the efforts made in connection with the June 2009 Offering.

On October 23, 2009, we completed a registered direct offering of 4,583,335 units, with each unit consisting of one common share and a warrant to purchase 0.40 of a common share, at a price of $1.20 per unit (the "October 2009 Offering"). Total proceeds raised through the October 2009 Offering amounted to $5.5 million, less cash transaction costs of approximately $0.4 million. The purchasers in this offering were new and existing institutional investors, and the securities described above were offered by us pursuant to a shelf prospectus dated September 27, 2007 and a prospectus supplement dated October 19, 2009.

We granted a total of 4,583,335 warrants (the "October 2009 Investor Warrants") to the institutional investors who participated in the October 2009 Offering. Each October 2009 Investor Warrant entitles the holder to purchase 0.40 of a common share at an exercise price of $1.25 per share. The October 2009 Investor Warrants are exercisable between October 23, 2009 and October 23, 2014, and, upon complete exercise, would result in the issuance of an aggregate of 1,833,334 common shares.

We estimated the fair value attributable to the October 2009 Investor Warrants of $1.3 million as of the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 2.46%, expected volatility of 84.3%, an expected term of 5 years, dividend yield of 0.0% and an issue-date market share price of $1.09. Transaction costs allocated to the October 2009 Investor Warrants amounted to approximately $0.1 million.

We granted a total of 320,832 warrants (the "October 2009 Compensation Warrants") to the sole placement agent engaged in connection with the October 2009 Offering. Each October 2009 Compensation Warrant entitles the holder to purchase 0.40 of a common share at an exercise price of $1.50 per share. The October 2009 Compensation Warrants are exercisable between April 23, 2010 and October 23, 2012, and, upon complete exercise, would result in the issuance of 128,333 common shares.

We estimated the fair value attributable to the October 2009 Compensation Warrants of $86,653 as at the date of grant by applying the Black-Scholes pricing model, to which the following additional assumptions were applied: a risk-free annual interest rate of 1.57%, expected volatility of 103.4%, an expected term of 3 years, dividend yield of 0.0% and an issue-date market share price of $1.09. The initial fair value of the October 2009 Compensation Warrants has been accounted for as additional transaction costs, since the instruments were granted to the sole placement agent as part of the terms of the underlying engagement and in recognition of the efforts made in connection with the October 2009 Offering.

The terms of all aforementioned warrants are substantially the same, with the exception of the exercise price and contractual period of exercise, as discussed above. In particular, all warrants may be exercised, at the option of the holder, by cash payment of the exercise price or, upon the existence of certain conditions, by "cashless exercise", which means that in lieu of paying the aggregate exercise price for the shares being purchased upon exercise of the warrants in cash, the holder would receive the number of shares underlying the warrants equal to the quotient obtained by applying a formula, as defined by the terms of each warrant. We will not receive additional proceeds to the extent that warrants are exercised by cashless exercise.

The exercise price and number of common shares issuable on exercise of all outstanding warrants may be adjusted in certain circumstances, including stock dividends or splits, subsequent rights offerings, pro-rata distributions and pursuant to transactions involving the merger or consolidation of the Company with another entity or other Fundamental Transaction, as defined in the warrants.

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Additionally, and notwithstanding anything to the contrary, in the event of any type of Fundamental Transaction, as defined in the warrants, the Company or any successor entity shall, at our option, have the right to require the holders thereof to exercise the warrants, or, at the holder's option, purchase the warrants from the holders by paying the holders an amount of cash equivalent to the Black-Scholes value, as defined, of the remaining unexercised portion of the warrants on the date of the consummation of an aforementioned Fundamental Transaction.

Subsequent to year-end

On February 22, 2011, we entered into an "At-the-Market" ("ATM") sales agreement, under which we may, at our discretion, from time to time during the 24-month term of the agreement, sell up to a maximum of 12.5 million of our common shares through ATM issuances on the Nasdaq for aggregate gross proceeds not to exceed $19.8 million, being the amount remaining available for distribution, as at February 22, 2011, under our current registration statement on Form F-3. The common shares will be sold at market prices prevailing at the time of a sale of the common shares, and, as a result, prices may vary.

On March 10, 2011, we issued approximately 1.7 million common shares in connection with the aforementioned ATM agreement, for gross proceeds of approximately $3.2 million.

On March 9, 2011, we announced that we had entered into an agreement with Yakult for the development, manufacture and commercialization of perifosine in all human uses, excluding leishmaniasis, in Japan. Under the terms of this agreement, Yakult will make an initial non-refundable upfront payment to us of €6.0 million (approximately $8.3 million). Also per the agreement, we will be entitled to receive up to a total of €44.0 million (approximately $60.9 million) upon achieving certain pre-established milestones, including clinical and regulatory events in Japan. Furthermore, we will be entitled to receive double-digit royalties on future net sales of perifosine in the Japanese market. We have also agreed to supply perifosine to Yakult on a cost-plus-basis.

Results of Operations

Quarterly Consolidated Results of Operations Information

(in thousands, except for per share data)

	Quarters ended			
	December 31, 2010	**September 30, 2010**	**June 30, 2010**	**March 31, 2010**
	$	**$**	**$**	**$**
Revenues	**9,971**	5,726	5,584	6,422
Loss from operations	**(4,211)**	(6,088)	(7,589)	(7,340)
Net loss	**(2,741)**	(10,147)	(4,450)	(5,880)
Net loss per share				
Basic and diluted	**(0.03)**	(0.12)	(0.06)	(0.09)

	Quarters ended			
	December 31, 2009	**September 30, 2009**	**June 30, 2009**	**March 31, 2009**
	$	**$**	**$**	**$**
Revenues	40,182	8,565	8,379	6,111
Earnings (loss) from operations	11,511	(9,789)	(12,238)	(13,442)
Net earnings (loss)	12,032	(11,288)	(13,080)	(12,388)
Net earnings (loss) per share				
Basic and diluted	0.19	(0.19)	(0.24)	(0.23)

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Net earnings (loss) per share are (is) based on each reporting period's weighted average number of shares outstanding, which may differ on a quarter-to-quarter basis. As such, the sum of the quarterly net earnings (loss) per share amounts may not equal year-to-date net loss per share.

Fourth Quarter 2010 Results

Revenues were $10.0 million for the quarter ended December 31, 2010, compared to $40.2 million for the same quarter in 2009. The significant decrease in revenues is due primarily to our having recognized, in December 2009, the remaining unamortized portion, or approximately $30.4 million, of the upfront payment received from sanofi-aventis U.S L.L.C. ("sanofi"), as discussed below, partly offset by the increase in 2010 royalties attributable to the contingent payment of $2.5 million due from Cowen, also as discussed below. Additionally, the decrease is attributable to the recognition of remaining deferred revenues, amounting to approximately $1.8 million, associated with agreements related to the use of ozarelix, an intangible asset that was deemed to be fully impaired in December 2009.

Net research and development ("R&D") expenses were $5.1 million for the quarter ended December 31, 2010, compared to $10.6 million for the same quarter in 2009. The comparative decrease in R&D expenses primarily results from the progressive completion through the end of 2009 of efficacy and safety studies associated with our Phase 3 program for cetrorelix in benign prostatic hyperplasia ("BPH"). The decrease is also explained by a comparatively lower overall volume of R&D expenses, most notably given the fact that most costs related to our ongoing Phase 3 program with perifosine are borne by our North American partner, Keryx.

Selling, general and administrative ("SG&A") expenses were $3.1 million for the quarter ended December 31, 2010, compared to $6.2 million for the same quarter in 2009. The decrease in SG&A expenses is predominantly related to the expensing, in December 2009, of the remaining unamortized portion, or approximately $3.0 million, of the royalty paid to the Tulane Educational Fund ("Tulane") in connection with the agreement entered into with, and subsequently terminated by, sanofi, as discussed below.

Depreciation and amortization expenses for the quarter ended December 31, 2010 amounted to $0.6 million, compared to $8.1 million for the same quarter in 2009. The comparative decrease is attributable to the fact that, in December 2009, and following our announcements that our second Phase 3 study with cetrorelix in BPH had not reached its primary endpoint and that sanofi had decided to terminate the related development, commercialization and license agreement (discussed below), we recognized an impairment charge equivalent to the remaining carrying value of cetrorelix, or approximately $3.9 million, as part of amortization expense. Further, in January 2010, Spectrum Pharmaceuticals Inc., to whom we have granted an exclusive license to develop and commercialize ozarelix for all potential indications in all worldwide territories, excluding certain Asian markets, announced that it had terminated its development program with ozarelix in BPH. Consequently, we recognized an impairment charge of approximately $1.4 million as part of amortization expense in the fourth quarter of 2009.

Net (loss) earnings amounted to ($2.7 million), or ($0.03) per basic and diluted share, for the quarter ended December 31, 2010, compared to $12.0 million, or $0.19 per basic and diluted share, for the same quarter in 2009. The significant quarter-over-quarter decrease in net earnings is largely attributable to the significant decrease in license fee revenues, partly offset by lower comparative R&D expenses and by decreased SG&A expenses and depreciation and amortization charges, as discussed above.

