Aeterna Zentaris Inc.
Form 20-F
March 28, 2012
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UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 20-F

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

x **Annual Report Pursuant to Section 13 or 15(d) of The Securities Exchange Act of 1934 for the fiscal year ended December 31, 2011**
OR

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

¨ **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: 104,762,096 common shares as at December 31, 2011.

Indicate by check mark whether the registrant is a well-known seasoned issuer, as defined in Rule 405 of the Securities Act. Yes ¨ No x

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 ¨ No x

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

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate Web site, if any, every Interactive Data File required to be submitted and posted pursuant to Rule 405 of Regulation S-T (§232.405 of this chapter) during the preceding 12 months (or for such shorter period that the registrant was required to submit and post such files). Yes ¨ No x

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 ¨	Accelerated filer x	Non-accelerated filer ¨

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

US GAAP ¨	International Financial Reporting Standards as issued by the International Accounting	Other ¨

Standards Board x

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 ¨ Item 18 ¨

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 ¨ No x

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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, references to CAN$ are to Canadian dollars and references to EUR are to euros. Unless otherwise indicated, the statistical and financial data contained in this annual report are presented as at December 31, 2011.

This annual report on Form 20-F also contains certain information regarding products or product candidates that may potentially compete with our products and product candidates, and such information has been primarily derived from information made publicly available by the companies developing such potentially competing products and product candidates and has not been independently verified by Aeterna Zentaris Inc.

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 our research and development (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 disclaim any obligation to update any such factors or to publicly announce the result of any revisions to any of the forwards-looking statements contained herein to reflect future results, events or developments except if 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 comprehensive loss data set forth in this Item 3.A with respect to the years ended December 31, 2011 and 2010, and the consolidated statement of financial position data as at December 31, 2011 and 2010, have been derived from the audited consolidated financial statements listed in Item 18, which have been prepared in accordance with International Financial Reporting Standards (IFRS), as issued by the International Accounting Standards Board (IASB). The consolidated statement of operations data set forth in this Item 3.A with respect to the years ended December 31, 2009, 2008 and 2007, and the consolidated balance sheet data as at December 31, 2009, 2008 and 2007, have been derived from our previous consolidated financial statements not included herein, which were 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 Comprehensive Loss

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

Derived from financial statements prepared in accordance with IFRS

	Years ended December 31,	
	2011	**2010**
	$	**$**
Revenues		
Sales and royalties	**31,306**	24,857
License fees and other	**4,747**	2,846
	36,053	27,703
Operating expenses		
Cost of sales	**27,560**	18,700
Research and development costs, net of refundable tax credits and grants	**24,517**	21,257
Selling, general and administrative expenses	**16,170**	12,552
	68,247	52,509
Loss from operations	**(32,194)**	(24,806)
Finance income	**6,239**	1,800
Finance costs	**(8)**	(5,445)
Net finance income (costs)	**6,231**	(3,645)
Loss before income taxes	**(25,963)**	(28,451)
Income tax expense	**(1,104)**	-
Net loss	**(27,067)**	(28,451)
Other comprehensive (loss) income:		
Foreign currency translation adjustments	**(789)**	1,001
Actuarial gain (loss) on defined benefit plans	**(1,335)**	191
Comprehensive loss	**(29,191)**	(27,259)
Net loss per share		
Basic and diluted	**(0.29)**	(0.38)
Weighted average number of shares outstanding		
Basic and diluted	**94,507,988**	75,659,410

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Consolidated Statements of Operations Data

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

Derived from financial statements prepared in accordance with Canadian GAAP

	Years Ended December 31,		
	2009	2008	2007
	$	$	$
Revenues	63,237	38,478	42,068
Operating expenses			
Cost of sales, excluding depreciation and amortization	16,501	19,278	12,930
Research and development costs	44,217	57,448	39,248
Research and development tax credits and grants	(403)	(343)	(2,060)
Selling, general and administrative expenses	16,040	17,325	20,403
Depreciation and amortization			
Property, plant and equipment	3,285	1,515	1,562
Intangible assets	7,555	5,639	4,004
Impairment of long-lived assets held for sale			735
	87,195	100,862	76,822
Loss from operations	(23,958)	(62,384)	(34,754)
Other income (expenses)			
Interest income	349	868	1,904
Interest expense	(5)	(118)	(85)
Foreign exchange gain (loss)	(1,110)	3,071	(1,035)
Loss on disposal of long-lived assets held for sale		(35)	
Loss on disposal of equipment		(44)	(28)
	(766)	3,742	756
Loss before income taxes from continuing operations	(24,724)	(58,642)	(33,998)
Income tax (expense) recovery		(1,175)	1,961
Net loss from continuing operations	(24,724)	(59,817)	(32,037)
Net loss from discontinued operations			(259)
Net loss for the year	(24,724)	(59,817)	(32,296)
Net loss per share from continuing operations			
Basic and diluted	(0.43)	(1.12)	(0.61)

Net loss per share			
Basic and diluted	(0.43)	(1.12)	(0.61)
Weighted average number of shares			
Basic and diluted	56,864,484	53,187,470	53,182,803

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Consolidated Statements of Operations Data

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

Derived from reconciliation to US GAAP

	Years ended December 31,		
	2009*	2008*	2007*
	$	$	$
Net loss for the year	(16,794)	(56,070)	(37,428)
Of which:			
Net loss from continuing operations	(16,794)	(56,070)	(36,415)
Net loss from discontinued operations			(1,013)
Net loss per share from continuing operations			
Basic and diluted	(0.30)	(1.05)	(0.68)
Net loss per share from discontinued operations			
Basic and diluted			(0.02)
Net loss per share			
Basic and diluted	(0.30)	(1.05)	(0.70)
Weighted average number of shares			
Basic and diluted	56,864,484	53,187,470	53,182,803

Consolidated Statement of Financial Position Data

(in thousands of US dollars)

Derived from financial statements prepared in accordance with IFRS for 2011 and 2010, and Canadian GAAP for 2009, 2008 and 2007

	As at December 31,				
	2011	2010	2009*	2008*	2007*
	$	$	$	$	$
Cash and cash equivalents	46,881	31,998	38,100	49,226	10,272
Short-term investments		1,934		493	31,115
Working capital	42,254	29,444	29,745	39,554	37,325
Restricted cash	806	827	878		
Total assets	75,369	61,448	86,262	108,342	123,363
Warrant liability short-term	42	955	*	*	*
Warrant liability long-term	9,162	13,412	*	*	*
Long-term payable	29	90	143	172	
Share capital	101,884	60,900	41,203	30,566	30,566
Shareholders (deficiency) equity	(4,546)	(17,575)	9,226	21,475	88,591

* We adopted IFRS in 2011 with a transition date of January 1, 2010. The selected financial information for the years ended December 31, 2009, 2008 and 2007 is derived from financial statements that were presented in accordance with Canadian GAAP and has not been restated in accordance with IFRS. Consequently, the selected financial information for the years ended December 31, 2009, 2008 and 2007 may not be comparable with the corresponding selected financial information for the years ended December 31, 2011 and 2010. Please refer to Critical Accounting Policies, Estimates and Judgments for the policy differences between Canadian GAAP and IFRS.

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Consolidated Statement of Financial Position Data

(in thousands of US dollars)

Derived from financial statements prepared in accordance with IFRS for 2011 and 2010, and US GAAP for 2009, 2008 and 2007

	As at December 31,				
	2011	**2010**	**2009***	**2008***	**2007***
	$	**$**	**$**	**$**	**$**
Cash and cash equivalents	46,881	31,998	38,100	49,226	10,272
Short-term investments		1,934		493	31,115
Working capital	42,254	29,444	29,745	39,554	37,325
Restricted cash	806	827	878		
Total assets	75,369	61,448	84,116	100,001	109,182
Warrant liability, short-term	42	955	*	*	*
Warrant liability, long-term	9,162	13,412	1,351	*	*
Long-term payable	29	90	143	172	
Share capital	101,884	60,900	33,226	22,589	22,589
Shareholders (deficiency) equity	(4,546)	(17,575)	5,729	13,134	74,410

* We adopted IFRS in 2011 with a transition date of January 1, 2010. The selected financial information for the years ended December 31, 2009, 2008 and 2007 is derived from financial statements that were presented in accordance with Canadian GAAP and has not been restated in accordance with IFRS. Consequently, the selected financial information for the years ended December 31, 2009, 2008 and 2007 may not be comparable with the corresponding selected financial information for the years ended December 31, 2011 and 2010. Please refer to Critical Accounting Policies, Estimates and Judgments for the policy differences between Canadian GAAP and IFRS.

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, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010, we had an accumulated deficit of $189.0 million as at December 31, 2011. Our operating losses have adversely impacted, and will continue to adversely impact, our working capital, total assets and

shareholders deficiency. 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 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 ultimately do not 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.

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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. For example, our partner Keryx is conducting phase 3 trials of perifosine for the treatment of colorectal cancer and multiple myeloma. The enrollment of the trial in colorectal cancer has been completed and results of this trial are expected in the near future. If the results of this clinical trial are unfavorable, it would likely adversely affect our and our partners perifosine development programs. In addition, unfavorable results in this trial could adversely affect our stock price. 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 preclinical 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 preclinical 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. Preclinical 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 preclinical and clinical tests can be interpreted in different ways, which could delay, limit or prevent regulatory approval. It may take us 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, we may encounter unforeseen problems and delays in the approval process. Though we may engage a contract research organization (a CRO) 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 (the 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, preclinical 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 preclinical 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 preclinical 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 preclinical 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.

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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 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.

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. Such trials 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 North America. 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.

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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 continue to be subject to stringent ongoing government regulation for our products and, even if we obtain regulatory approvals, for our product candidates.

The manufacture, marketing and sale of our products and product candidates are and will continue to be subject to strict and ongoing regulation, even if regulatory authorities approve our product candidates. 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.

We and our contract manufacturers are and will continue to 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.

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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.

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

We will likely 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 would result in dilution to our shareholders. Moreover, the incurrence of indebtedness 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 more than 12 months following year-end. 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.

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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 in their respective territories. 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 either Keryx or Yakult does 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 such 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 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, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010 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, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010 that management believed that the Company had, as at December 31, 2011, 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 s assumptions will not change in our future financial statements.

Since our inception, we have incurred losses, accumulated deficits and negative cash flows from operations. We expect that this will continue throughout 2012.

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 with third parties, the global credit markets may adversely affect the ability of potential third parties to pursue such transactions with us. 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 could be material uncertainties related to certain adverse conditions and events that could cast significant doubt on our ability to remain a going concern.

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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.

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

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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 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 may also 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.

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.

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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 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.

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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.

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.

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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.

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.

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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 into this Annual Report on Form 20-F. See, for example, Note 5 to our audited consolidated financial statements as at December 31, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 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, 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.

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 (IND) 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 preclinical 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 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 to our licensing partners who are responsible for marketing such 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.

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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 preclinical 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 and, as such, we are subject to environmental and occupational safety laws regulating the use of such materials. If we violate these laws, we could incur significant fines or liabilities or suffer 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, 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

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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 consolidated 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 consolidated financial statements, which may be inaccurate if we fail to remedy such material weakness.

It is possible that we may be a 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 Material U.S. Federal Income Tax Considerations 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.

We believe that we were not a PFIC for the 2011 taxable year. However, 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. Thus no assurance can be provided that we will not be classified as a PFIC for the 2012 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 in respect of a warrant.

Under recently enacted U.S. tax legislation and subject to future guidance, if the Company is a PFIC, U.S. Holders will be required to file an annual information return with the Internal Revenue Service (IRS) (on IRS Form 8621, which PFIC shareholders will be required to file with their income tax or information returns) relating to their ownership of our common shares. Pursuant to Notice 2011-55, the IRS has suspended this new

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filing requirement for U.S. Holders that are not otherwise required to file the current version of the IRS Form 8621 until the IRS releases a subsequent revision of IRS Form 8621, modified to reflect the recently enacted U.S. tax legislation. Guidance has not yet been issued regarding the information required to be included on such form. This new filing requirement is in addition to any pre-existing reporting requirements that apply to a U.S. Holder s interest in a PFIC (which the recently enacted tax legislation and IRS Notice 2011-55 do not affect).