Largely given the presence, in our fourth quarter 2010 revenues, of the $2.5 million in additional, non-recurring royalty consideration payable by Cowen (discussed below), and excluding any impact of

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foreign exchange gains or losses, we expect that the net loss for the first quarter of 2011 will increase, as compared to the fourth quarter of 2010.

Consolidated Statements of Operations Information

(in thousands, except for share and per share data)

	Years ended December 31,		
	2010	**2009**	**2008**
	$	**$**	**$**
Revenues			
Sales and royalties	**24,857**	20,957	29,462
License fees and other	**2,846**	42,280	9,016
	27,703	63,237	38,478
Operating expenses			
Cost of sales, excluding depreciation and amortization	**18,700**	16,501	19,278
Research and development costs, net	**19,859**	43,814	57,105
Selling, general and administrative expenses	**11,875**	16,040	17,325
Depreciation and amortization			
Property, plant and equipment	**1,005**	3,285	1,515
Intangible assets	**1,492**	7,555	5,639
	52,931	87,195	100,862
Loss from operations	**(25,228)**	(23,958)	(62,384)
Other income (expenses)			
Unrealized gain on held-for-trading financial instrument	**687**		
Interest income	**207**	349	868
Interest expense	**(26)**	(5)	(118)
Foreign exchange gain (loss)	**1,170**	(1,110)	3,071
Other	**(28)**		(79)
	2,010	(766)	3,742
Loss before income taxes	**(23,218)**	(24,724)	(58,642)
Income tax expense			(1,175)
Net loss	**(23,218)**	(24,724)	(59,817)
Net loss per share			
Basic and diluted	**(0.31)**	(0.43)	(1.12)
Weighted average number of shares			
Basic and diluted	**75,659,410**	56,864,484	53,187,470

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Revenues

Revenues are derived primarily from sales and royalties as well as from license fees. Sales are derived from the manufacturing of Cetrotide®, marketed for reproductive health assistance for *in vitro* fertilization, as well as from active pharmaceutical ingredients.

Royalties are derived from Cetrotide® and, prior to the fourth quarter of 2008, were payable by our partner, ARES Trading S.A. ("Merck Serono"). Beginning on October 1, 2008, royalty revenues derived from Merck Serono's net sales of Cetrotide® are recognized via the periodic amortization, under the units-of-revenue method, of proceeds received in connection with the sale in December 2008 of the underlying future royalty stream to Cowen. Per the terms of the related purchase and sale agreement, we received net proceeds of $51.5 million from Cowen and were entitled to receive an additional payment of $2.5 million contingent on 2010 net sales of Cetrotide® reaching a specified level. This additional consideration was earned in 2010 and received in February 2011, and the corresponding amount has been recorded as royalty revenues in our consolidated statement of operations.

License fees are derived from non-periodic milestone payments, R&D contract fees and upfront payments, and amortization thereof, received from our licensing partners. Significant license fee revenues have resulted from our agreement with sanofi, related to the development, commercialization and licensing of cetrorelix in BPH, entered into in March 2009 and terminated in January 2010 following our announcement that our second Phase 3 study with the compound had not reached its primary endpoint.

Sales and royalties increased to $24.9 million for the year ended December 31, 2010, compared to $21.0 million and $29.5 million for each of the years ended December 31, 2009 and 2008, respectively. In addition to the recognition, in 2010, of the additional royalty consideration payable by Cowen (discussed above), 2010 sales and royalties were positively impacted by an increase in Cetrotide® sales in non-Japanese markets, as compared to 2009.

The decrease from 2008 to 2009 is mainly related to lower royalty revenues having been recognized in 2009 in connection with our agreement with Merck Serono. Amortization of the proceeds received from Cowen for the year ended December 31, 2009 was lower than the royalty revenues generated and payable directly by Merck Serono during 2008. Additionally, sales volumes of Cetrotide® were slightly lower during the year ended December 31, 2009, as compared to 2008.

Excluding the impact of foreign exchange rate fluctuations, sales and royalties are expected to decrease in 2011 to between approximately $19.0 million and $21.0 million, largely given the future absence of the non-recurring contingent consideration payable by Cowen, as discussed above.

License fee and other revenues totalled $2.8 million for the year ended December 31, 2010, compared to $42.3 million and $9.0 million for each of the years ended December 31, 2009 and 2008, respectively. The significant decrease from 2009 to 2010, as well as the significant increase from 2008 to 2009, is almost exclusively attributable to the upfront payment received and recognized in 2009 from sanofi, as well as from the full recognition of other previously deferred revenues associated with ozarelix, another BPH-related compound that was deemed impaired in December 2009.

License fee revenues in 2011 are expected to be largely similar to the amounts recorded during 2010, excluding any revenue associated with the licensing agreement entered into with Yakult, discussed above.

Operating Expenses

Cost of sales increased to $18.7 million (75% of sales and royalties) for the year ended December 31, 2010 from $16.5 million (79% of sales and royalties) and $19.3 million (65% of sales and royalties) for each of the years ended December 31, 2009 and 2008, respectively. Excluding the impact

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of the additional contingent royalty consideration earned and payable by Cowen (discussed above), cost of sales as a percentage of sales and royalties for the year ended December 31, 2010 was approximately 84%, representing a consistently comparable increase from 2009. Changes in margin are dependent both upon our sales mix (including dosing level) and upon geographic coverage, both of which tend to vary on a period-to-period basis.

The decrease from 2008 to 2009 is largely attributable to the absence of Impavido® sales during the first three months of 2009, compared to the same period in 2008. We sold our rights related to the manufacture, production, distribution, marketing, sale and use of that compound in March, 2008. The increase in cost of sales as a percentage of sales and royalties from 2008 to 2009 is largely attributable to the comparative decrease in royalty revenues, as discussed above.

We expect cost of sales as a percentage of sales and royalties to remain within the range of 80% to 85% during 2011, as compared to 2010.

R&D costs, net of tax credits and grants include: employee compensation and fringe benefits; third-party costs; building rental, service and maintenance; and other expenses, as shown in the first table below. Third-party R&D costs consist of, among other expenses, external studies and collaborative work performed by contract research organizations, laboratory supplies and services, active pharmaceutical ingredient (or raw material) costs and patent protection expenses. These third-party costs are tracked by product or project, as shown in the second table below. All other R&D costs, including Company employee compensation and benefits, are not charged to specific products or projects, since the number of clinical and preclinical product candidates or development projects tends to vary from period to period and since internal resources are utilized across multiple projects over any given period of time.

Net R&D costs were $19.9 million for the year ended December 31, 2010, compared to $43.8 million and $57.1 million for each of the years ended December 31, 2009 and 2008, respectively. The decrease from 2009 to 2010 is primarily attributable to the winding down and termination of development activities related to cetrorelix in BPH subsequent to our announcements that our related Phase 3 studies had not reached their primary endpoints in 2009. The decrease is also explained by a comparatively lower overall volume of R&D expenses, most notably given the fact that most costs related to our ongoing Phase 3 program with perifosine are borne by our North American partner, Keryx.

The decrease in R&D costs from 2008 to 2009 is largely attributable to a lower volume of expenses having been incurred in 2009 related to the continued advancement during the first nine months of 2009, followed by the winding down of our development activities linked to cetrorelix in BPH.

The following table summarizes our net R&D costs by nature of expense:

	Years ended December 31,		
	2010	**2009**	**2008**
	$	**$**	**$**
		(in thousands)	
Employee compensation and fringe benefits	**9,153**	10,845	14,088
Third-party costs	**8,138**	28,925	39,142
Facilities rent and maintenance	**1,773**	1,891	1,883
Other costs	**1,482**	2,556	2,335
R&D tax credits and grants	**(687)**	(403)	(343)
	19,859	43,814	57,105

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The following table summarizes primary third-party R&D costs, by product, incurred by the Company during the years ended December 31, 2010, 2009 and 2008.

Product	Status	Indication	Years ended December 31, 2010		2009		2008	
			$	%	$	%	$	%
			(in thousands, except percentages)					
Cetrorelix	Phase 3*	BPH*	**2,046**	**25.1**	23,812	82.3	27,246	69.6
AEZS-130 (Solorel®)	Phase 3	Endocrinology (diagnosis of AGHD)	**865**	**10.6**	592	2.0		
Perifosine	Phases 2 and 3	Oncology	**968**	**11.9**	304	1.1	2,426	6.2
Ozarelix	Phase 2*	BPH*			366	1.3	254	0.7
AEZS-108	Phase 2	Oncology	**2,089**	**25.7**	409	1.4	1,300	3.3
AEZS-112	Phase 1	Oncology	**259**	**3.2**	430	1.5	996	2.5
AEZS-129, AEZS-131, AEZS-132, Erk PI3K	Preclinical	Endocrinology and oncology	**923**	**11.4**	1,151	4.0	1,991	5.1
AEZS-123 / Ghrelin receptor	Preclinical	Endocrinology and oncology			530	1.8	1,224	3.1
AEZS-115 / LHRH antagonist	Preclinical	Endocrinology and oncology			235	0.8	1,047	2.7
AEZS-120 / Vaccine	Preclinical	Oncology	**149**	**1.8**	403	1.4	27	
Other	Preclinical	Oncology and endocrinology	**839**	**10.3**	693	2.4	2,631	6.8
			8,138	**100.0**	28,925	100.0	39,142	100.0

* Development activities terminated in the last quarter of 2009 and beginning of 2010.