For a more detailed discussion of the potential tax impact of us being a PFIC, see Item 10.E Taxation Material U.S. Federal Income Tax Considerations 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.

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 the NASDAQ Global Market (NASDAQ) or the Toronto Stock Exchange (the 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, 2011, the closing price of our common shares ranged from $1.43 to $2.58 on NASDAQ and from C$1.41 to C$2.51 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 volumes 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;

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- 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.

A period of large price decline in the market price of our common shares could increase the risk that securities class action litigation could be initiated against us. 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.

If we are unsuccessful in maintaining NASDAQ s minimum bid price or other requirements in the future and are unable to subsequently regain compliance within the applicable grace period, our common shares will be subject to delisting. 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 of the NASDAQ Capital Market, other than compliance with the minimum closing bid price requirement. If the application to the NASDAQ Capital Market were approved, then we would 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 or whether an application to the NASDAQ Capital Market would 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.

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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 by reference 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 acquired 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, 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. The Company moved this office to a new location in December 2011 at 25 Mountainview Blvd., Suite 203, Basking Ridge, NJ 07920.

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 Basking Ridge, New Jersey in the United States.

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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 and AEZS-108 (zoptarelin doxorubicin) in oncology, AEZS-130 (macimorelin) in endocrinology, as well as on strategic and 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 NASDAQ under the trading symbol AEZS .

The Company s agent for service of process and SEC matters in the United States is its wholly-owned subsidiary, Aeterna Zentaris, Inc., located at 25 Mountainview Blvd., Suite 203, Basking Ridge, NJ 07920.

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 development priorities in oncology are the completion of Phase 3 trials with perifosine in colorectal cancer (CRC) and in multiple myeloma (MM), as well as the further advancement of AEZS-108, for which we have successfully completed a Phase 2 trial in advanced endometrial and advanced ovarian cancer. We are planning for the initiation of a pivotal program with AEZS-108 in endometrial cancer and also a Phase 2 trial in triple-negative breast cancer. AEZS-108 is also in development in other cancer indications, including castration- and taxane-resistant prostate cancer, as well as refractory bladder cancer.

Our pipeline also encompasses other earlier-stage programs in oncology. AEZS-112, an oral anticancer agent which involves three mechanisms of action (tubulin, topoisomerase II and angiogenesis inhibition) has completed a Phase 1 trial in advanced solid tumors and lymphoma. Additionally, several novel targeted potential anti-cancer candidates such as AEZS-120, a live recombinant oral tumor vaccine candidate, as well as our phosphoinositide 3-kinase (PI3K)/Erk inhibitors AEZS-129, AEZS-131, AEZS-132 and their respective follow-up compounds are currently in preclinical development.

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Our lead program in endocrinology, a Phase 3 trial under a Special Protocol Assessment (SPA) obtained from the FDA with AEZS-130 as an oral diagnostic test for adult growth hormone deficiency (AGHD), has been completed with positive results. We are planning to file an NDA for the registration of AEZS-130 in the United States, subject to a successful pre-NDA meeting with the FDA. Furthermore, AEZS-130 is in a Phase 2A trial for the treatment of cancer cachexia.

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 preclinical programs as described below, some of which are conducted through grants from various governmental agencies. Our vision is to become a fully-integrated specialty biopharmaceutical company.

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Our product pipeline

Pipeline table

Status of our drug pipeline as at March 27, 2012

Discovery	Preclinical	Phase 1	Phase 2	Phase 3	Commercial
~120,000 compound library	AEZS-120 Prostate cancer vaccine (oncology)	AEZS-112 (oncology)	Perifosine Multiple cancers	Perifosine Refractory advanced colorectal cancer Multiple myeloma	Cetrotide® (*in vitro* fertilization)
	AEZS-129, 131, 132 and 136; Erk & PI3K inhibitors (oncology)		AEZS-108 Endometrial cancer Triple-negative breast cancer Ovarian cancer Castration-and taxane-resistant - prostate cancer Refractory bladder cancer	AEZS-130 Diagnostic in adult growth hormone deficiency (endocrinology)	
	AEZS-137 (Disorazol Z) (oncology)		Ozarelix Prostate cancer		
	AEZS-125 (LHRH-Disorazol Z) (oncology)		AEZS-130 Therapeutic in cancer cachexia		
Partners			Perifosine: **Keryx**	Perifosine: **Keryx**	Cetrotide®: **Merck Serono**

North America	North America	World except
Handok	**Handok**	Japan
Korea	Korea	**Nippon**
Yakult	**Yakult**	**Kayaku /**
Japan	Japan	**Shionogi**
Hikma	**Hikma**	Japan
Middle East/	Middle East/	
North Africa	North Africa	
Ozarelix:		
Spectrum		
World (ex-Japan for		
oncology indications,		
ex-Korea and ex-other		
Asian countries for		
BPH indication)		
Handok		
Korea and other Asian countries for BPH		
indication		
Nippon Kayaku		
Japan for oncology		
indications		

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Oncology

Our highest priorities in oncology are with perifosine, to complete the two Phase 3 trials in CRC and in MM, and to continue our Phase 2 program in multiple cancers, as well as the further advancement of AEZS-108, for which we successfully completed Phase 2 trials in advanced endometrial and advanced ovarian 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 metastatic colorectal cancer (mCRC), MM and in Phase 2 studies for the treatment of other cancers. We believe perifosine is the most advanced anticancer compound of its class in late-stage development. Perifosine as monotherapy is also being explored in chronic lymphocytic leukemia (CLL). The FDA has granted perifosine orphan-drug designation in MM and in neuroblastoma and Fast Track designations in both MM and refractory advanced CRC. Additionally, an agreement was reached with the FDA to conduct the Phase 3 trials in both of these indications under a SPA. Perifosine has also been granted Orphan Medicinal Product designation from the European Medicine Agency (EMA) in MM, and has received positive Scientific Advice from the EMA for both the MM and advanced CRC 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 Yakult for Japan, to Handok Pharmaceuticals Co. Ltd. (Handok) for Korea and to Hikma Pharmaceuticals PLC (Hikma) for the Middle East and North Africa (MENA) region.

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 successfully completed. AEZS-108 is also in development in other cancer indications, including refractory bladder and castration-and taxane-resistant prostate cancer. A pivotal trial in endometrial cancer and also a Phase 2 trial in triple-negative breast cancer are expected to be initiated in 2012. We have obtained orphan-drug status for AEZS-108 in advanced ovarian cancer from the FDA and from the Committee for Orphan Medicinal Products (COMP) of the EMA. An IND in the United States is in place for the treatment of prostate, bladder and triple-negative breast cancer. We own the worldwide rights to AEZS-108 and also have a collaboration agreement with Ventana Medical Systems, Inc. (Ventana), a member of the Roche Group, to develop a companion diagnostic for the immunohistochemical determination of LHRH-receptor expression, for AEZS-108.

Endocrinology

In endocrinology, aside from Cetrotide®, we completed a Phase 3 trial with AEZS-130, which would be the first oral diagnostic test for AGHD.

AEZS-130

AEZS-130, a ghrelin agonist, is an orally available novel synthetic small molecule that stimulates the secretion of growth hormone. We completed a Phase 3 trial under a SPA obtained from the FDA and after conclusion of a successful pre-NDA meeting with the FDA, we plan to file a NDA in the United States. AEZS-130 has been granted orphan-drug designation by the FDA. In addition to the diagnostic indication, we believe that AEZS-130 has potential application 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 (AIDS). Furthermore, the FDA agreed to allow for the initiation of a physician sponsored IND Phase 2A trial in cancer induced cachexia. The study is currently conducted under a cooperative research and development agreement (CRADA) with the Michael E. DeBakey Veterans Affairs Medical Center which will be funding the study.

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Clinical and Preclinical Programs

Additionally, we are advancing AEZS-112 in Phase 1, an oral anticancer agent which involves three mechanisms of action, (tubulin, topoisomerase II and angiogenesis inhibition), as well as several preclinical programs with targeted potential development candidates. Among the agents that we expect to reach the clinical development stage in the coming years are: AEZS-120 (oral prostate cancer vaccine), AEZS-129, AEZS-131 and AEZS-132 or their respective follow-up compounds (Erk and PI3K inhibitors).

We also continue to perform targeted drug discovery activities from which we are able to derive preclinical 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 or with fundings from governmental organizations.

1.0 ONCOLOGY

1.1 SIGNAL TRANSDUCTION INHIBITORS

1.1.1 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 antitumor capacities. In tumor cells, perifosine has demonstrated interactions with vital signal transduction mechanisms and induction of programmed cell death (apoptosis).

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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 antitumor 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.

Based on findings in various tumor models, the U.S. National Cancer Institute (NCI), 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 CRC and MM, and in Phase 2 studies for the treatment of other cancers, and we believe is the most advanced anticancer compound of its class in late-stage development.

Perifosine as monotherapy is also being explored in CLL. The FDA has granted perifosine orphan-drug designation in MM and in neuroblastoma and Fast Track designations in both refractory advanced CRC and MM. Additionally, an agreement was reached with the FDA to conduct the Phase 3 trials in both of these indications under a SPA. Perifosine has also been granted Orphan Medicinal Product designation from the EMA in MM, and has received positive Scientific Advice from the EMA for both the advanced CRC and MM 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, to Yakult for Japan and Hikma for the MENA region.

On July 12, 2011, the European Patent Office (EPO) granted a patent for the use of alkylphosphocholines, more specifically perifosine, in the preparation of a medicament for the treatment of benign and malignant tumours, prior to and/or during the treatment with approved antitumor antimetabolites such as 5-fluorouracil (5-FU) and capecitabine. Filed on July 29, 2003, the patent (EP #1 545 553) entitled, *Use of Alkyl Phosphocholines in Combination with Anti-Tumours Medicaments*, became effective as of July 13, 2011, following its announcement in the European Patent Bulletin, and will expire on July 28, 2023.

1.1.1.1 Perifosine CRC

Phase 2 trial perifosine + capecitabine

On October 5, 2011, we announced that a manuscript, entitled *Randomized Placebo-Controlled Phase 2 Trials of Perifosine Plus Capecitabine as Second- or Third-Line Therapy in Patients with Metastatic Colorectal Cancer*, had been published in the October 3, 2011 online edition of the *Journal of Clinical Oncology* (JCO).

This randomized, double-blind, placebo-controlled study was conducted at 11 centers across the United States. Patients with 2nd or 3rd line mCRC were randomized to receive capecitabine (Xeloda®), an approved drug for mCRC, at a dose of 825 mg/m^2 BID (total daily dose of 1,650 mg/m^2) on days 1 - 14 every 21 days, plus either perifosine or placebo at 50 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.

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The patients in the study were heavily pretreated, 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: 89% of the patients received FOLFIRI (irinotecan + 5-FU + leucovorin); 74% FOLFOX (oxaliplatin + 5-FU + leucovorin); 66% were previously treated with both FOLFIRI and FOLFOX; 79% received Avastin®; and 50% Erbitux®. Prior treatment with single agent capecitabine was excluded.

The primary endpoints of this study were to measure 1) time to progression (TTP), 2) overall response rate (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 stable disease (SD). Safety of perifosine + capecitabine vs. placebo + capecitabine in this patient population was evaluated as a secondary endpoint.

Best response and median TTP of perifosine + capecitabine vs. placebo + capecitabine were as follows:

Group	N*	CR N (%)	PR N (%)	ORR N (%)	SD > 12 N (%)	CBR N (%)	Median TTP
Perifosine + capecitabine	20	1(5%)	3(15%)	4(20%)	11 weeks (55%)	15(75%)	27.5 weeks (95% CI**, 12.1 to 48.1)
Placebo + capecitabine	15	0	1 (7%)	1(7%)	5 weeks (33%)	6(40%)	10.1 weeks (95% CI, 6.6 to 13.0)

* N = Number of patients

** CI = Confidence interval

Perifosine + capecitabine more than doubled TTP vs. placebo + capecitabine with a statistically significant p-value <.001. In addition, perifosine + capecitabine more than doubled the ORR and almost doubled the CBR vs. placebo + capecitabine.