We expect that our total R&D expenses for 2011 will increase as compared to 2010, largely due to the expected incurrence of higher third-party costs associated with the advancement of our Phase 3 trial with perifosine in the multiple myeloma indication, and more specifically in connection with the clinical and validation initiatives related to that study. However, most of these expected third-party costs will be reimbursed by our partner, Keryx, and those reimbursements will be recorded as license fee and other revenues.

SG&A expenses decreased to $11.9 million for the year ended December 31, 2010, compared to $16.0 million and $17.3 million for each of the years ended December 31, 2009 and 2008, respectively. The decrease from 2009 to 2010 is related primarily to the absence, in 2010, of the royalty paid to Tulane, amounting to approximately $3.0 million, as noted above, to euro-to-US dollar exchange rate fluctuations, largely due to the comparative weakening in 2010 of the euro against the US dollar and to the continued implementation of general and administrative cost-saving measures.

The decrease from 2008 to 2009 is related to comparative euro-to-US dollar exchange rate fluctuations and to the absence in 2009 of certain non-recurring corporate expenses due to cost-saving measures that were implemented beginning in the second quarter of 2008, despite the additional selling expenses charged during 2009 as pertaining to the royalty paid to Tulane, as discussed above.

Depreciation and amortization expenses decreased to a combined $2.5 million for the year ended December 31, 2010, compared to $10.8 million and $7.2 million for each of the years ended December 31, 2009 and 2008, respectively. The significant decrease from 2009 to 2010 is attributable primarily to the absence, in 2010, of impairment charges.

The increase in depreciation and amortization expenses from 2008 to 2009 is attributable to the impairment charges related to cetrorelix, ozarelix and certain items of property, plant and equipment utilized exclusively in the development activities related to cetrorelix, as discussed above. This year-over-year increase was offset in large proportion by the impairment charge of $2.4 million

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recorded in 2008 related to teverelix, an intangible asset that was deemed impaired in the fourth quarter of 2008.

Loss from operations amounted to $25.2 million for the year ended December 31, 2010, compared to $24.0 million and $62.4 million for each of the years ended December 31, 2009 and 2008, respectively. Our loss from operations from 2009 to 2010 increased mainly due to the absence, in 2010, of licence fee revenues commensurate with the significant amounts recognized in 2009 and despite the comparative reductions in net R&D costs, SG&A expenses and depreciation and amortization charges.

The significant decrease from 2008 to 2009 in loss from operations is due to the significant year-over-year increase in license fee revenues, associated mainly with agreements for cetrorelix and ozarelix, combined with lower comparative R&D and SG&A expenses, partly offset by increased depreciation and amortization expenses and by lower comparative margins on Cetrotide® sales.

Other income (expenses) are comprised primarily of foreign exchange gains and losses, which result from the impact of changes in the value of currencies such as the US dollar and the Canadian dollar ("CAN$") against the Group's functional currency, the euro. Foreign exchange gains and losses are recorded upon settlement or revaluation of non-euro-denominated balances.

Foreign exchange gain (loss) amounted to $1.2 million for the year ended December 31, 2010, compared to ($1.1 million) and $3.1 million for each of the years ended December 31, 2009 and 2008, respectively.

During the first six months of 2010, the euro weakened progressively against the US dollar, losing approximately 14.2% of its value since December 31, 2009. During that time period, we recorded significant foreign exchange gains, mainly as a result of the periodic revaluation of our US dollar-denominated cash and cash equivalents, a significant portion of which resulted from the two registered direct offerings completed in April and June 2010, as noted above. However, the euro recovered partially during the second half of 2010, strengthening approximately 9.4% against the US dollar since June 30, 2010. As a result, we recorded substantial foreign exchange losses on transactions and balances denominated in US dollars during that period. Overall, the net depreciation in the euro against the US dollar during the twelve months ended December 31, 2010 amounted to approximately 6.6%, resulting in a net foreign exchange gain of $1.2 million.

On a comparative basis, the euro strengthened almost consistently against the US dollar on average during the twelve months ended December 31, 2009 by approximately 13.0%. As a result, net foreign exchange losses recorded were significantly higher during the year ended December 31, 2009, as compared to 2010, where we posted a net foreign exchange gain.

The year-end conversion rates from the euro and Canadian dollar to the US dollar can be summarized as follows:

	As at December 31,		
1 US dollar equivalent to:	**2010**	**2009**	**2008**
	$	**$**	**$**
Euro	**0.7468**	0.7007	0.7145
Canadian dollar	**0.9946**	1.0510	1.2180

Income tax expense of $1.2 million for the year ended December 31, 2008 is largely attributable to a minimum tax payable in Germany due to the tax accounting ramifications of the sale of future royalties to Cowen, referred to above.

In 2011, we do not expect to record any significant income tax recovery or expense in our foreign or domestic entities.

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Net loss was $23.2 million, or $0.31 per basic and diluted share, for the year ended December 31, 2010, compared to $24.7 million, or $0.43 per basic and diluted share, and $59.8 million, or $1.12 per basic and diluted share, for each of the years ended December 31, 2009 and 2008, respectively. The decrease in our 2010 net loss, as compared to 2009, is attributable to a reduction in net R&D costs, lower SG&A expenses and lower depreciation and amortization charges, as well as to higher net foreign exchange gains, as discussed above, offset by the significant reduction of licence fee revenues and a lower margin on sales of Cetrotide®.

The significant decrease in net loss from 2008 to 2009 is due to the significant year-over-year increase in license fee revenues, associated mainly with agreements for cetrorelix and ozarelix, combined with lower comparative R&D, SG&A and income tax expenses, partly offset by lower comparative sales and royalties and increased depreciation and amortization expenses and foreign exchange losses.

We expect that our net loss for the year 2011 will increase as compared to 2010, primarily due to higher expected comparative R&D costs and excluding the impacts related to foreign exchange gains or losses, our licensing agreement entered into with Yakult, discussed above, and our conversion to IFRS.

Consolidated Balance Sheet Information

	As at December 31,		
	2010	**2009**	**2008**
	$	**$**	**$**
		(in thousands)	
Cash and cash equivalents	**31,998**	38,100	49,226
Short-term investments	**1,934**		493
Accounts receivable and other current assets	**10,243**	10,913	12,005
Restricted cash	**827**	878	
Property, plant and equipment, net	**3,096**	4,358	6,682
Other long-term assets	**28,476**	32,013	39,936
Total assets	**76,574**	86,262	108,342
Accounts payable and other current liabilities	**13,427**	19,211	22,121
Current portion of long-term payable	**60**	57	49
Long-term payable	**90**	143	172
Non-financial long-term liabilities*	**50,558**	57,625	64,525
Total liabilities	**64,135**	77,036	86,867
Shareholders' equity	**12,439**	9,226	21,475
Total liabilities and shareholders' equity	**76,574**	86,262	108,342

* Comprised mainly of deferred revenues and employee future benefits.

2010 compared to 2009

The decrease in cash and cash equivalents as at December 31, 2010, as compared to December 31, 2009, is due to recurring disbursements and other variations in components of our working capital, as well as to the comparative weakening in 2010 of the euro against the US dollar. However, this decrease is significantly offset by the receipt of approximately $25.0 million of net proceeds in connection with two registered direct offerings, completed in April and June 2010.

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Other long-term assets, comprised of intangible assets, goodwill and deferred charges, decreased in 2010 due to euro-to-US dollar exchange rate fluctuation impacts and due to recurring amortization charges on those assets (excluding goodwill).

The decrease in accounts payable and other current liabilities results to a large degree from settlements of trade payables, income tax liability and other accrued expenses, including balances representing certain residual obligations related to former cetrorelix activities.

Non-financial long-term liabilities decreased from 2009 to 2010 due to recurring amortization of deferred revenues as well as due to euro-to-US dollar exchange rate fluctuation impacts.

The increase in shareholders' equity from December 31, 2009 to December 31, 2010 is attributable to an increase in share capital and warrants, following the completion of the registered direct offerings discussed above, partly offset by the increase in our deficit due to the net loss for the year ended December 31, 2010.

2009 compared to 2008

The decrease in cash and cash equivalents as at December 31, 2009, compared to December 31, 2008 is due primarily to recurring cash flows used in operating activities and by the reduction of currently available cash due to a transfer of funds to a restricted account, as discussed below, largely offset by the receipt of proceeds from sanofi and to the receipt of net proceeds of approximately $14.3 million in connection with two registered direct offerings.

The decrease in property, plant and equipment as at December 31, 2009, compared to December 31, 2008 is due largely to the impairment charge that was taken against certain items utilized exclusively in the development activities related to cetrorelix, as discussed above.

The decrease in other long-term assets primarily includes the reduction to intangible assets, which in turn was attributable to the impairment charges taken on cetrorelix and ozarelix, as discussed above. Additionally, the reduction is attributable to deferred charges amounting to approximately $0.7 million, which were deferred in 2008, but which were included as a reduction to share capital in connection with the June 2009 Offering, as discussed above.

The reduction in non-financial long-term liabilities is attributable mainly to deferred revenues, which in 2009 were lower following both the ongoing amortization of the proceeds received from Cowen and the full recognition of previously deferred amounts associated with license and development agreements related to the use of ozarelix, as discussed above.

The decrease in shareholders' equity from 2008 to 2009 is attributable to the increase in consolidated deficit due to the 2009 net loss and to the decrease in accumulated other comprehensive income, offset in large proportion by the increase in share capital and warrants following the two registered direct offerings discussed above.

Financial Liabilities, Obligations and Commitments

We have certain contractual obligations and commercial commitments. Commercial commitments mainly include R&D services and manufacturing agreements related to the production of Cetrotide®

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and to other R&D programs. The following table summarizes future cash requirements with respect to these obligations.

		Payments due by period			
	Carrying amount	Less than 1 year	1 to 3 years	4 to 5 years	After 5 years
	$	$	$	$	$
		(in thousands)			
Operating leases	10,397	2,150	3,809	3,642	796
Commercial commitments	17,887	9,167	8,720		
Long-term payable	150	60	90		
	28,434	11,377	12,619	3,642	796

Per our agreement with Cowen, discussed above, we may be required to remit quarterly make-whole payments related to royalty rate reductions that could materialize between Cowen and Merck Serono. No make-whole payments were paid or became payable during 2010 or 2009, nor do we expect to be required to remit any such make-whole payments in the future.

Also per our agreement with Cowen, we have agreed to make a one-time cash payment to Cowen in the event that cetrorelix is approved for sale by European regulatory authorities in an indication other than *in vitro* fertilization. Such a payment could range from $5.0 million to a maximum of $15.0 million. No one-time cash amount was paid or became payable during 2010 or 2009, nor do we expect to be required to make such a payment in the future, particularly given the fact that our development activities related to cetrorelix have been terminated.

Outstanding Share Data

As at March 24, 2011, there were 85,265,033 common shares issued and outstanding and 6,807,463 stock options outstanding. Warrants outstanding as at March 24, 2011 represent a total of 12,795,885 equivalent common shares.

Capital disclosures

Our objective in managing capital is to ensure sufficient liquidity to fund our R&D activities, SG&A expenses, working capital and capital expenditures.

We endeavour to manage our liquidity to minimize dilution to our shareholders. Non-dilutive activities have included the sale of non-core assets and rights to future royalties, the collection of investment tax credits and grants, interest income, licensing fees, service and royalties. More recently, however, we have raised additional capital through registered direct offerings, as discussed above.

Our capital management objective remains the same as that of previous years. The policy on dividends is to retain cash to keep funds available to finance the activities required to advance our product development pipeline.

We are not subject to any capital requirements imposed by any regulators or any other external source.

It is important to note that historical expenditure patterns cannot be taken as an indication of future expenditures. The amount and timing of expenditures and availability of capital resources vary substantially from period to period, depending on the level of research and development activity being undertaken at any one time and on the availability of funding from investors and prospective commercial partners.

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Liquidity, Cash Flows and Capital Resources

We endeavour to finance our operations and capital expenditures mainly through cash flows from product sales, license fee revenues and other non-dilutive activities. However, we have also completed registered direct offerings during the year ended December 31, 2009 and more recently, in April and June 2010, as noted above.

Our cash, cash equivalents and short-term investment amounted to $33.9 million as at December 31, 2010, compared to $38.1 million as at December 31, 2009. Possible additional operating losses and/or possible investments in complementary businesses or products may require additional financing. As at December 31, 2010, cash and cash equivalents of the Company included CAN$0.5 million and €1.4 million.

Based on our assessment, which took into account current cash levels, the completion of the registered direct offerings discussed above, as well as our strategic plan and corresponding budgets for 2011 and projections for 2012 and 2013, we believe that we have sufficient financial resources to fund planned expenditures and other working capital needs for at least, but not limited to, the 12-month period from the balance sheet date of December 31, 2010.

We may endeavour to secure additional financing, as required, through strategic alliance arrangements or through other non-dilutive activities, as well as via the issuance of new share capital pursuant to, for example, the ATM sales agreement referred to above.

The variations in our liquidity by activity are explained below.

Operating Activities

Cash flows used in operating activities amounted to $31.1 million for the year ended December 31, 2010, compared to $24.1 million and $1.3 million for each of the years ended December 31, 2009 and 2008, respectively. The net increase in cash used in operating activities from 2009 to 2010 is attributable to the fact that our 2009 operating cash flows had been positively impacted by the cash proceeds received from sanofi as an upfront payment on our former cetrorelix-related licensing and development agreement. Similar cash payments were not received during 2010. Additionally, settlements of our trade accounts receivable were lower in 2010, as compared to 2009. However, the decrease in cash provided by operating activities in 2010 was partially offset by a significant reduction in cash R&D and other prepaid expenditures.

The significant increase in cash used in operating activities from 2008 to 2009 is attributable to the receipt of net cash proceeds of $51.5 million in 2008 from Cowen, compared to the lower cash proceeds of $30.0 million from sanofi in 2009, as discussed above. Also, operating cash payments for prepaid expenses and accounts payable were higher during 2009 as compared to 2008.

We expect net cash used in operating activities to decrease during 2011, as compared to 2010.

Financing Activities

Net cash provided by (used in) financing activities amounted to $25.4 million for the year ended December 31, 2010, compared to $14.2 million and ($1.2 million) for each of the years ended December 31, 2009 and 2008, respectively. The increases in net cash provided by financing activities from 2008 to 2009 and from 2009 to 2010 are attributable almost entirely to the registered direct offerings discussed above.

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Investing Activities

Cash (used in) provided by investing activities amounted to ($0.1 million) for the year ended December 31, 2010, compared to ($1.1 million) and $42.0 million for each of the years ended December 31, 2009 and 2008, respectively.

The significant decrease from 2008 to 2009 relates in large proportion to the sale and maturity of short-term investments as well as to the disposals of the building and land in Quebec City and of Impavido®, in 2008. Also, as discussed above, during 2009, we transferred approximately $0.9 million to a restricted cash account. Changes to restricted cash balances, including any interest earned thereon, are reported in the statement of cash flows as investing activities.

Critical Accounting Policies and Estimates

Our consolidated financial statements are prepared in accordance with Canadian GAAP. A summary of significant and pertinent measurement and disclosure differences between Canadian and US GAAP is provided in note 24 to our 2010 consolidated financial statements. The preparation of financial statements in accordance with generally accepted accounting principles requires management to make estimates and assumptions that affect the reported amounts of assets and liabilities and the disclosures of contingent assets and liabilities at the date of the financial statements, as well as the reported amounts of revenues and expenses during the reporting years. Significant estimates are generally made in connection with the calculation of revenues, inventory and research and development expenses, as well as in determining the allowance for doubtful accounts, valuation allowance for future income tax assets, the useful lives of property, plant and equipment and intangible assets with finite lives, the valuation of intangible assets and goodwill, the fair value of stock options and warrants granted, employee future benefits and certain accrued liabilities. We base our estimates on historical experience, where relevant, and on various other assumptions that we believe to be reasonable under the circumstances. Actual results could differ from those estimates.

The following section summarizes our critical accounting policies and other policies that require the most significant judgment and estimates in the preparation of our consolidated financial statements.

Revenue Recognition and Deferred Revenues

We are currently in a phase in which potential products are being further developed or marketed jointly with strategic partners. Existing licensing agreements usually foresee one-time payments (upfront payments), payments for research and development services in the form of cost reimbursements, milestone payments and royalty receipts for licensing and marketing product candidates. Revenues associated with those multiple-element arrangements are allocated to the various elements based on their relative fair value.

Agreements containing multiple elements are divided into separate units of accounting if certain criteria are met, including whether the delivered element has stand-alone value to the customer and whether there is objective and reliable evidence of the fair value of the undelivered obligation(s). The consideration received is allocated among the separate units based on each unit's fair value and the applicable revenue recognition criteria are applied to each of the separate units.

License fees representing non-refundable payments received upon the execution of license agreements are recognized as revenue upon execution of the license agreements when we have no significant future performance obligations and when collectibility of the fees is assured. Upfront payments received at the beginning of licensing agreements are not recorded as revenue when received but are amortized based on the progress to the related research and development work. This progress is based on estimates of total expected time or duration to complete the work, which is compared to

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the period of time incurred to date in order to arrive at an estimate of the percentage of revenue earned to date.

Milestone payments, which are generally based on developmental or regulatory events, are recognized as revenue when the milestones are achieved, collectibility is assured, and when there are no significant future performance obligations in connection with the milestones.

Royalty revenue, based on a percentage of sales of certain declared products sold by third parties, is recorded when we have fulfilled the terms in accordance with the contractual agreement and have no future obligations, the amount of the royalty fee is determinable and collection is reasonably assured.