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

Group	Median OS
Perifosine + capecitabine	17.7 months (95% CI, 8.5 to 24.6)
Placebo + capecitabine	7.6 months (95% CI, 5.0 to 16.3)

Of notable interest 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 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 weeks) N (%) p=0.066	Median TTP p<.001	Median OS p=0.0088
Perifosine + capecitabine	14(70%)	1 PR /8 SD (64%)	17.6 weeks (95% CI, 12 to 36)	15.1 months (95% CI, 7.2 to 22.3)

Capecitabine	11(73%)	0 PR /3 SD (27%)	9.0 weeks (95% CI, 6.6 to 11.0)	6.5 months (95% CI, 4.8 to 10.9)

* 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

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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.

Based on the data, in which the combination of perifosine + capecitabine demonstrated statistical significance with respect to median OS and median time to tumor progression, the investigators concluded that the perifosine + capecitabine combination showed promising clinical activity compared to single-agent capecitabine, and that the difference in clinical outcome seen with the addition of perifosine was impressive.

Phase 3 trial and regulatory milestones

On February 3, 2010, we announced that our partner Keryx had reached an agreement with the FDA on a SPA for the Phase 3 Xeloda® + Perifosine Evaluation in Colorectal Cancer Treatment (X-PECT) trial for perifosine in patients with refractory mCRC.

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 mCRC. Patients must have failed available therapy including 5-FU, 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 OS, with secondary endpoints including ORR (CR + PR), progression-free survival (PFS) and safety. Approximately 70 U.S. sites are participating in the study and un-blinding of the study will be triggered by 360 events of death. Dr. Johanna Bendell, Director of GI Oncology Research for the Sarah Cannon Research Institute, Nashville, Tennessee, leads the Phase 3 investigational team. Patient recruitment for this study was completed in July 2011.

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 CRC. 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 CRC. We do not intend to initiate any additional studies with perifosine for this indication. Therefore, for the development of perifosine in both MM and CRC, 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 27, 2011, we announced completion of patient recruitment for the ongoing Phase 3 trial with perifosine in refractory advanced CRC, involving over 465 patients from 65 sites in the United States. This Phase 3 X-PECT trial is a randomized (1:1), double-blind trial comparing the efficacy and safety of perifosine + capecitabine vs. placebo + capecitabine. The primary endpoint is OS, with secondary endpoints including ORR (CR + PR), PFS and safety. Approximately 360 events of death will trigger the unblinding of the study.

On August 31, 2011, an independent Data Safety Monitoring Board (DSMB) completed an interim safety and futility analysis of the Phase 3 X-PECT study of perifosine in patients with refractory advanced CRC and recommended that the study continue to completion, as planned.

On January 3, 2012, we announced that our Japanese partner, Yakult, had initiated a Phase 1/2 trial in Japan to assess the safety and efficacy of perifosine in combination with a chemotherapeutic agent, capecitabine, in patients with refractory advanced CRC. The primary endpoint of the Phase 1 portion of the trial is the safety profile of perifosine in combination with capecitabine. The primary endpoint of the Phase 2 portion is efficacy (Disease Control Rate). The initiation of this trial on December 27, 2011 triggered a milestone receivable of $2.6 million (according to the agreement signed in March 2011 for perifosine in Japan).

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Competitors for Perifosine in CRC

Products on the market:

Surgery is often the main treatment for early stage CRC. When CRC metastasizes (spreads to other parts of the body such as the liver), chemotherapy is commonly used. Treatment of patients with recurrent or advanced CRC depends on the location of the disease. Currently, there are seven approved drugs for patients with mCRC: 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 (5-FU; leucovorin; oxaliplatin) and FOLFIRI (5-FU; leucovorin; irinotecan), or used after one another. Bevacizumab, a vascular endothelial growth factor (VEGF) monoclonal antibody, is commonly administered with chemotherapy. Typically, patients who fail 5-FU, oxaliplatin, irinotecan, and bevacizumab-containing therapies, and who have wild-type KRAS status receive epidermal growth factor receptor (EGFR) monoclonal antibody therapy with either cetuximab or panitumumab. Once patients progress on these agents, there are no further standard treatment options.

Product / mode of action	Company	Sales
Avastin® (bevacizumab) / a humanized monoclonal antibody targeting vascular endothelial growth factor	Manufactured by Genentech/Roche	According to Decision Resources (Jan. 2012), a research and advisory firm focusing on pharmaceutical and healthcare issues (Decision Resources (January 2012)) the 2010 G7* sales were estimated to be $2.3 billion
Oxaliplatin / platinum agent	Sanofi s Eloxatin/Eloxatine, Yakult Honsha s Elplat, generics	According to Decision Resources (January 2012), the 2010 G7* sales were estimated to be $2.0 billion
Erbitux® (cetuximab) / a chimeric monoclonal antibody that specifically blocks EGFR	Manufactured and distributed in North America: Bristol-Myers Squibb Co. and Eli Lilly and Co. Distributed in the rest of the world by Merck KGaA.	According to Decision Resources (January 2012), the 2010 G7* sales were estimated to be $1.1 billion
Xeloda® (capecitabine) / oral fluoropyrimidine which generates fluorouracil preferentially in tumor tissues by enzymatic cascade	Manufactured by Roche	According to Decision Resources (January 2012), the 2010 G7* sales were estimated to be $636 million
Vectibix® (panitumumab) / a recombinant, human IgG2 kappa monoclonal antibody that binds specifically to the human EGFR	Manufactured by Amgen	According to Decision Resources (January 2012), the 2010 G7* sales were estimated to be $266.1 million

* G7 = United States, France, Germany, Italy, Spain, UK and Japan.

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Products in Phase 3 development:

According to Decision Resources (January 2012), the most promising emerging therapies for CRC are the following: perifosine, regorafenib, aflibercept and ramucirumab.

Product / mode of action*	Company*	Development Status*
Regorafenib (BAY 73-4506) / oral multi-kinase inhibitor targeting angiogenic, stromal and oncogenic kinases	Bayer HealthCare Pharmaceuticals / Onyx Pharmaceuticals, Inc.	Phase 3 completed
Aflibercept (Zaltrap™) / intravenous angiogenesis inhibitor	Sanofi / Regeneron Pharmaceuticals	Regulatory applications for marketing approval submitted (FDA and EMA)
Ramucirumab (IMC-1121B) / intravenous fully human immunoglobulin G1 Mab targeted to VEGF-2	ImClone Systems (a subsidiary of Eli Lilly)	Phase 3

* Source: Company s Website

Market Data CRC

According to the American Cancer Society, CRC is the third most common cancer in both men and women in the United States. An estimated 103,170 cases of colon and 40,290 cases of rectal cancer are expected to occur in 2012 and nearly 51,690 people will die from the disease.

According to Decision Resources (January 2012), 368,000 patients will be diagnosed in Stage II-IV in the G7 market in 2014. In 2010, the total market for stage IV-second-line CRC treatments accounted for 25% of major-market sales ($1.8 billion) and 5% ($360 million) for the total market for the third-line CRC treatment.

Treatable pool stage IV patient population*		
	3rd line in 2014	2nd line in 2014
USA	28,320	41,650
EU-5	48,070	74,480
Japan	19,750	35,270

* According to Decision Resources (January 2012).

1.1.1.2 Perifosine MM

Phase 1/2 trial Perifosine in combination with Revlimid® (lenalidomide) + 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 + dexamethasone. Four cohorts of ³6 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, then 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 3 non-hematologic toxicity, Grade 4 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,

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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 40 mg/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 (³10%) 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 ³5% 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 8 patients and dexamethasone reduced in 6 patients.

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

Updated results of this Phase 1 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 PFS in responding patients of 10.9 months. Median OS still was not reached and was at 17 months at time of analysis.

On December 6, 2010 at the American Society of Hematology s (ASH) 52nd annual meeting in Orlando, Florida, we announced final positive results for this Phase 1 trial. The final data showed a 73% objective response rate (minimal response or better) with a 50% PR or better, a median PFS of 10.8 months, and a median duration for OS 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.

Best Response (N = 30 pts)	**N (%)**	**Duration on Tx (months) Median (range)**	**³ PR**	**³ MR**
Near complete response (nCR)	4(13%)	32+, 32+, 28, 6	50%	73%
Very good partial response (VGPR)	3(10%)	35, 7, 4		
Partial response (PR)	8(27%)	Median 5.5 (4 29)		
Minimal reponse (MR)	7(23%)	Median 12 (2 34)		

Stable disease (SD)	6(20%)	Median 3 (2 30)
Progressive disease (PD)	2(7%)	9, 4 weeks

Phase 1/2 trial Perifosine in combination with Velcade® (bortezomib) + dexamethasone

Keryx presented preliminary 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 in December 2008 during the meeting of the ASH and in February 2009 at the 12th International Multiple Myeloma Meeting. Final results were presented during the ASH meeting in December 2009. The Phase 1 stage of the study enrolled a total of 18 patients in 4 cohorts of 3 patients each with dosing of perifosine

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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 PD. For the Phase 1 portion, DLT was defined as any Grade 3 non-hematologic toxicity, Grade 4 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.

On October 13, 2011, we announced that the manuscript, entitled *Perifosine Plus Bortezomib and Dexamethasone in patients with Relapsed/Refractory Multiple Myeloma Previously Treated with Bortezomib: Results of a Multicenter Phase 1/2 Trial,* had been published in the October 10, 2011 online edition of the *Journal of Clinical Onclogy* (JCO), in which Phase 1/2 combination activity of perifosine in the treatment of advanced MM patients was reported.

Eighty-four patients were enrolled in a combined Phase 1/2 study (18 patients in the Phase 1 component and 66 patients in the Phase 2 component), including 74 (88%) with relapsed/refractory MM. Sixty-one patients (73%) were bortezomib refractory and 43 (51%) were refractory to bortezomib with dexamethasone. The median number of prior treatments was five (range, one to 13 treatments). Prior therapies received at any time point included bortezomib (100%), with patients receiving a median of two (range, one to four) prior bortezomib-based regimens; dexamethasone (98%); lenalidomide (Revlimid®) (76%); thalidomide (Thalomid®) (75%); and prior stem cell transplant (58%).

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). 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	22%	17	38%	14	31%

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Reported for the first time was median PFS and 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, 16.3, 31.1)***

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

** Kaplan Meier methodology was used to determine OS figures.

*** Results from the abstract *Perifosine Plus Bortezomid and Dexamethasone in Relapsed/Refractory Multiple Myeloma Patients Previously Treated with Bortezomib: Final Results of a Phase 1/2 Trial* (abstract # 815) were presented as an oral presentation at the ASH Annual Meeting and Exposition (Dec. 2011) in San Diego, California.

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)	30.4 months (95% CI, 17.8, NR)***
Bortezomib refractory (N = 53)	5.7 months (95% CI, 4.3, 6.4)	22.5 months (95% CI, 14.2, 31.1)***

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 OS figures.

*** Results from the abstract *Perifosine Plus Bortezomid and Dexamethasone in Relapsed/Refractory Multiple Myeloma Patients Previously Treated with Bortezomib: Final Results of a Phase 1/2 Trial* (abstract # 815) were presented as an oral presentation at the ASH Annual Meeting and Exposition (Dec. 2011) in San Diego, California.

No unexpected adverse events have been observed. Therapy was generally well-tolerated, and toxicities, including gastrointestinal side-effects and fatigue, proved manageable. No treatment-related mortality was seen. The investigators concluded the data reported for both safety and efficacy in this patient population were encouraging for the continued study of perifosine.

Phase 3 trial and regulatory milestones

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 MM 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 had received orphan-drug designation for perifosine from the FDA for the treatment of MM. 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 United States. 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 MM. 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

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relapsed/refractory MM (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 PFS and secondary endpoints include ORR, OS and safety. 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 COMP of the EMA, for the treatment of MM. 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, preauthorization inspection and post-authorization activities, and could provide ten years of market exclusivity in the EU, once approved for the treatment of MM.