Proceeds received in connection with the sale of rights to future royalties are deferred and recognized over the life of the license agreement pursuant to the "units-of-revenue" method, as discussed above.

Revenues from sales of products are recognized when title passes to customers, which is at the time goods are shipped, when there are no future performance obligations, when the purchase price is fixed and determinable, and when collection is reasonably assured.

Impairment of Long-Lived Assets and Goodwill

Property, plant and equipment and intangible assets with finite lives are reviewed for impairment when events or circumstances indicate that carrying values may not be recoverable. Impairment exists when the carrying value of the asset is greater than the undiscounted future cash flows expected to be provided by the asset. The amount of impairment loss, if any, is the excess of its carrying value over its fair value, which in turn is determined based upon discounted cash flows or appraised values, depending of the nature of assets.

Goodwill, which represents the excess of the purchase price over the fair values of the net assets of entities acquired at the respective dates of acquisition, is tested for impairment annually, or more frequently if events or changes in circumstances indicate that the carrying value of the reporting unit to which the goodwill is assigned may exceed the fair value of the reporting unit.

In the event that the carrying amount of a reporting unit, including goodwill, exceeds its fair value, an impairment loss is recognized in an amount equal to the excess. Fair value of goodwill is estimated in the same way as goodwill is determined at the date of the acquisition in a business combination, that is, the excess of the fair value of the reporting unit over the fair value of the identifiable net assets of the reporting unit.

Income Taxes

We operate in multiple jurisdictions, and our earnings are taxed pursuant to the tax laws of these jurisdictions. Our effective tax rate may be affected by changes in, or interpretations of, tax laws in any given jurisdiction, utilization of net operating losses and tax credit carry-forwards, changes in geographical mix of income and expense, and changes in management's assessment of matters, such as the ability to realize future tax assets. As a result of these considerations, we must estimate our income taxes in each of the jurisdictions in which we operate. This process involves estimating our actual current tax exposure, together with assessing temporary differences resulting from differing treatment of items for tax and accounting purposes. These differences result in future tax assets and liabilities, which are included in our consolidated balance sheet. We must then assess the likelihood that our future tax assets will be recovered from future taxable income and establish a valuation allowance if, based on available information, it is more likely than not that some or all of the future income tax assets will not be realized.

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Significant management judgment is required in determining our provision for income taxes, our income tax assets and liabilities, and any valuation allowance recorded against our net income tax assets. The valuation allowance is based on our estimates of taxable income by jurisdiction in which we operate and the period over which our income tax assets will be recoverable. In the event that actual results differ from these estimates or we adjust these estimates in future periods, we may need to amend our valuation allowance, which could materially impact our financial position and results of operations.

Stock-Based Compensation Costs

We account for all forms of employee stock-based compensation using the fair value-based method. This method requires that we make estimates about the risk-free interest rate, the expected volatility of our shares and the expected life of the awards.

International Financial Reporting Standards

We are currently finalizing our evaluation of the impacts that likely will result from preparing our consolidated financial statements in accordance with IFRS. The adoption of IFRS will have an impact on our consolidated financial statements, as well as on certain operational and performance measures, beginning on January 1, 2011 and on a retrospective comparative basis beginning on January 1, 2010.

As previously disclosed, we have developed a formal plan for IFRS conversion and the related transition from current standards. To date, we have completed a full diagnostic, in which all existing international standards were examined in comparison with corresponding Canadian guidance, and significant differences between IFRS and Canadian GAAP were documented in order to plan for more detailed analysis, which has been the focus of our conversion project's solutions development phase and which is substantially complete.

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The key elements of our conversion plan and related status are provided below.

Key element	Solutions development activities	Status
Accounting policies	Detailed review of IFRS guidance expected to be in effect on December 31, 2011 in order to perform quantitative and qualitative analyses of relevant differences between those standards and current Canadian GAAP.	This review is substantially complete.
	Preparation of technical memoranda in order to analyze and substantiate any changes in accounting policy that will be required as at the date of transition to IFRS.	All technical memoranda have been prepared and are in the process of being reviewed and finalized. Certain changes in accounting policies have been identified and confirmed (see some of our preliminary conclusions below).
	Consideration of optional and mandatory exemptions available under IFRS 1.	See below for an update regarding IFRS 1.
Information technology ("IT") and information-gathering	Identification of any changes to systems or processes that will be required in order to comply with the provisions of IFRS, both during the year of transition and upon first-time adoption.	The impact on our systems and information-gathering processes continues to be evaluated in connection with the identification of necessary policy changes. We do not anticipate any significant changes to any of our current IT systems.
Internal control over financial reporting	Review and revision, where necessary, of any internal controls or related activities (including disclosure controls and procedures) following the identification of significant changes that are expected to result upon conversion to IFRS and during the year of transition.	We are in the process of reviewing current internal control documentation to ensure that key controls and activities are appropriate vis-à-vis any policy changes identified. We are also designing and implementing controls related to our transition-year comparative quarterly and annual consolidated financial statements. However, we do not expect that changes to our current internal control activities will be significant.
Training	Provision of support and training to ensure that appropriate personnel have adequate knowledge of IFRS.	We have been providing training where necessary and have been transitioning relevant IFRS knowledge and policy decisions to appropriate employees. Additionally, we have provided updates, on a quarterly basis, to our Audit Committee and executive management team.

Our solutions development activities completed to date have allowed us to conclude that the adoption of certain international standards will result in a significant change to current accounting policies, reported financial statement amounts or disclosures. With the exception of IFRS 1, selected

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areas of international guidance examined to date that are relevant to our business, and the corresponding expected impact that likely will result from the application thereof, are presented below.

Accounting topic	Accounting difference and expected impact
Impairment of assets discounting of estimated future cash flows	International Accounting Standard ("IAS") 36, *Impairment of Assets* ("IAS 36"), introduces the "value in use" concept, which, when used to determine a given asset's recoverable amount, requires the use of future cash flows that are discounted using a pre-tax rate that reflects an assessment of risks specific to the asset subject to impairment testing. Under Canadian GAAP, estimates of future cash flows used in assessing whether an impairment loss exists are not discounted. As a result, where there is an indication that an asset may be impaired, impairment losses are more likely to be recognized under IFRS. We expect that the application of IAS 36 will result in a reduction of the carrying value of our intangible asset, Cetrotide®, of approximately $12.9 million, with a corresponding increase in our accumulated deficit, in our transition-date balance sheet as at January 1, 2010. Consequently, future amortization expense will be lower due to the reduction of the carrying value of Cetrotide®.
Capitalization of transaction costs sale of future royalties	We incurred approximately $4.8 million in transaction costs in connection with the sale of future royalty transaction entered into with Cowen in December 2008. Under Canadian GAAP, we have deferred and are amortizing these costs as royalty sale transaction expenses based on the "units-of-revenue" method in the same manner and over the same period in which the related deferred revenues are recognized as royalty revenues. We have determined that, in the absence of specific international guidance, the aforementioned transaction costs would not have been capitalized under IFRS. As such, we expect to derecognize the remaining unamortized portion of the related asset (short-term and long-term portions), or $4.7 million, with a corresponding increase in our accumulated deficit, in our transition-date balance sheet as at January 1, 2010.
Financial instruments contingent settlement provisions	IAS 32, *Financial Instruments: Presentation* ("IAS 32"), provides more precise guidance than Canadian GAAP with respect to the classification of financial instruments, including share purchase warrants, with contingent settlement provisions. Under Canadian GAAP, we have classified all outstanding share purchase warrants as shareholders' equity, where the instruments are reported at their grant-date fair value. Under the provisions of IAS 32, these warrants would be classified as liabilities and marked to market at each reported balance sheet date, and any changes to fair value would be recognized in the consolidated statement of operations. This treatment is similar to current US GAAP requirements, which are discussed in note 25 to our 2010 consolidated financial statements. We expect that the application of IAS 32 to our outstanding share purchase warrants will increase our liabilities by approximately $1.7 million, with a corresponding reduction of our shareholders' equity, in our transition-date balance sheet as at January 1, 2010. This expected adjustment includes both the reclassification of our warrants and the cumulative, retroactive impact that fair value accounting would have had on our consolidated financial statements at that date. Additionally, the change in accounting treatment would have increased our consolidated net loss for the year ended December 31, 2010 by approximately $6.4 million, being the net change in the total warrant liability that would have been recorded under IFRS.
Employee benefits actuarial gains and losses	IAS 19, *Employee Benefits* ("IAS 19"), allows actuarial gains and losses to be recognized either in other comprehensive income or in profit or loss, so long as the treatment is consistent over all plans and over time. Under Canadian GAAP, we have recognized such gains or losses through profit or loss. However, management has decided that actuarial gains and losses will be recognized in other comprehensive income on a prospective basis (see also below), and, as such, there will be no impact on our transition-date balance sheet.
Presentation of depreciation and amortization expenses	Under IFRS, we will present our consolidated statements of operations in conformity with the "function of expense" classification, as prescribed by IAS 1, *Presentation of Financial Statements*. This classification option requires that all expenses, including depreciation and amortization charges, be allocated to the functional items that are most relevant to our business. Under Canadian GAAP, we report depreciation and amortization expenses separately, without allocation to functional line items. Beginning on the date of transition, however, depreciation and amortization expenses will be allocated to components of research and development costs and under selling, general and administrative expenses, as applicable.

It should be noted that the differences outlined above are not a complete list of topics that are relevant to our business. As such, as we finalize our solutions development activities, we may identify

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other areas that could result in significant quantitative or qualitative impacts upon IFRS adoption or thereafter in comparison to currently applied Canadian GAAP. Additionally, the expected adjustments provided above are unaudited.

As we continue to analyze any potential quantitative adjustments and policy decisions that need to be made upon full conversion to IFRS, we have reached some key preliminary conclusions related to the application of IFRS 1.

IFRS 1 provides authoritative guidance for use in the conversion of a set of financial statements (and interim financial reports for part of that period) from another basis of accounting to IFRS. The basic concept of IFRS 1 is that the adoption of IFRS should be applied retrospectively, meaning that an entity should present its first financial statements using IFRS as if IFRS had been applied and effective from the date of the entity's inception. However, due to the fact that full retrospective application is unlikely to be achievable in a cost-effective manner, IFRS 1 offers certain optional exemptions to first-time preparers of IFRS financial statements. Any, all or none of these exemptions may be taken.

Presented below are our preliminary conclusions with respect to some key IFRS 1 optional exemptions, as applicable to our business.

Accounting topic	**IFRS 1 exemption explained**	**Preliminary conclusion**
Business combinations	IFRS 1 allows first-time preparers to elect not to restate business combinations that have occurred prior to the date of transition (January 1, 2010) in accordance with IFRS 3, *Business Combinations* ("IFRS 3").	We will elect to apply this exemption and apply IFRS 3 only to any business combinations that may occur after the date of transition, without restating any prior business combinations.
Valuation of property, plant and equipment	IFRS 1 permits first-time preparers to measure selected assets at fair value and use that fair value as deemed cost of those assets in the transition date balance sheet.	We will not utilize this optional exemption and continue to use the cost model for property, plant and equipment as at the date of transition to IFRS.
Foreign currency translation adjustments	IFRS 1 permits first-time preparers to eliminate the cumulative translation adjustment ("CTA") balance (a component of accumulated other comprehensive income) at the date of transition.	We will eliminate our date of transition CTA balance by adjusting our opening accumulated deficit.
Employee benefits actuarial gains and losses	IFRS 1 permits first-time preparers to avoid applying retrospectively the change in accounting policy related to actuarial gains and losses pursuant to IAS 19 (discussed above) as at the date of transition to IFRS.	No opening balance sheet adjustment will be made to other comprehensive income related to actuarial gains and losses. Instead, those gains and losses will be recognized in other comprehensive income prospectively, beginning on January 1, 2010.

Outlook for 2011

Perifosine

We expect to continue the development of perifosine in collaboration with our partner, Keryx, who is responsible, in accordance with the terms of our license agreement, for the development and registration of perifosine in North America. We have access to all corresponding data at no additional cost; hence, we expect to benefit from current development activities in order to achieve registration in territories excluding North America.

Our primary focus will be on the advancement of the ongoing Phase 3 registration studies in both refractory advanced colorectal cancer and multiple myeloma, in conformity with the SPA received by Keryx from the FDA. Furthermore, we have obtained positive scientific advice from the EMA relative to a development and regulatory pathway so as to extend the reach of perifosine to European territories for the refractory advanced colorectal cancer and multiple myeloma indications. Consequently, we are not expecting to invest in any additional trials in Europe in refractory advanced

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colorectal cancer or multiple myeloma, since the EMA does not require that any studies be performed in addition to the studies currently in progress. For the ongoing Phase 3 study in multiple myeloma, we will contribute to the recruitment of patients outside the US and to other aspects of the ongoing study; however, our partner Keryx will reimburse us for most of the corresponding costs.

Additionally, we will advance the preparation of our regulatory filings and our commercialization strategy ex-North America. Further, we will continue to accumulate Phase 1 and 2 results in multiple indications and we expect to initiate, with the collaboration of Keryx, an additional clinical trial in CLL.

AEZS-108

We expect to define our regulatory strategy for endometrial cancer with both the FDA and the EMA, with the goal of initiating a pivotal study in that indication.

Additional proof-of-concept and investigator-driven Phase 1/2 studies, such as the ongoing study in refractory bladder cancer performed with the University of Miami and in castration refractory prostate cancer with University of Southern California, will continue to progress.

We also expect to initiate further proof-of-concept studies in patients with LHRH receptor-positive cancers such as pancreatic and triple negative breast cancer.

AEZS-130 (Solorel®)

We expect to complete the ongoing Phase 3 study and file an NDA for Solorel® as a diagnostic for AGHD in the United States.

We also expect to start a proof-of-concept study in cancer-induced cachexia.

Revenue expectations

Revenues are expected to decrease slightly in 2011, as compared to 2010, excluding any revenue associated with the licensing agreement entered into with Yakult, discussed above.

Cost reduction and development focus

During 2011, we expect to focus our R&D efforts on our later-stage compounds, including perifosine, AEZS-108 and Solorel®. Earlier-stage projects will be associated with grants, R&D credits or collaboration agreements. With our focused strategy, we can expect an increase of our R&D expenses to between $21.0 million and $23.0 million for the whole of 2011, as compared to $20.5 million in 2010. However, certain R&D expenses will be reimbursed by our partner, Keryx, as mentioned above.

With regard to our SG&A expenses, in light of the continuous cost-saving measures already in place, we now expect to slightly reduce our SG&A costs for the whole of 2011, as compared to 2010.

We expect that our overall operating burn in 2011 will decrease, as compared to 2010, due most notably to the receipt of €6.0 million (approximately $8.3 million) in connection with our licensing agreement entered into with Yakult.

Financial and Other Instruments

Foreign Currency Risk

Since we operate internationally, we are exposed to currency risks as a result of potential exchange rate fluctuations related to non-intragroup transactions. In particular, fluctuations in the US dollar and

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Canadian dollar exchange rates against the euro could have a potentially significant impact on our results of operations.

For the year ended December 31, 2010, we were not a party to any forward-exchange contracts, and no forward-exchange contracts were outstanding as at March 22, 2011.

Credit Risk

Financial instruments that potentially subject the Company to concentrations of credit risk consist primarily of cash and cash equivalents, restricted cash and accounts receivable. Cash and cash equivalents and restricted cash balances are maintained with high-credit quality financial institutions. Also, no accounts receivable balance due to the Company that is past due as at December 31, 2010 is significant. Consequently, management considers the risk of non-performance related to cash and cash equivalents, restricted cash and accounts receivable to be minimal.

Generally, we do not require collateral or other security from customers for trade accounts receivable; however, credit is extended following an evaluation of creditworthiness. In addition, we perform ongoing credit reviews of all our customers and establish an allowance for doubtful accounts when accounts are determined to be uncollectible.

Related Party Transactions and Off-Balance Sheet Arrangements

We did not enter into transactions with any related parties during the year ended December 31, 2010.

As at December 31, 2010, we did not have any interests in variable interest entities or any other off-balance sheet arrangements.

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Item 6. Directors, Senior Management and Employees

A. Directors and senior management

The following table sets forth information about our directors and corporate officers as at March 24, 2011.

Name and Place of Residence	Position with Aeterna Zentaris
Aubut, Marcel Quebec, Canada	Director
Blake, Paul Pennsylvania, United States	Senior Vice President and Chief Medical Officer
Dorais, José P. Quebec, Canada	Director
Engel Juergen Alzenau, Germany	President and Chief Executive Officer and Director
Ernst, Juergen Brussels, Belgium	Chairman of the Board and Director
Lapalme, Pierre Quebec, Canada	Director
Limoges, Gérard Quebec, Canada	Director
MacDonald, Pierre Quebec, Canada	Director
Métivier, Amélie Quebec, Canada	Assistant Secretary
Meyers, Michael New York, United States	Director
Pelliccione, Nicholas New York, United States	Senior Vice President, Regulatory Affairs and Quality Assurance
Seeber, Matthias Frankfurt, Germany	Senior Vice President, Administration and Legal Affairs
Shapiro, Elliot Quebec, Canada	Corporate Secretary
Turpin, Dennis Quebec, Canada	Senior Vice President and Chief Financial Officer

There are no family relationships among any of the directors or executive officers of the Company and its subsidiaries. The following is a brief biography of each of our directors and senior officers.