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

Competitors for Perifosine in MM

Products on the market

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

Product / mode of action*	Company*	Development Status*
Velcade® (bortezomib) / proteasome inhibitor	Takeda/Janssen-Cilag/Janssen	According to Decision Resources (January 2012), the 2010 G7** sales were estimated to be $1.14 billion
Caelyx® /Doxil® (pegylated liposomal doxorubicin) / topoisomerase II inhibitor and DNA intercalating agent	Janssen (J&J): Doxil® / Merck & Co.: Caelyx	According to Decision Resources (January 2012), the 2010 G7** sales were estimated to be $36.2 million
Thalomid® (thalidomide) / antiangiogenic compound	Celgene Corporation	According to Decision Resources (January 2012), the 2010 G7** sales were estimated to be $383.9 million
Revlimid® (lenalidomide) / oral immunomodulatory drug	Celgene Corporation	According to Decision Resources (January 2012), the 2010 G7** sales were estimated to be $1.94 billion

* Source: Company s Website

** G7 = United States, France, Germany, Italy, Spain, UK and Japan.

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Products in Phase 3 development:

Product / mode of action*	Company*	Development Status*
Carfilzomib / selective, irreversible proteasome inhibitors	Onyx Pharmaceuticals	- NDA submitted to the FDA
Pomalidomide / small-molecule, immunomodulatory drugs (angiogenesis, modulation of proinflammatory and regulatory cytokines, and immune stimulation of T cells and NK cells)	Celgene Corporation	- Phase 2 and Phase 3 - Phase 3 - Phase 2 completed
Panobinostat (LBH5893) / highly potent pan-deacetylase inhibitor targeting the epigenetic regulation of multiple oncogenic pathways	Novartis	Phase 3
Zolinza (vorinostat MK0683) / oral histone deacetylase inhibitor	Merck	Phase 3 completed
Elotuzumab (HuLuc63) / humanized MAb	Bristol-Myers Squibb/Abbott	Phase 3

* Source: Company s Website

Market Data MM

MM is the second most common blood cancer in the United States and constitutes approximately 1% of all cancers (*source: NCI*).

According to Decision Resources Patient Data Base (March 2012), 49,840 patients will be diagnosed in the G7 market in 2014.

Treatable pool relapsed/refractory patient population*

	3rd line in 2014	2nd line in 2014
USA	10,080	14,830
EU-5	11,840	17,410
Japan	2,860	4,200

* According to Decision Resources Patient Data Base (March 2012).

1.1.1.3 Perifosine Other indications

Perifosine is also studied in different indications including gliomas, RCC, sarcoma, Hodgkin s lymphoma (HL), neuroblastoma and other indications. As a monotherapy, perifosine is being explored in CLL.

On April 4, 2011, we announced that two posters on perifosine were presented at the 102nd annual meeting of the AACR at the Orange County Convention Center in Orlando, Florida. Perifosine demonstrated antitumor activity in several gastric cancer cell lines. Furthermore, perifosine enhanced the antitumor activity of 5-FU in parts of the cell lines, including 5-FU resistant cell lines. 5-FU is the active metabolite of the prodrug Xeloda, which is approved for the treatment of advanced gastric cancer in many countries including Japan.

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Perifosine also markedly enhanced the antitumor activity of the cellular TRAIL based treatment and was able to overcome TRAIL resistance both *in vitro* and *in vivo*. The results are in line with other studies demonstrating the synergistic effects of perifosine with cytotoxic drugs, including bortezomib and 5-FU.

On December 12, 2011, we reported encouraging preclinical data for perifosine in HL. *In vitro* data from HL cell lines showed that perifosine combined with sorafenib induced increased apoptosis, while *in vivo* data for the same combination treatment for HL significantly increased survival in mice. Data were presented during the ASH Annual Meeting and Exposition, in San Diego, California.

The study was conducted to investigate, *in vitro* and *in vivo*, the activity and mechanism(s) of action of perifosine in combination with sorafenib by using three HL cell lines (HD-MyZ, L-540, HDLM-2). In the *in vitro* experiments perifosine/sorafenib treatment resulted in synergistic cell growth inhibition and cell death induction in HD-MyZ and L-540 cell lines, but not in the HDLM-2 cell line. Cell cycle arrest, down-modulating of the MAPK and PI3K/Akt pathways as well as caspase-independent cell death was observed, which was associated with severe mitochondrial dysfunction. Further, expression of genes involved in amino acid metabolism, cell cycle, DNA replication and cell death was shown to be modulated. In addition, overexpression of the tribbles homologue 3 [TRIB3] was observed.

In vivo, perifosine/sorafenib treatment significantly increased survival in the HD-MyZ model (45 vs. 81 days, as compared to controls), with 25% tumor-free mice at the end of the 200-day observation period. In the L-540 model, subcutaneous tumor volume was also reduced as compared to controls (by 42%), perifosine (by 35%) or sorafenib (by 46%) alone. In HD-MyZ and L-540 but not HDLM-2, the combined treatment induced an increase in tumor necrosis (2- to 8-fold, P £.0001) and in tumor apoptosis (2- to 2.5-fold, P £.0001). In 2 of 3 HL cell lines, perifosine/sorafenib combination treatment induced potent antitumor effects. A significant increase in mitochondrial injury and apoptosis and a marked reduction of cell viability were observed in the *in vitro* experiments. *In vivo* combination treatment increased survival and inhibited tumor growth.

On December 13, 2011, we reported encouraging clinical data for an ongoing Phase 2 clinical study in patients with refractory/relapsed HL during the ASH Annual Meeting and Exposition.

Preliminary response data showed that perifosine combined with sorafenib significantly increased median PFS in refractory/relapsed HL patients with high phosphorylation levels of Erk and Akt as compared to patients with low baseline phosphorylation levels of Erk and Akt.

The study evaluates phosphorylation levels of Erk (pErk) and Akt (pAkt) in circulating lymphocytes from patients enrolled in two consecutive Phase 2 trials evaluating activity and safety of sorafenib as a single agent or in combination with perifosine in relapsed/refractory HL patients. Four patients were treated with sorafenib alone at a dose level of 400 mg BID and twenty-one patients received a 4-week treatment with perifosine alone at a dose level of 50 mg BID, followed by a perifosine/sorafenib combination therapy with 50 mg BID and 400 mg BID, respectively. Circulating lymphocytes were evaluated for their phosphorylation levels of Erk and Akt, in order to assess predictive value of the phosphokinase levels for therapy responses.

Clinical response data showed that baseline pErk and pAkt levels were significantly higher in responsive patients, as compared to unresponsive patients. The pErk and pAkt levels measured after 60 days of therapy with perifosine combined with sorafenib were significantly reduced in responsive patients. The median baseline value of pErk and pAkt efficiently discriminated responsive and unresponsive patients which was associated with a significantly improved median PFS for patients with baseline pErk ³43% and/or pAkt >23%. Based on these data, the correlation of baseline pErk and pAkt levels with objective responses and time to tumor progression will need to be validated in prospective studies. In conclusion, refractory/relapsed HL patients with increased baseline levels of pErk and pAkt demonstrated increased PFS when treated with perifosine in combination with sorafenib.

1.1.1.4 Partners for perifosine

A CRADA was put in place with the National Institutes of Health/the NCI in May 2000.

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A cooperation and license agreement was signed in September 2002 with Access Oncology, Inc. (AOI), for the use of perifosine as an anticancer 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 partners studies, as well as milestone payments and scale-up royalties (from high single to low double digit) 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 (approximately $8.4 million). Also per the agreement, we are entitled to receive in addition, up to a total of 44 million (approximately $57.1 million) upon achieving certain pre-established milestones, including clinical and regulatory events in Japan. Furthermore, we will be supplying perifosine to Yakult on a cost-plus basis and we will be entitled to receive double-digit royalties on future net sales of perifosine in the Japanese market. Yakult will be responsible for the development, registration and commercialization of perifosine in Japan.

On November 23, 2011, we announced the signing of an exclusive commercialization and licensing agreement with Hikma for the registration and marketing of perifosine for the MENA region. Under the terms of the agreement, we received an upfront payment of $0.2 million and are entitled to receive up to a total of $1.8 million upon achieving certain pre-established milestones. Furthermore, we will be supplying perifosine to Hikma on a cost-plus basis and are entitled to receive double-digit royalties on future net sales of perifosine in the MENA region. Hikma will be responsible for the registration and commercialization of perifosine in the MENA region. We own rest of the world rights to perifosine.

1.1.2 Erk/PI3K inhibitors and dual kinase inhibitors

The Ras/Raf/Mek/Erk and the PI3K/Akt signaling pathways are prime targets for drug discovery in proliferative diseases such as cancer. The results of research to date indicate that both the MAPK and the PI3K signaling pathways represent therapeutic intervention points for the clinical treatment of malignant tumors.

Our multi-parameter optimization program for kinase inhibitor selectivity, cellular efficacy, physicochemical and *in vitro* ADMET properties has led to the identification of small molecular compounds with a unique kinase selectivity profile. Our kinase research program comprises the investigation of different compounds for single Erk inhibition, single PI3K inhibition and dual Erk/PI3K kinase inhibition.

1.1.2.1 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 antitumor 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.

On March 22, 2011, we presented preclinical results for AEZS-129 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-129 was identified as a potent inhibitor of class I PI3Ks lacking activity against mTOR. Lack of mTOR activity is considered to potentially lead to a better safety profile. In biochemical and cellular assays, AEZS-129 demonstrated favorable properties in early *in vitro* ADMET screening, including

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microsomal stability, plasma stability and screening against a safety profile composed of receptors, enzymes and cardiac ion-channels. *In vitro*, the compound was shown to be a selective ATP-competitive inhibitor of PI3K with a broad antiproliferative activity against a broad panel of tumor cell lines. *In vivo*, AEZS-129 showed excellent plasma exposure and significant tumor growth inhibition in several tumor xenografts models, including A-549 (lung), HCT-116 (colon) and Hec1B (endometrium). These data suggest that AEZS-129 is a promising compound for clinical intervention of the PI3K/Akt pathway in human tumors.

1.1.2.2 AEZS-131

On March 22, 2011, we presented preclinical results for AEZS-131 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-131 was established as a small molecular compound that inhibits Erk in the low nanomolar (nM) range and shows an excellent selectivity profile. Further characterization experiments revealed an ATP-competitive mode of action and the potent inhibition of the cellular downstream target Rsk1 in tumor cells. The frontrunner, AEZS-131, produces cell cycle arrest in G1 and results in growth inhibition of cancer cells. Furthermore, the potential of combination therapy of AEZS-131 with inhibitors of the PI3K pathway was addressed and the analysis of combination effects on tumor cell proliferation has been presented. These results support the evaluation of selective Erk inhibitors as antiproliferative agents either as monotherapy or in combination with inhibitors of the PI3K/Akt pathway.

On April 5, 2011, we announced results for AEZS-131 at the AACR s annual meeting. Results showed that AEZS-131 is an orally active small molecular compound that selectively inhibits Erk 1/2 with an IC50 of 4nM, blocks cellular Rsk-1 phosphorylation, modulates downstream cellular substrate activation, arrests tumor cells in G1 and inhibits the growth of multiple human tumor cell lines in the nM range. In *in vivo* pharmacokinetic studies, AEZS-131 showed a favorable PK profile. Antitumor activity was studied in *in vivo* mouse xenograft experiments utilizing the HCT 116 CRC model. AEZS-131 significantly inhibited tumor growth and was well tolerated at daily doses up to 120 mg/kg. Focus on inhibition of downstream kinase Erk 1/2 activity as a therapeutic target may be attractive because the pharmacologic inhibition of Erk 1/2 reverses Ras and Raf activation in cells which also demonstrate resistance to common Raf inhibitors, such as PLX-4720/4032.

On August 30, 2011, we presented preclinical data on AEZS-131 at the American Chemical Society National Meeting in Denver, Colorado. Data showed that the *in vitro* antiproliferative efficacy proved to be excellent in diverse human tumor cell lines and that GI_{50} values in the low nM range were obtained. *In vivo* antitumor activity was studied in a mouse xenograft experiment utilizing the human HCT-116 colon cancer model. Up to 74% inhibition of tumor growth was achived with daily oral doses of 30 120 mg/kg. Our medicinal chemistry programs are supported by X-ray crystallography and modeling towards the optimization of pyrido[2,3-b]pyrazines as novel series of kinase inhibitors.

On December 9, 2011, we announced positive preclinical data in triple-negative breast cancer (TNBC) for AEZS-131. Data showed that AEZS-131 selectively inhibits Erk at low nM concentrations and induces G1 arrest. Accordingly, the cytotoxic effect of AEZS-131 was most pronounced in TNBC cell lines with mutations in the MAPK pathway. Data were presented at the CTRC - AACR San Antonio Breast Cancer Symposium, in San Antonio, Texas. AEZS-131 was tested to check for selectivity, inhibition of Rsk-phosphorylation (cellular substrate of Erk), mode of action and cleavage of PARP. Cytotoxic efficacy was evaluated in a selection of TNBC cell lines, with or without mutations in the MAPK signal transduction pathway, by MTT assay. The study showed that AEZS-131 selectively inhibited Erk with an IC50<4nM. Phosphorylation of Rsk-1, the cellular substrate of Erk, was inhibited with an IC50 of 158 nM. AEZS-131 induced cell cycle arrest in G1 dose-dependently and cleavage of PARP. EC50 values were below 1µM for cell lines with mutations in the MAPK pathway. TNBC cell lines without mutations in the MAPK pathway were less responsive.

1.1.2.3 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.

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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 antitumor 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.

On March 22, 2011, we presented preclinical results for AEZS-132 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-132 is a unique dual inhibitor of PI3K and Erk in the nM range and exerts high selectivity against other serine threonine and tyrosine kinases. AEZS-132 is also an ATP-competitive inhibitor, with a broad antiproliferative profile *in vitro*, a favorable safety profile and beneficial ADME properties. *In vivo* pharmacokinetic experiments showed plasma profiles expected to result in positive antitumor efficacy, and led to significant antitumor activity in mouse xenograft models, including HCT-116 (colon), A-549 (lung), and Hec1B (endometrium). Cellular inhibition of the downstream targets p-Akt and p-Rsk was confirmed within the *in vivo* tumor studies. In summary, we hope that AEZS-132, by targeting the PI3K and MAPK pathways, will be especially suited to treat tumors with over-activation of both pathways.

1.2 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.

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

In June 2006 and on November 27, 2006, we disclosed positive Phase 1 results regarding AEZS-108 in patients with gynaecological and breast cancers. 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. Infusion was well tolerated at all dosages without supportive treatment. Pharmacokinetic analyses showed dose-dependent plasma levels of AEZS-108 and only minor release of doxorubicin. The Phase 1 open-label, multicenter, 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 antitumor 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. Recommended dose for Phase 2 trial was 267 mg/m^2 given once every three weeks (*source: Emons et al. Dose escalation and pharmacokinetic study of AEZS-108 (AN-152), an LHRH agonist linked to doxorubicin, in women with LHRH receptor-positive tumors. Gynecol Oncol. 2010 Dec;119(3):457-61*).

1.2.1 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, M.D., Chairman of the Department of Obstetrics & Gynaecology at the University of Göttingen, Germany, this open-label, multicenter and multinational 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. As part of a personalized healthcare approach, the study selected patients with tumors expressing LHRH receptors, the key element in the targeting mechanism of AEZS-108. 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 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 TTP, survival, toxicity, as well as adverse effects. 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 PR among patients with a diagnosis of platinum-resistant ovarian cancer. The second stage of patient recruitment for the endometrial cancer indication was reached in November 2008 and was based on the report of one CR and two PR 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 and in patients with advanced or recurrent endometrial cancer. Of the 42 patients with LHRH receptor-positive ovarian cancer and the 43 patients with LHRH receptor-positive endometrial cancer who entered study AGO-GYN, 5 completed their study treatment. A preliminary evaluation showed that both indications met the 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 PFS and, ultimately, OS.

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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.

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.

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*, 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.

Forty-two patients with platinum-resistant ovarian cancer entered the study. Efficacy included PR in 5 patients (11.9%) and stable disease for more than 12 weeks in 11 patients (26.2%). Based on those data, a CBR of 38% can be estimated. Median TTP and OS were 3.5 months (104 days) and 15.6 months (475 days), respectively. OS 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 September 14, 2011, we presented positive final Phase 2 efficacy and safety data for AEZS-108 in advanced endometrial cancer at the European Society of Gynecological Oncology in Milan, Italy. Data showed that AEZS-108, administered as a single agent at a dosage of 267 mg/m^2 every 3 weeks was active, well tolerated and that OS is similar to that reported for modern triple combination chemotherapy, but was achieved with lower toxicity. The primary endpoint was the response rate as defined by the RECIST. Secondary endpoints included safety, TTP and OS.

In all, of 43 patients treated with AEZS-108, 39 were evaluable for efficacy. Efficacy confirmed by independent response review included 2 CR, 10 PR, and 17 patients with SD. Based on those data, the estimated ORR (ORR = CR+PR) was 30.8% and the CBR (CBR = CR+PR+SD) was 74.4%. Responses in patients previously treated with chemotherapy included 1 CR, 1 PR and 2 SDs in 8 of the patients with prior use of platinum/taxane regimens. Median TTP and OS were 7 months and 13.7 months, respectively.

Overall, tolerability of AEZS-108 was good and commonly allowed retreatment as scheduled. Severe (Grade 3 or 4) toxicity was mainly restricted to rapidly reversible leukopenia and neutropenia, associated with fever in only 1 patient who had been treated only 3 weeks after a surgery. Good tolerability of AEZS-108 was also reflected by a low rate of severe non hematological possibly drug-related adverse events which included single cases each of nausea, diarrhea, fatigue, general health deterioration, creatinine elevation, and blood potassium decrease. No cardiac toxicity was reported.

Competitors for AEZS-108 in Endometrial Cancer

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

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Product / mode of action*	Company*	Development Status*
Ixabepilone / microtubule inhibitor	Bristol-Myers Squibb	Phase 3
Letrozole / non-steroidal aromatase inhibitor	Novartis	- Phase 2 completed - Phase 3 (University of California, Davis)
XL-147 (Exelixis) / potent and highly selective inhibitor of the class I PI3K family of lipid kinases which targets the PI3K/PTEN pathway	Sanofi	Phase 2

* Source: Company s Website and www.clinicaltrials.gov

Market Data Endometrial Cancer

According to the American Cancer Society, endometrial cancer is the most common invasive gynecologic cancer in women in the United States, with an estimated 47,130 new cases expected to occur in 2012. This disease primarily affects postmenopausal women at an average age of 60 years at diagnosis. In the United States, it is estimated that approximately 8,010 women will die of endometrial cancer in 2012.

According to Datamonitor Healthcare (March 2010), a research and advisory firm focusing on therapeutic, strategic and ehealth market analysis and competitive intelligence, the incidence of endometrial cancer in the seven major pharmaceutical markets was 92,180 patients and is forecasted to reach about 98,500 cases by 2019.

1.2.2 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 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 OS. 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 IND application for AEZS-108 in LHRH receptor-positive urothelial (bladder) cancer. Following this approval from the FDA, this trial will be conducted by Dr. Gustavo Fernandez 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 PFS.

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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 both Phase 1/2 trials.

On September 26, 2011, we announced positive interim data for the Phase 1 portion of our Phase 1/2 trial with AEZS-108 in castration-and taxane-resistant prostate cancer at the ESMO meeting, Stockholm, Sweden. This is a single arm study with a Phase 1 lead-in to a Phase 2 clinical trial. The primary endpoint of the Phase 1 portion is safety. The primary objective of the Phase 2 portion is to evaluate the clinical benefit of AEZS-108 for these patients. Twelve patients entered the study: 3 patients each received AEZS-108 at the lower dose levels of 160 and and 210 mg/m^2, and 6 patients at 267 mg/m^2. Data on 10 patients were presented as 2 patients were too early for evaluation. AEZS-108 was generally well tolerated and there were no dose limiting toxicities so far. The only Grade 3 and 4 toxicities were hematologic in nature. At the time, there were three Grade 4 toxicities and six Grade 3 toxicities. Signs of therapeutic activity included 5 patients with PSA regression. Three out of 4 evaluable patients with radiologic evaluable disease achieved stable disease per RECIST. The Phase 2 extension is planned after completion of the toxicity assessment in the final dose level of the Phase 1 portion of the study. In correlative studies, drug internalization was demonstrated for the first time in captured circulating tumor cells of patients, thus validating the principle of targeted tumor therapy with AEZS-108 in a clinical setting.

On February 3, 2012, we reported positive updated results for the Phase 1 portion of our ongoing Phase 1/2 study in castration- and taxane-resistant prostate cancer (CRPC) with AEZS-108. This is a single-arm study with a Phase 1 lead-in portion (testing 3 dose levels) to a Phase 2 clinical trial. The primary endpoint of the Phase 1 portion is safety. The primary objective of the Phase 2 portion is to evaluate the clinical benefit of AEZS-108 for these patients. Data were presented by Jacek Pinski, M.D., Ph.D., Associate Professor of Medicine at the Norris Comprehensive Cancer Center of the University of Southern California, during a poster session at the American Society of Clinical Oncology Genitourinary Cancers Symposium which is being held in San Francisco. Prior interim data on this study were presented at the European Society of Medical Oncology Congress in September 2011. The trial is being supported by a three-year US$1.6 million grant from the National Institutes of Health to Dr. Pinski.

The results were based on 13 patients that were treated on 3 dose levels: 3 at 160 mg/m^2, 3 at 210 mg/m^2, and 7 at 267 mg/m^2. Overall, AEZS-108 was well tolerated among this group of heavily pretreated older patients. To date, there have been 2 dose limiting toxicities; both were cases of asymptomatic Grade 4 neutropenia at the 267 mg/m^2 dose level and both patients fully recovered. The Grade 3 and 4 toxicities were primarily hematologic. There has been minimal non-hematologic toxicity, most frequently fatigue and alopecia. Despite the low doses of AEZS-108 in the first cohorts, there was some evidence of antitumor activity. One patient received 8 cycles (at 210 mg/m^2) due to continued benefit. Among the 5 evaluable patients with measurable disease, 4 achieved stable disease. At the time of submission of the abstract, a decrease in PSA was noted in 6 patients. Six of 13 (46%) treated patients received at least 5 cycles of therapy with no evidence of disease progression at 12 weeks. Correlation studies on circulating tumor cells (CTCs) demonstrated the uptake of AEZS-108 into the targeted tumor. After completion of 3 additional patients at the 210 mg/m^2 dose level, the study is expected to be extended into the Phase 2 portion.

1.2.3 AEZS-108 Triple-Negative Breast Cancer

On October 25, 2011, we announced that the FDA had completed the review of the IND application filed by Alberto J. Montero M.D. of the Sylvester Comprehensive Cancer Center and concluded that Dr. Montero may proceed with the initiation of a randomized Phase 2 trial in chemotherapy refractory triple-negative (ER/PR/HER2-negative) LHRH receptor-positive metastatic breast cancer with AEZS-108. This open-label, randomized, two-arm, multicenter Phase 2 study will involve up to 74 patients who will be randomized in a 1:1 ratio into one of the two treatment arms: AEZS-108 (267 mg/m^2 every 21 days) [Arm A] or standard single agent cytotoxic chemotherapy [Arm B].

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The primary study endpoint will be PFS. Secondary endpoints will include ORR and OS. The study will also evaluate AEZS-108 s toxicity profile and the patients quality of life relative to conventional cytotoxic chemotherapy used in the control arm of this study.

1.2.4 AEZS-108 - Companion Diagnostic Tool

On January 5, 2012, we announced that we had entered into a collaboration agreement, dated December 19, 2011, with Ventana, to develop a companion diagnostic for the immunohistochemical determination of LHRH receptor expression, for AEZS-108. In humans, LHRH receptors are expressed in a significant proportion of endometrial, ovarian, breast, bladder, prostate and pancreatic tumors. AEZS-108 specifically targets LHRH receptors and could therefore prove to be more efficient in treating patients with these types of LHRH receptor-positive cancers.

1.2.5 AEZS-137 (Disorazol Z) / AEZS-125 (LHRH-Disorazol Z)

In search of new antitumor agents, we found that disorazol Z (AEZS-137), isolated from the myxobacterium *Sorangium cellulosum*, possesses remarkable, non-specific cytotoxicity in a panel of different tumor cell lines. Inhibition of tubulin polymerization, cell cycle arrest and efficient induction of apoptosis, have been identified as modes of action. AEZS-137 is thus a bacterially produced compound with cytotoxic activity in the sub-nanomal range.