Marcel Aubut has served as a director on our Board since 1996. Mr. Aubut is a managing partner of Heenan Blaikie Aubut LLP, a law firm. The countless companies and boards with which Marcel Aubut has been involved with over the years demonstrate his versatility and, above all, his vast experience in the world of business. These include, among others, Atomic Energy of Canada, Olymel L.P. (Olybro), Boralex Power Income Fund, Triton Electronik, Whole Foods Market Canada, Hydro-Québec (Executive Committee), Purolator Courier Ltd., Tremblant Resort, Cinar Inc., La Laurentienne générale, La Laurentienne vie, Investors Group Inc., Transforce Inc., Intra Continental Insurers Ltd., the National Hockey League Pension Society, Boréal Assurances Agricoles Inc.,

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Entreprises Premier CDN Ltée, Les Industries Amisco Ltée, Donohue Matane Inc., La Société de développement du Loisir et du Sport du Québec, the Canadian Olympic Committee, the Canadian Olympic Foundation, member of VANOC's Audit Committee, Governance and Ethics Committee and Observer Team, Sodic Québec Inc., Innovatech Québec, Textile Dionne, Canada's Sports Hall of Fame, the Committee for the 2002 Quebec City Olympic Games Bid, the Committee for the 2015 Toronto Pan American Games Bid, la Fondation Nordiques, etc. He has also presided over the establishment of numerous industrial projects in the greater region of Quebec City.

Paul Blake was appointed our Senior Vice President and Chief Medical Officer in August 2007. Prior to joining us, Dr. Blake was Chief Medical Officer of Avigenics, Inc. since January 2007. In 2005, he was Senior Vice President, Clinical Research and Regulatory Affairs at Cephalon, Inc. before being promoted to Executive Vice President, Worldwide Medical & Regulatory Operations. From 1992 to 1998, he held the position of Senior Vice President and Medical Director, Clinical Research and Development at SmithKline Beecham Pharmaceuticals (now GSK). Dr. Blake earned a medical degree from the London University, Royal Free Hospital. He was elected Fellow of the American College of Clinical Pharmacology, Fellow of the Faculty of Pharmaceutical Medicine, Royal College of Physicians in the UK, and he is a Fellow of the Royal College of Physicians in the UK. Dr. Blake is also a Director of Oxford BioMedica (non-executive) and member of its remuneration committee.

José P. Dorais has served as a director on our Board since 2006. Mr. Dorais is a partner of Miller Thomson Pouliot LLP where he mainly practices administrative, corporate, business and international trade law. Over his 35-year career, he has worked in both the private and public sectors; in the latter he acted as Secretary to the Minister of Justice and as Secretary of the consulting committee on the Free Trade Agreement for the Quebec Provincial Government. Mr. Dorais has been a member of numerous boards of directors, including the Société des Alcools du Québec, Biochem Pharma and St-Luc Hospital in Montreal. He is now a member of the Board of Alliance Films, the Société Générale de Financement and Chairman of the Board of Recyc-Québec. He holds a law degree from the University of Ottawa and is a member of the Quebec Bar.

Juergen Engel was appointed President and Chief Executive Officer, effective September 1, 2008, after having up to such time served as our Executive Vice President and Chief Scientific Officer. He became a director on our Board in 2003. Dr. Engel has been Managing Director of AEZS Germany, the Company's principal subsidiary, since the beginning of 2001. Before that, he was in charge of all research and development activities of ASTA Medica AG. He is member of the Advisory Board of GIG, Berlin and ElexoPharm, Saarbrücken. He served as a member of the Board of Directors of Isotechnika Pharma Inc until February 2011.

Juergen Ernst was appointed Chairman of the Board, effective August 13, 2007, after having been Interim President and Chief Executive Officer from April 11, 2008 until August 31, 2008. He has served as a director on our Board since 2005. A seasoned executive with more than 20 years of pharmaceutical industry expertise mainly in the field of corporate development and pharmaceutical product marketing, Mr. Ernst was worldwide General Manager, Pharmaceutical Sector of Solvay S.A., before retiring in 2004. He has served as a director of Pharming Group N.V., Leiden, Netherlands since April 15, 2009.

Pierre Lapalme has served as a director on our Board since December 2009. Mr. Lapalme has over the course of his career held numerous senior management positions in various global life sciences companies. He is former Senior Vice-President, Sales and Marketing for Ciba-Geigy (which subsequently became Novartis) and former Chief Executive Officer and Chairman of the Board of Rhone-Poulenc Pharmaceuticals Inc. in Canada and in North America, as well as Executive Vice-President and Chief Executive Officer of Rhone-Poulenc-Rorer Inc. North America (now sanofi-aventis), where he supervised the development, manufacturing and sales of prescription products in North and Central America. Mr. Lapalme served on the Board of the National Pharmaceutical Council USA and was a Board member of the Pharmaceutical Manufacturers Association of Canada, where he

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played a leading role in reinstituting patent protection for pharmaceuticals. Until recently, he was Board member and Chairman of the Board of Sciele Pharma Inc. which was acquired by Shionogi and Co. Ltd. Mr. Lapalme is currently Chairman of the Board of Biomarin Inc., Chairman of the Board of Pediapharm Inc. and Board member of Algorithme Pharma Inc. He studied at the University of Western Ontario and at INSEAD, France.

Gérard Limoges has served as a director on our Board since 2004. Mr. Limoges served as the Deputy Chairman of Ernst & Young LLP Canada until his retirement in September 1999. After a career of 37 years with Ernst & Young, Mr. Limoges has been devoting his time as a director of a number of companies. Mr. Limoges began his career with Ernst & Young in Montreal in 1962. After graduating from the Management Faculty of Université de Montréal (HEC Montréal) in 1966, he wrote the CICA exams the same year (Honors: Governor General's Gold Medal for the highest marks in Canada and Gold Medal of the Ordre des Comptables Agréés du Québec). He became a chartered accountant in 1967 and partner of Ernst & Young in 1971. After practicing as auditor since 1962 and partner since 1971, he was appointed Managing Partner of the Montreal Office in 1979 and Chairman for Quebec in 1984 when he also joined the National Executive Committee. In 1992, he was appointed Vice-chairman of Ernst & Young Canada and the following year, Deputy Chairman of the Canadian firm. After retirement from public practice at the end of September 1999, he was appointed Trustee of the School board of Greater Montreal (1999), member of the Quebec Commission on Health Care and Social Services (2000-2001) and special advisor to the Rector of the University de Montreal and affiliate schools (2000-2003). Mr. Limoges is currently participating, at the request of the Board of the University of Montreal, in the selection of the Dean of the Faculty of Medicine. Mr. Limoges is a board member or trustee and chairman of the audit committees of the following public companies: Aeterna Zentaris Inc., Atrium Innovations Inc. (TSX), Hartco Inc. (TSX), Hart Stores Inc. (TSX) and Homburg Canada Real Estate Investment Trust. He is also a board member of various private companies and charities. Mr. Limoges received the Order of Canada in 2002.

Pierre MacDonald has served as a director on our Board since November 2000. Mr. MacDonald is President and CEO of MacD Consult Inc., a management consulting firm in international finance and marketing, based in Montreal. He served as the Senior Vice President for Eastern Canada for Bank of Montreal, a position which involved the review and evaluation of the financial statements and creditworthiness of borrowers in a wide variety of industries. In December 1995, he was elected to the National Assembly of Quebec and became Minister of International Trade and Technology. He was also named Vice Chairman of the Treasury Board of the Government of Quebec. He also served as the Chairman of the Audit Committee of Teleglobe Inc. for six years. Mr. MacDonald received Bachelor of Arts, Bachelor of Commerce and Master of Commerce degrees from Laval University in Québec.

Amélie Métivier, Assistant Secretary. Ms. Métivier has served as our Assistant Secretary since April 2009. In addition, Ms. Métivier is currently a lawyer at the law firm of Ogilvy Renault LLP with a business law and transaction-oriented practice, where she has worked since 2003. She is a member of the *Barreau du Québec* since 2006, and holds an LL.B. (2004) degree from Université de Montréal.

Michael Meyers, M.P.H. is a co-founding member, Chief Executive Officer and Chief Investment Officer of Arcoda Capital Management LP ("Arcoda"), a private investment fund manager. Prior to founding Arcoda in 2007, Mr. Meyers was a Partner and Portfolio Manager of two other money management firms located in New York. Between 2000 and 2003 Mr. Meyers was a Managing Director, Partner and Director of a life sciences venture capital firm located in New York and Zurich, Switzerland. Between 1997 and 2000, Mr. Meyers was Director, Biotechnology and Pharmaceutical Investment Banking at Merrill Lynch & Co. Between 1993 and 1997, Mr. Meyers was Vice President, Health Care Investment Banking at Cowen & Company. Prior to Cowen & Company, Mr. Meyers was Special Assistant to the Chief Executive Officer of St. Barnabas Hospital System. Mr. Meyers began his career as a Biotechnology and Medical Device Research Associate at Hambrecht & Quist in New York. Mr. Meyers holds an M.P.H. in Health Policy and Management from Columbia University and an A.B.

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in Biology from Brandeis University in Massachusetts. Mr. Meyers has also served on the Board of Directors of six companies at various times.

Nicholas J. Pelliccione was appointed our Senior Vice President, Regulatory Affairs and Quality Assurance in May 2007. In previous roles, Dr. Pelliccione has been responsible for the clinical/preclinical and CMC regulatory aspects of new drugs in the oncology, anti-infectives, cytokines and cardiovascular therapy areas, leading to several approvals. He served as Senior Vice President, Regulatory and Pharmaceutical Sciences at Chugai Pharma USA from May 2005 until March 2007. Prior to his experience at Chugai, Dr. Pelliccione spent more than 15 years at Schering Plough Corporation holding positions with increasing responsibility from Manager of Regulatory Affairs, Oncology to, prior to his departure, Vice President, Global Regulatory Affairs, Chemistry, Manufacturing and Controls. Dr. Pelliccione holds a Ph.D. in Biochemistry from Mount Sinai School of Medicine, New York and a BS in Chemistry from Polytechnic University.