In order to obtain a specifically acting antitumor agent, we have linked disorazol Z to [D-Lys^6] LHRH. The resulting conjugate, AEZS-125, has been characterized with respect to *in vitro* and *in vivo* antitumor activity. In CD 1 nu/nu mice xenografted with the LHRH receptor-positive, human ovarian carcinoma cell line OVCAR-3, we have shown tumor suppression by single administration of AEZS-125 in doses as low as 45 nmol/kg (0.1 mg/kg). Proof of concept for this approach is the far more efficient tumor suppression obtained with AEZS-125 in comparison to equimolar doses of disorazol Z itself. The results were published during the 99th AACR Annual Meeting in April 2008 in San Diego, California.

On March 24, 2011, we were awarded a $1.5 million grant from the German Ministry of Education and Research to develop, up to the clinical stage, cytotoxic conjugates of the proprietary cytotoxic compound AEZS-137 and peptides targeting G-protein coupled receptors, including the LHRH receptors. The compounds being developed will combine the targeting principle successfully employed in Phase 2 with AEZS-108 (doxorubicin and LHRH receptor targeting agent) with the novel cytotoxic disorazol Z. Furthermore, diagnostic tools systematically assessing the receptor expression in tumor specimens will be developed to allow the future selection of patients and tumor types with the highest chance of benefiting from this personalized medicine approach. The grant will be payable as a partial reimbursement of qualifying expenditures over a three-year period. The qualified project will be performed with Morphisto GmbH and the Helmholts Institute in Saarbrücken, Germany, which will receive additional funding of approximately US$0.7 million. Researchers from the departments of Gynecology and Obstetrics at both the University of Göttigen and the University of Würzburg, Germany, will also be part of the collaboration.

On November 16, 2011, we announced the presentation of a poster at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics on encouraging preclinical data for AEZS-137. Data showed that AEZS-137 possesses outstanding cytotoxicity in a highly diverse panel of 60 different tumor cell lines, and also underlined the identification of important aspects of this novel natural compound s mechanism of action. AEZS-137 has been identified as a tubulin binding agent with highly potent antitumor properties. Cell cycle analysis revealed that AEZS-137 arrested cells in the G2/M phase and subsequently induced apoptosis with remarkable potency, as shown by subnanomolar EC50 values. Currently, experiments are under way to determine the tubulin binding site for disorazol Z and to identify further mechanisms of action of this novel highly potent agent. To expand our AEZS-108 technology platform, we aim to evaluate the utility of disorazol Z as a cytotoxic component in a drug-targeting approach utilizing GPCR ligands as the targeting moieties for the treatment of GPCR over-expressing cancers.

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1.3 TUBULIN INHIBITORS / VASCULAR TARGETING AGENTS

1.3.1 AEZS-112 Development of a Low Molecular Weight Tubulin Inhibitor with Antiangiogenic 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 antitumor agents 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 improved efficacy in animal models, 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 a favorable safety and tolerability profile showing excellent *in vivo* activity in various tumor models including mammary, colon, melanoma and leukemia cancers at acceptable and very well tolerated doses administered orally once weekly. This compound acts through three mechanisms of action. Strong anticancer activity is combined with proapoptotic and antiangiogenic 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 nM concentration and induced apoptosis. AEZS-112 is not cross-resistant to cisplatin, vincristine and doxorubicine in cell lines resistant to these drugs. 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, multicenter, intermittent treatment Phase 1 trial is being conducted in the United States with Daniel D. Von Hoff, M.D., 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 was 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 200 x 3 mg/week, no clinically relevant 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

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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.

During the year 2011, we have developed a higher concentrated oral formulation of AEZS-112 in order to improve patient compliance. Clinical protocol is in preparation to continue to advance AEZS-112 with this new formulation.

1.4 IMMUNOTHERAPY / VACCINES

1.4.1 AEZS-120

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 filed in November 2006.

In 2007, the PSA vaccine (AEZS-120) was selected as the first preclinical development candidate of an antitumor vaccine. AEZS-120 is a live recombinant oral tumor vaccine candidate based on Salmonella typhi Ty21a as a carrier strain. Salmonella typhi Ty21a is an approved oral typhoid vaccine which has been safely applied in more than 350 million doses. The principle of AEZS-120 is based on the recombinant expression of prostate specific antigen fused to the B subunit of cholera toxin and a secretion signal in the presence of the Escherichia coli type I hemolysin secretion system. The proprietary system allows the secretion of the antigen together with an immunological adjuvant which has been demonstrated to be required for optimal induction of CD8 T-cell responses by recombinant Salmonella based bacterial vaccines. The proof-of-concept was already demonstrated for the mouse homologue of AEZS-120 in a mouse tumor challenge model.

A grant application was filed in Germany and was approved in 2008 for a preclinical development program for AEZS-120 with our research collaborators, the Department of Medical Radiation Biology and Cell Research, and the Department of Microbiology of the University of Würzburg, Germany. 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.

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On July 20, 2011, we reached a key milestone in this non-clinical development program of AEZS-120 which encompassed the full development of a GMP process, including GMP production and quality testing of a clinical batch, as well as a non-clinical safety and toxicology package. Once this non-clinical program will have been completed and subject to a positive review by regulatory authorities, we aim to start Phase 1 clinical development in 2012. AEZS-120 has been developed through a research collaboration with the Department of Medical Radiation Biology and Cell Research, and the Department of Microbiology of the University of Würzburg, Germany. The collaboration was funded with a total of $890,000 for us and $870,000 for the university partner by the German Ministry of Education and Research (BMBF) for a period of three years. As part of the collaboration, a melanoma vaccine based on the recombinant expression of a modified B-Raf protein has been generated.

2.0 ENDOCRINOLOGY

2.1 AEZS-130 ORAL GHRELIN AGONIST

AEZS-130, a ghrelin agonist, is a novel orally active small molecule that stimulates the secretion of growth hormone by binding the ghrelin receptor (GHSR-1a). It can be used in both endocrinology and in oncology indications. In endocrinology, we have completed a Phase 3 trial for its use as a simple oral diagnostic test for AGHD. AEZS-130 works by stimulating a patient s growth hormone secretion, which normally only occurs during sleep, after which a healthcare provider will measure how well the body responds to that stimulation based on the patient s growth hormone levels over a period of time. Low growth hormone levels, despite giving an effective stimulating agent, confirm a diagnosis of AGHD. AEZS-130 has been granted orphan-drug designation by the FDA for use as a diagnostic test for growth hormone deficiency. We own the worldwide rights to AEZS-130 which, if approved, would become the first orally administered diagnostic test for AGHD. In oncology, the FDA has allowed for the initiation of a physician sponsored IND Phase 2A trial with AEZS-130 in cancer induced cachexia, a disease which leads to significant weight loss and diminished functional performance. Since ghrelin agonists such as AEZS-130 have been shown to stimulate food intake and increase body weight in rats and mice, AEZS-130 could lead to better quality of life for patients with cancer induced cachexia. Ghrelin agonists have been in clinical trials for over a decade and have demonstrated good safety and efficacy profiles.

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 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 as a diagnostic test for AGHD was suspended.

In June 2009, we reported that, after having regained from Ardana in the prior year 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 for the diagnostic of growth hormone secretion deficiency.

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During the same month, the first clinical data relating to the use of AEZS-130 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, the responses to the orally administered AEZS-130 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 which could be the first oral diagnostic test approved for growth hormone deficiency (GHD). We 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 NDA for approval of AEZS-130 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 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.

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 has potential applications for the treatment of cachexia, a condition frequently associated with severe chronic diseases such as cancer, chronic obstructive pulmonary disease and AIDS.

On July 14, 2010, we announced the presentation of a poster on AEZS-130, 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 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, 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 first part of the study conducted by our former partner, Ardana, was a two-way cross-over study and included 42 patients with confirmed AGHD or multiple pituitary hormone deficiencies and a low insulin-like growth factor-I. A 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 AEZS-130 given in an oral solution of 0.5 mg/ml.

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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 receive a dose of 0.5 mg/kg body weight of AEZS-130; 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.

On July 26, 2011, we announced the completion of the Phase 3 study on AEZS-130 as a first oral diagnostic test for AGHD and the decision to meet with the FDA for the future filing of a NDA for the registration of AEZS-130 in the United States.

On August 30, 2011, we announced favorable top-line results of our completed Phase 3 study with AEZS-130 as a first oral diagnostic test for AGHD. Results showed that AEZS-130 reached its primary endpoint demonstrating >90% area-under-the-curve (AUC) of the Receiver Operating Characteristic (ROC) curve, which determines the level of specificity and sensitivity of the product. Importantly, the primary efficacy parameters show that the study achieved both specificity and sensitivity at a level of 90% or greater. In addition, 8 of the 10 newly enrolled AGHD patients were correctly classified by a pre-specified peak GH threshold level. The use of AEZS-130 was shown to be safe and well tolerated overall throughout the completion of this trial. We are currently proceeding with further detailed analyses of the data and preparing for a pre-NDA meeting with the FDA in the upcoming months, which would be followed by the filing of a NDA for the registration of AEZS-130 in the United States.

On November 28, 2011, we announced that the FDA had completed the review of an IND application filed by Jose M. Garcia, M.D., Ph.D., Assistant Professor, Division of Diabetes Endocrinology and Metabolism, Departments of Medicine and Molecular and Cell Biology, Baylor College of Medicine and Michael E. DeBakey Veterans Affairs Medical Center, in Houston Texas and concluded that Dr. Garcia may proceed with the initiation of a Phase 2A trial to assess the safety and efficacy of repeated doses of AEZS-130, in patients with cancer cachexia. Cachexia, which is characterized by diminished appetite and food intake in cancer patients, is defined as an involuntary weight loss of at least 5% of the pre-illness body weight over the previous 6 months.

On March 8, 2012, we announced that the Michael E. DeBakey Veterans Affairs Medical Center, in Houston, Texas, initiated a Phase 2A trial assessing the safety and efficacy of repeated doses of AEZS-130 in patients with cancer cachexia. The study is conducted under a CRADA between the Company and the Michael E. DeBakey Veterans Affairs Medical Center which is funding the study.

This is a double-blind, randomized, placebo-controlled Phase 2A trial to test the effects of different doses of the ghrelin agonist, AEZS-130, in 18 to 26 patients with cancer-cachexia. AEZS-130 will be provided by Aeterna Zentaris. The study will involve 3 sequential groups receiving differing doses of AEZS-130. Each dose group will have 6 patients who will receive AEZS-130 and 2-4 patients who will receive placebo. After analysis of safety and efficacy at each dose level vs. placebo, a decision will be taken either to decrease or increase the dose. If there are major safety issues, the study will be stopped. For this study, adequate efficacy will be defined as a ³0.8 kg of body weight gain or a ³50 ng/mL increase in plasma IGF-1 levels. The primary objective of the study is to evaluate the safety and efficacy of repeated oral administration of AEZS-130 at different doses daily for 1 week in view of developing a treatment for cachexia. The following parameters will be recorded to assess efficacy during the study: change of body weight, change of IGF-1 plasma levels, and change of quality of life score (Anderson Symptom Assessment Scale, FACIT-F). Other secondary objectives will include food intake, and changes in the following: appetite, muscle strength, energy expenditure, reward from food and functional brain connectivity.

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Competitors for AEZS-130

Competitors for AEZS-130 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 no longer available in the United States. This test is administered i.v.; and
- glucagon test, which is simple to perform and is considered relatively 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.

Oral administration of AEZS-130 offers more convenience and simplicity over the current GHD tests used, requiring either i.v. or i.m. administration. Additionally, AEZS-130 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 has not thus far. These factors may be limiting the use of GHD testing and may enable AEZS-130 to become the diagnostic test of choice for GHD.

Market Data AGHD

According to the Hormone Foundation, in the United States, about 35,000 adults have GHD, with about 6,000 newly diagnosed each year (source: Hormone Foundation s Website).

2.2 LHRH ANTAGONISTS

2.2.1 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.

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2.2.1.1 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 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. 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.

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In November 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.

Cetrorelix clinical development has been terminated in all indications other than *in vitro* fertilization (COS/ART). Furthermore, we focus on our manufacturing on behalf of Merck Serono and Shionogi.

2.2.2 Ozarelix

Ozarelix is a modified LHRH antagonist which is a linear decapeptide sequence. Ozarelix is a fourth-generation LHRH antagonist designed to extend the suppression of testosterone levels, which 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 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 our patent rights and know-how to develop, use, make, have made, sell, offer for sale, have sold, import, export and 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 us. Also per the amended agreement, we 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, we 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 our consolidated statement of operations for the year ended December 31, 2009.

2.2.2.1 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 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%.

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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.

Our partner, Spectrum, is currently recruiting patients in Phase 2 trial for the treatment of prostate cancer. This is an international, multicenter, open-label, randomized study assessing the safety and efficacy of a monthly dosing regimen of ozarelix versus goserelin depot in men with prostate cancer (*source: www.clinicaltrials.gov*).

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 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 at which 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 IND 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.

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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 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 NDA 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.

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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 have been granted orphan drug designations for perifosine in the MM and neuroblastoma indications, for AEZS-108 for the treatment of advanced ovarian cancer and for AEZS-130 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 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.

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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 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 , which are 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 approximately 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.

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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.

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.

Perifosine combined with antimetabolites:

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

U.S. patent application 10/632,187, filed July 2003 and further continuation applications US 12/751,454, US 12/751,608 and US 12/798,267 are still pending. Granted patents will be expire in July 2023;

European patent 1 545 553 expires in July 2023;

Japanese patent application 2004-525354, filed July 2003 is still pending. Granted patent will expire in July 2023.

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.

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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.

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.

U.S. patent 5,198,533 provided protection in U.S.A. for the compound cetrorelix per se. This patent expired in extended status in October 2010.

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.

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.

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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.

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

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
EP 1 545 553	Alkylphosphocholines combined with antimetabolites	Europe	2023-07-28
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. 7,605,121	Oligopeptide lyophilisate, their preparation and use	United States	2014-02-22
U.S. 7,790,686	Injection solution of an LHRH antagonist	United States	2023-10-28
AEZS-112			
U.S. 7,365,081	Indole derivatives and their use as medicaments	United States	2017-09-08

EP 1 309 585	Indole derivatives and their use as medicaments	Germany, United Kingdom, France, Switzerland and others	2021-07-26

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Patent No.	Title	Country	Expiry Date
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

* Including Patent Term Extension

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, 2011.

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 27, 2012.

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
25 Mountainview Blvd., Suite 203, Basking Ridge, NJ 07920	Fully occupied for management, R&D and administration	3,188	Leased
Weismüllerstr. 50 D-60314 Frankfurt-am-Main, Germany	Fully occupied for management, R&D, business development and administration	46,465	Leased

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Item 4A. Unresolved Staff Comments

None.

Item 5. Operating and Financial Review and Prospects

Highlights

Perifosine

§ March 9, 2011: We announced that we had entered into an agreement with Yakult Honsha Co. Ltd. (Tokyo: 2267) (Yakult) for the development, manufacture and commercialization of perifosine in all human uses, excluding leishmaniasis, in Japan. Under the terms of the agreement, we received an initial $8.4 million upfront payment and are entitled to receive additional payments of up to $57.1 million upon achieving certain pre-established milestones including clinical and regulatory events in Japan. Furthermore, we will be supplying perifosine to Yakult on a cost-plus-basis and be entitled to receive double-digit royalties on future net sales of perifosine in Japan. Yakult will be responsible for the development, registration and commercialization of perifosine in Japan.

§ April 4, 2011: We announced that two posters on our lead anticancer agent, perifosine, had been presented at the 102nd annual meeting of the American Association for Cancer Research (AACR) held at the Orange County Convention Center in Orlando, Florida. The first poster demonstrated perifosine s antitumor activity in several gastric cancer cell lines. Furthermore, perifosine enhanced the antitumor activity of 5-fluorouracil (5-FU) in parts of the cell lines - including 5-FU resistant cell lines. As for the second poster, perifosine markedly enhanced the antitumor activity of the cellular TRAIL based treatment and was able to overcome TRAIL resistance both *in vitro* and *in vivo*. The results are in line with other studies demonstrating the synergistic effects of perifosine with cytotoxic drugs, including bortezomib and 5-FU.

§ July 12, 2011: We announced that the European Patent Office had granted a patent for the use of alkylphosphocholines, more specifically perifosine, in combination with antitumor anti-metabolites, which include among others 5-FU and capecitabine in the preparation of a medicament for the treatment of benign and malignant tumors. This patent will expire on July 28, 2023.

§ July 27, 2011: We announced the completion of patient recruitment (over 465 patients) for the ongoing Phase 3 trial with perifosine in refractory advanced colorectal cancer (CRC).

§ August 31, 2011: We announced the completion of the pre-specified safety and futility interim analysis by the Data Safety Monitoring Board (DSMB) for the Phase 3 trial with perifosine in refractory advanced colorectal cancer. The DSMB recommended that the trial proceed as planned.

§ October 5, 2011: We announced that an article on perifosine had been published in the October 2011 online issue of the *Journal of Clinical Oncology* (JCO), in which positive Phase 2 results in metastatic colorectal cancer were reported.

§ October 13, 2011: We announced that an article on perifosine had been published in the October 2011 online issue of the JCO, in which positive Phase 1/2 results in multiple myeloma were reported.

§ November 23, 2011: We announced the signing of an exclusive commercialization and licensing agreement with Hikma Pharmaceuticals PLC (Hikma) (LSE: HIK) (NASDAQ Dubai: HIK) for the registration and marketing of perifosine for the Middle East and North Africa (MENA) region. Under the terms of the agreement, we received an upfront payment of $0.2 million and are entitled to receive up to a total of $1.8 million upon achieving certain pre-established milestones. Furthermore, we will be supplying perifosine to Hikma on a cost-plus-basis

and are entitled to receive double-digit royalties on future net sales of perifosine in the MENA region. Hikma will be responsible for the registration and commercialization of perifosine in the MENA territory.

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§ December 12, 2011: We announced encouraging preclinical data for perifosine in Hodgkin s lymphoma (HL). *In vitro* data from HL cell lines showed that perifosine combined with sorafenib induced increased apoptosis, while *in vivo* data for the same combination treatment for HL significantly increased survival in mice. Data were presented during the American Society of Hematology (ASH) Annual Meeting and Exposition held in San Diego, California.

§ December 13, 2011: We announced encouraging clinical data for an ongoing Phase 2 clinical study in patients with refractory/relapsed HL. Preliminary response data showed that perifosine combined with sorafenib significantly increased median progression free survival (PFS) in refractory/relapsed HL patients with high phosphorylation levels of Erk and Akt, as compared to patients with low baseline phosphorylation levels of Erk and Akt. Data were presented during the ASH Annual Meeting and Exposition held in San Diego, California.

§ On January 3, 2012: We announced that our Japanese partner, Yakult had initiated a Phase 1/2 trial in Japan to assess the safety and efficacy of perifosine, in combination with a chemotherapeutic agent, capecitabine, in patients with refractory advanced CRC. The initiation of this trial on December 27, 2011 triggered a milestone receivable of $2.6 million.

AEZS-108

§ September 14, 2011: We presented positive final Phase 2 efficacy and safety data for AEZS-108 (zoptarelin doxorubicin) in advanced endometrial cancer at the 17th International Meeting of the European Society of Gynecological Oncology in Milan, Italy. Furthermore, a Parallel Scientific Advice process was granted by and initiated with the United States Food and Drug Administration (the FDA) and the European Medicines Agency (EMA), with the aim of establishing a pivotal program in endometrial cancer.

§ September 26, 2011: We announced positive interim data for the Phase 1 portion of our Phase 1/2 trial with AEZS-108 in castration- and taxane-resistant prostate cancer (CRPC) at the European Society of Medical Oncology (ESMO) meeting in Stockholm, Sweden.

§ October 25, 2011: We announced that the FDA had agreed to allow for the initiation by Alberto J. Montero, M.D., of the Sylvester Comprehensive Cancer Center of an Investigational New Drug (IND) Phase 2 trial in triple-negative breast cancer (TNBC) with AEZS-108.

§ On January 5, 2012: We announced an agreement, dated December 19, 2011, with Ventana Medical Systems, Inc., a member of the Roche Group, to develop a companion diagnostic for the immunohistochemical determination of LHRH-receptor expression, for AEZS-108.

AEZS-120

§ July 20, 2011: We announced that we had reached a key milestone in the non-clinical development of AEZS-120, our oral prostate cancer vaccine candidate. The program encompassed the full development of a Good Manufacturing Practice (GMP) process as well as a safety and toxicology package.

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AEZS-129

§ On March 22, 2011: We presented preclinical results for AEZS-129 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-129 was identified as a potent inhibitor of class I phosphoinositide 3-kinase s (PI3K) lacking activity against mTOR. Data suggest that AEZS-129 is a promising compound for clinical intervention of the PI3K/Akt pathway in human tumors.

AEZS-130

§ July 26, 2011: We announced the completion of the Phase 3 study for AEZS-130 (macimorelin) as a first oral diagnostic test for adult growth hormone deficiency (AGHD).

§ August 30, 2011: We reported favorable top-line results for the completed Phase 3 study for AEZS-130 as a first oral diagnostic test for AGHD. We also announced our intention to meet with the FDA for the future filing of a New Drug Application (NDA). The results showed that AEZS-130 reached its primary endpoint demonstrating >90% area-under-the-curve (AUC) of the Receiver Operating Characteristic (ROC) curve, which determines the level of specificity and sensitivity of the product.

§ November 28, 2011: We announced that the FDA agreed to allow for the initiation by Jose M. Garcia, M.D., Ph.D., of Baylor College of Medicine and Michael E. DeBakey of Veterans Affairs Medical Center of an IND Phase 2A trial to assess the safety and efficacy of repeated doses of AEZS-130 in cancer-cachexia.

§ On March 8, 2012: We announced that the Michael E. DeBakey of Veterans Affairs Medical Center, in Houston, Texas, had initiated a Phase 2A trial with AEZS-130 in patients with cancer-cachexia.

AEZS-131

§ March 22, 2011: We presented preclinical results for AEZS-131 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-131 was established as a small molecular compound that inhibits Erk in the low nanomolar (nM) range and shows an excellent selectivity profile. Results support the evaluation of selective Erk inhibitors as antiproliferative agents either as monotherapy or in combination with inhibitors of the PI3K/Akt pathway.

§ April 5, 2011: We announced that a poster on our highly selective Erk 1/2 inhibitor anticancer compound, AEZS-131, had been presented at the 102nd annual meeting of the AACR. Results showed that AEZS-131 selectively inhibits Erk 1/2 with an IC50 of 4nM, blocks cellular Rsk-1 phosphorylation, modulates downstream cellular substrate activation, arrests tumor cells in G1 and inhibits the growth of multiple human tumor cell lines in the nanomolar range. In *in vivo* pharmacokinetic studies, AEZS-131 showed a favorable PK profile.

§ August 30, 2011: We announced preclinical results demonstrating antitumor activity in different human tumor cell lines for AEZS-131 at the American Chemical Society National Meeting in Denver, Colorado.

§ December 9, 2011: We announced positive preclinical data in TNBC for AEZS-131 at the 34th Annual San Antonio Breast Cancer Symposium. Data showed that AEZS-131 selectively inhibits Erk at low nanomolar concentrations and induces G1 arrest. Accordingly, the cytotoxic effect of AEZS-131 was most pronounced in TNBC cell lines with mutations in the MAPK pathway.

AEZS-132

§ On March 22, 2011: We presented preclinical results for AEZS-132 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. In summary, AEZS-132 is a unique dual kinase inhibitor targeting the PI3K and MAPK pathways, expected to be especially suited to treat tumors with over-activation of both pathways.

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AEZS-137

§ March 24, 2011: We announced that we had been awarded a grant of $1.5 million from the German Ministry of Education and Research to develop, up to the clinical stage, cytotoxic conjugates of the proprietary cytotoxic compound AEZS-137 (disorazol Z) and peptides targeting G-protein coupled receptors, including luteinizing hormone releasing hormone (LHRH) receptors.

§ November 16, 2011: We announced the presentation of a poster on encouraging preclinical data for AEZS-137 at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics, held at the Moscone Center West, in San Francisco. AEZS-137 has been identified as a tubulin binding agent with highly potent antitumor properties. Cell cycle analysis revealed that AEZS-137 arrested cells in the G2/M phase and subsequently induced apoptosis with remarkable potency, as shown by subnanomolar EC50 values.