Matthias Seeber was appointed our Senior Vice President, Administration and Legal Affairs in December 2008. Mr. Seeber served as Managing Director of AEZS Germany since July 2003 up to his most recent appointment. Prior to that, he had assumed the position of Investor Relations Manager of Altana AG, following several years in the banking industry with Deka Investment Management and Dresdner Bank AG. Mr. Seeber is a member of the Deutsche Vereinigung für Finanzanalyse und Asset Management (DVFA/CEFA). He obtained his M.B.A. from George Mason University Graduate School of Business Administration in the United States.

Elliot Shapiro was appointed our Corporate Secretary in April 2009. In addition, Mr. Shapiro is currently a partner and a lawyer at the law firm of Ogilvy Renault LLP with a business law and transaction-oriented practice, where he has worked since 1999. He has been a member of the *Barreau du Québec* since 2000. Mr. Shapiro holds B.C.L. (1999), LL.B. (1999) and B.A. (1993) degrees from McGill University.

Dennis Turpin was appointed our Senior Vice President and Chief Financial Officer in August 2007. Prior to that, he served as our Vice President and Chief Financial Officer since June 1999. Mr. Turpin joined Aeterna Zentaris in August 1996 as Director of Finance. Prior to that, he was Director in the tax department at Coopers Lybrand, now PricewaterhouseCoopers, from 1988 to 1996 and worked as an auditor from 1985 to 1988. Mr. Turpin earned his Bachelor's degree in Accounting from Laval University in Québec. He obtained his license in accounting in 1985 and became a chartered accountant in 1987.

B. Compensation

A. Compensation of Outside Directors

The compensation paid to the Company's directors is designed to (i) attract and retain the most qualified people to serve on the Board and its committees, (ii) align the interests of the Company's directors with those of its shareholders, and (iii) provide appropriate compensation for the risks and responsibilities related to being an effective director. This compensation is recommended to the Board by the Corporate Governance, Nominating and Human Resources Committee (the "Governance Committee"). During the most recently completed financial year, the Governance Committee was composed of three (3) directors, each of whom is independent, namely Messrs. Pierre MacDonald, José P. Dorais and Juergen Ernst. One of the members of the Governance Committee, Juergen Ernst, is Chairman of the Board.

The Board has adopted a formal mandate for the Governance Committee, which is available on our website at www.aezsinc.com. The mandate of the Governance Committee provides that it is responsible for (i) assisting the Board in developing our approach to corporate governance issues, (ii) proposing new Board nominees, (iii) assessing the effectiveness of the Board and its committees,

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their respective chairs and individual directors and (iv) making recommendations to the Board with respect to directors' compensation.

In light of prevailing economic and market conditions, as well as the various ongoing cost-saving measures implemented by the Company in the past two years, the Governance Committee recommended and the Board approved reduction to directors' and committee members' retainers and attendance fees, such reductions having taken effect as at January 1, 2010.

We did not retain the services of any external compensation consultant in or with respect to the financial year ended December 31, 2010.

Annual Retainers and Attendance Fees

Annual retainers and attendance fees are paid on a quarterly basis to the members of the Board who are not employees of the Company or its subsidiaries ("Outside Directors") as described in the table below.

Type of Compensation	**Annual compensation for the year 2010 (in units of home country currency)**
Chairman's Retainer	45,000
Vice Chairman's Retainer[(1)]	15,000
Board Retainer	15,000
Board Meeting Attendance Fees	1,000 per meeting
Audit Committee Chair Retainer	15,000
Audit Committee Member Retainer	4,000
Audit Committee Meeting Attendance Fees	1,000
Governance Committee Chair Retainer	12,000
Governance Committee Member Retainer	2,000
Governance Committee Meeting Attendance Fees	1,000

(1) There is currently no Vice Chairman of the Board.

All amounts in the above table are paid to Board and committee members in their home country currency.

The President and Chief Executive Officer is the only member of the Board who is not an Outside Director. Therefore, he is not compensated in his capacity as a director. The Chairman is an Outside Director and is compensated as such. Outside Directors are reimbursed for travel and other out-of-pocket expenses incurred in attending Board or committee meetings.

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Outstanding Option-Based Awards and Share-Based Awards

The following table shows all awards outstanding to each Outside Director up to the end of the financial year ending and as at December 31, 2010:

	Option-based Awards					Share-based Awards		
Name	Issuance Date (mm-dd-yyyy)	Number of Securities Underlying Unexercised Options[1] (#)	Option Exercise Price (CAN$)	Option Expiration Date (mm-dd-yyyy)	Value of Unexercised In-the-money Options[2] (CAN$)	Issuance Date (mm-dd-yyyy)	Number of Shares or Units of Shares that have Not Vested (#)	Market or Payout Value of Share-based Awards that have Not Vested ($)
Aubut, Marcel	12-04-2001	5,000	6.18	12-31-2011				
	12-16-2002	15,000	3.68	12-15-2012				
	12-11-2003	30,000	1.74	12-10-2013				
	12-14-2004	15,000	5.83	12-13-2014				
	12-13-2005	15,000	3.53	12-12-2015				
	01-04-2007	5,000	4.65	01-03-2017				
	12-11-2007	25,000	1.82	12-10-2017				
	12-08-2008	15,000	0.55	12-08-2018	17,550			
	12-09-2009	20,000	0.95	12-08-2019	15,400			
	12-08-2010	30,000	1.52	12-07-2020	6,000			
Dorais, José P.	12-08-2010	30,000	1.52	12-07-2020	6,000			
Ernst, Juergen	02-25-2005	15,000	5.09	02-24-2015				
	12-13-2005	15,000	3.53	12-12-2015				
	01-04-2007	5,000	4.65	01-03-2017				
	12-11-2007	25,000	1.82	12-10-2017				
	11-14-2008	100,000	0.65	11-13-2018	107,000			
	12-08-2008	15,000	0.55	12-08-2018	17,550			
	12-09-2009	20,000	0.95	12-08-2019	15,400			
	12-08-2010	30,000	1.52	12-07-2020	6,000			
Lapalme, Pierre	12-09-2009	20,000	0.95	12-08-2019	15,400			

	12-08-2010	30,000	1.52	12-07-2020	6,000
Limoges, Gérard	12-14-2004	15,000	5.83	12-13-2014	
	12-13-2005	15,000	3.53	12-12-2015	
	01-04-2007	5,000	4.65	01-03-2017	
	12-11-2007	25,000	1.82	12-10-2017	
	12-08-2008	15,000	0.55	12-08-2018	17,550
	12-09-2009	20,000	0.95	12-08-2019	15,400
	12-08-2010	30,000	1.52	12-07-2020	6,000

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	Share-based Awards					Share-based Awards		
Name	Issuance Date (mm-dd-yyyy)	Number of Securities Underlying Unexercised Options[1] (#)	Option Exercise Price (CAN$)	Option Expiration Date (mm-dd-yyyy)	Value of Unexercised In-the-money Options[2] (CAN$)	Issuance Date (mm-dd-yyyy)	Number of Shares or Units of Shares that have Not Vested (#)	Market or Payout Value of Share-based Awards that have Not Vested ($)
MacDonald, Pierre	12-04-2001	5,000	6.18	12-31-2011				
	12-16-2002	24,000	3.68	12-15-2012				
	12-11-2003	30,000	1.74	12-10-2013				
	12-14-2004	15,000	5.83	12-13-2014				
	12-13-2005	15,000	3.53	12-12-2015				
	01-04-2007	5,000	4.65	01-03-2017				
	12-11-2007	25,000	1.82	12-10-2017				
	12-08-2008	15,000	0.55	12-08-2018	17,550			
	12-09-2009	20,000	0.95	12-08-2019	15,400			
	12-08-2010	30,000	1.52	12-07-2020	6,000			

(1) The number of securities underlying unexercised options represent all awards outstanding as at December 31, 2010.

(2) "Value of unexercised in-the-money options" at financial year-end is calculated based on the difference between the closing price of the common shares on the Toronto Stock Exchange (the "TSX") on the last trading day of the fiscal year (December 31, 2010) of CAN$1.72 and the exercise price of the options, multiplied by the number of unexercised options.

See "Summary of the Stock Option Plan" below for more details on the Stock Option Plan (as defined below).

Total Compensation of Outside Directors

The table below summarizes the total compensation earned by the Outside Directors during the financial year ended December 31, 2010 (all amounts are in US dollars):

	Fees earned ($)		Share-based Awards	Option-based Awards[2]	Non-Equity Incentive Plan Compensation	Pension Value	All Other Compensation[3]	Total
Name	Retainer[1]	Attendance[1]	($)	($)	($)	($)	($)	($)
Aubut, Marcel	14,550	3,395		35,793				53,738
Byorum, Martha[4]	6,942	2,000						