Corporate Developments

§ On February 22, 2011: We entered into an At-the-Market (ATM) sales agreement with MLV & Co. LLC (formerly McNicoll, Lewis & Vlak LLC) (MLV), under which we were able to sell up to 12.5 million of our common shares through ATM issuances on the NASDAQ Global Market (the NASDAQ) for aggregate gross proceeds not to exceed $19.8 million. On April 29, 2011, we completed the drawdowns from this ATM, bringing the total aggregate gross proceeds to $19.8 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 June 29, 2011: We entered into an additional ATM Sales Agreement (the June ATM Sales Agreement) with MLV, under which we could, at our discretion, from time to time during the 24-month term of the agreement, sell up to a maximum of 9.5 million of our common shares through ATM issuances on NASDAQ for aggregate gross proceeds not to exceed $24.0 million. On December 7, 2011, we completed the drawdowns from this ATM, bringing the total aggregate gross proceeds to $17.7 million.

Subsequent to Year-End

At-The-Market issuance program

§ On January 23, 2012, the Company, pursuant to its existing ATM sales agreement dated June 29, 2011, with MLV, was commencing a new ATM issuance program (January 2012 ATM Sales Agreement) under which it may, at its discretion, from time to time during the term of the sales agreement, sell up to a maximum of 10.4 million of its common shares through ATM issuances on NASDAQ up to an aggregate amount of $16.0 million.

§ From January 23, 2012 through March 15, 2012, the Company issued a total of 3.6 million common shares under the January 2012 ATM Sales Agreement for aggregate gross proceeds of $6.4 million, less cash transaction costs of $0.2 million and previously deferred transaction costs of $56,000.

AEZS-108

§ On February 3, 2012: We reported positive updated results for the Phase 1 portion of our ongoing Phase 1/2 study in CRPC with AEZS-108. Data, presented during a poster session at the American Society of Clinical Oncology Genitourinary Cancers Symposium in San Francisco, showed that AEZS-108 was well tolerated and demonstrated early evidence of antitumor activity in men with CRPC.

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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, 2011. In this MD&A, Aeterna Zentaris , the Company , we , us , our and the Group r 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, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010. Our consolidated financial statements have been prepared in accordance with International Financial Reporting Standards (IFRS) as issued by the International Accounting Standards Board (IASB).

All amounts in this MD&A are presented in US dollars, except for share, option and warrant data, per share and per warrant data and as otherwise noted.

Adoption of International Financial Reporting Standards (IFRS)

In 2008, the Canadian Accounting Standards Board confirmed that all publicly accountable enterprises must adopt IFRS in place of Canadian generally accepted accounting principles (Canadian GAAP) beginning on January 1, 2011 (for entities with a calendar year-end). As such, our consolidated financial statements as at December 31, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010 have been prepared in accordance with IFRS, as issued by the IASB, applicable to the preparation of annual financial statements.

Additionally, our consolidated statement of financial position as at January 1, 2010 and our comparative consolidated financial statements for 2010 have been adjusted to reflect our adoption of IFRS on a retrospective basis, effective on January 1, 2010 (the Transition Date). Consequently, 2010 comparative financial information presented in this MD&A reflects the consistent, retrospective application of IFRS.

These updated figures were reflected in our first quarter report to shareholders.

Our transition to IFRS resulted in a decrease of shareholders equity (deficiency) on the IFRS consolidated statement of financial position as at December 31, 2010 of approximately $30.0 million and an increase in 2010 comprehensive loss of approximately $4.1 million.

Our transition to IFRS also resulted in a decrease in shareholders equity (deficiency) of $20.1 million, a decrease in total assets of $17.7 million and an increase in total liabilities of $2.4 million, as at January 1, 2010. The decrease in shareholders equity (deficiency) was primarily a result of the impairment of Cetrotide® asset, the derecognition of deferred transaction costs and the liability accounting for share purchase warrants.

A complete description of our transition to IFRS, including reconciliations of previously reported Canadian GAAP information, is provided in note 29 to our consolidated financial statements as at December 31, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010.

In this MD&A, we also include comparative financial information derived from our consolidated financial statements as at December 31, 2010 and 2009 and for each of the years then ended which, prior to January 1, 2011, were prepared in accordance with Canadian GAAP.

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 , assuming , believe , could , expect , foresee , goal , guidance , intend , may , objective , o should , strive , target and will .

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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 European Medicines Agency (EMA), the Therapeutic Products Directorate of Health Canada or any other organization responsible for enforcing regulations in the pharmaceutical industry.

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.

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 furnish 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 specializing in oncology and endocrine therapy. Our pipeline encompasses compounds at all stages of development, from drug discovery through to marketed products. The highest development priorities in oncology are the completion of Phase 3 trials with perifosine in colorectal cancer and in multiple myeloma, as well as the further advancement of AEZS-108, for which we have successfully completed a Phase 2 trial in advanced endometrial and advanced ovarian cancer. We are planning for the initiation of a pivotal program with AEZS-108 in endometrial cancer and also a Phase 2 trial in triple-negative breast cancer. AEZS-108 is also in development in other cancer indications, including castration- and taxane-resistant prostate cancer, as well as refractory bladder cancer.

Our pipeline also encompasses other earlier-stage programs in oncology. AEZS-112, an oral anticancer agent which involves three mechanisms of action (tubulin, topoisomerase II and angiogenesis inhibition) has completed a Phase 1 trial in advanced solid tumors and lymphoma. Additionally, several novel targeted potential anti-cancer candidates such as AEZS-120, a live recombinant oral tumor vaccine candidate, as well as our PI3K/Erk inhibitors AEZS-129, AEZS-131, AEZS-132 and their respective follow-up compounds are currently in preclinical development.

Our lead program in endocrinology, a Phase 3 trial under a Special Protocol Assessment (SPA) obtained from the FDA with AEZS-130 as an oral diagnostic test for AGHD, has been completed with positive results. We are planning to file an NDA for the registration of AEZS-130 in the United States, subject to a successful pre-NDA meeting with the FDA. Furthermore, AEZS-130 is in a Phase 2A trial for the treatment of cancer-cachexia.

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Key Developments for the Year Ended December 31, 2011

Drug Development

Status of our drug pipeline as at March 27, 2012

Discovery	**Preclinical**	**Phase 1**	**Phase 2**	**Phase 3**	**Commercial**
~120,000 compound library	AEZS-120 Prostate cancer vaccine (oncology)	AEZS-112 (oncology)	Perifosine § Multiple cancers	Perifosine § Refractory advanced colorectal cancer	Cetrotide® (*in vitro* fertilization)
	AEZS-129, 131, 132 and 136; Erk/PI3K inhibitors (oncology)		§ AEZS-108 § Endometrial cancer	§ Multiple myeloma	
	AEZS-137 (Disorazol Z) (oncology)		§ Triple-negative breast cancer § Ovarian cancer § Castration- and taxane-resistant prostate cancer § Refractory bladder cancer	AEZS-130 § Diagnostic in adult growth hormone deficiency (endocrinology)	
	AEZS-125 (LHRH-Disorazol Z) (oncology)		Ozarelix § Prostate cancer		
			AEZS-130 § Therapeutic in cancer-cachexia		
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

Hikma	**Hikma**
Middle East/North Africa	Middle East/North Africa

Ozarelix:

Spectrum

World (ex-Japan for oncology indications, ex-Korea and ex-other Asian countries for BPH indication)

Handok

Korea and other Asian countries for BPH indication

Nippon Kayaku

Japan for oncology indications

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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 colorectal cancer and multiple myeloma, as well as in Phase 2 studies for the treatment of other cancers, and we believe is the most advanced anti-cancer compound of its class in late-stage development. The FDA has granted perifosine orphan-drug designation in multiple myeloma and in neuroblastoma and Fast Track designations in both refractory advanced colorectal cancer and multiple myeloma. Additionally, an agreement was reached with the FDA to conduct the Phase 3 trials in both of these indications under a SPA. Perifosine has also been granted Orphan Medicinal Product designation from the EMA in multiple myeloma, and based on discussions with the EMA, we believe the ongoing Phase 3 trials of perifosine in the colorectal cancer and multiple myeloma programs would be sufficient for registration in Europe for these indications, assuming positive data from the Phase 3 trials. Perifosine rights have been licensed to Keryx Biopharmaceuticals, Inc. (Keryx) for North America, to Handok Pharmaceuticals Co. Ltd. for Korea, to Yakult for Japan and to Hikma for the MENA region.

On March 8, 2011, we 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, non-refundable gross upfront payment to us of 6.0 million (approximately $8.4 million). Also per the agreement, we are entitled to receive in addition, up to a total of 44.0 million (approximately $57.1 million) upon achieving certain pre-established milestones, including the occurrence of certain clinical and regulatory events in Japan. As at December 31, 2011, following the achievement of a milestone, we have revenue and trade receivables recorded for an additional amount of 2.0 million (approximately $2.6 million) in connection with the agreement. Furthermore, we will be entitled to receive double-digit royalties on future net sales of perifosine in the Japanese market.

On November 23, 2011, we entered into an agreement with Hikma for the development, and commercialization of perifosine in all oncology uses. Under the terms of this agreement, Hikma made an initial, non-refundable gross upfront payment to us of $0.2 million. Also per the agreement, we are entitled to receive up to a total of $1.8 million upon achieving certain pre-established milestones, including the occurrence of certain regulatory events in certain countries in the MENA region. Furthermore, we will be entitled to receive double-digit royalties on future net sales of perifosine in the MENA market.

We have substantial continuing involvement in the aforementioned arrangements, including the use of commercially reasonable efforts to develop, apply for and obtain relevant regulatory approval for, manufacture and commercialize perifosine outside Japan and the MENA region, which will facilitate the ultimate commercialization process within Japan and the MENA region. Additionally, we are contractually obligated to ensure a stable supply of perifosine and related trial products to Yakult and Hikma throughout the ongoing development process and will maintain relevant patent rights over the term of the arrangements. Lastly, per the terms of the aforementioned agreements, we have agreed to supply perifosine, on a cost-plus basis, to Yakult and Hikma following regulatory approvals.

We have deferred the non-refundable license fees and we are amortizing the related payments as revenue on a straight-line basis over the duration of our continuing involvement, which approximates the estimated life cycle of the product that is currently under development and the expected period over which Yakult and Hikma will derive value from the use of, and access to, the underlying licenses.

In determining the period over which license revenues are to be recognized, and in addition to due consideration of our continuing involvement, as discussed above, we considered the remaining expected life of applicable patents as the most reasonable basis for estimating the underlying product s life cycle. However, we may adjust the amortization period based on appropriate facts and circumstances not yet known, including, but not limited to, the extension(s) of patents, the granting of new patents, the economic lives of competing products and other events that would significantly change the duration of our continuing involvement and performance obligations or benefits expected to be derived by Yakult and Hikma.

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Future milestones will be recognized as revenue individually and in full upon the actual achievement of the related milestone, given the substantive nature of each milestone. Lastly, upon initial commercialization and sale of the developed product, we will recognize royalty revenues as earned, based on the contractual percentage applied to the actual net sales achieved by Yakult and Hikma, as per the aforementioned agreements.

We were required to remit to the Japanese tax authorities $0.8 million of the gross proceeds received from Yakult and will be required to remit approximately 0.2 million (approximately $0.26 million) when the milestone receivable will be paid by Yakult. These amounts, which were withheld at source, were recognized as income tax expense in our consolidated statement of comprehensive loss.

On April 4, 2011, we announced that two posters on perifosine were presented at the 102nd annual meeting of the AACR at the Orange County Convention Center in Orlando, Florida. Perifosine demonstrated antitumor activity in several gastric cancer cell lines. Furthermore, perifosine enhanced the antitumor activity of 5-FU in parts of the cell lines, including 5-FU resistant cell lines. 5-FU is the active metabolite of the prodrug Xeloda, which is approved for the treatment of advanced gastric cancer in many countries including Japan.

Perifosine also markedly enhanced the antitumor activity of the cellular TRAIL based treatment and was able to overcome TRAIL resistance both *in vitro* and *in vivo*. The results are in line with other studies demonstrating the synergistic effects of perifosine with cytotoxic drugs, including bortezomib and 5-FU.

On July 12, 2011, we announced that the European Patent Office had granted a patent for the use of alkylphosphocholines, more specifically perifosine (octadecyl 1,1-dimethylpiperidino-4-yl phosphate), in the preparation of a medicament for the treatment of benign and malignant tumors, prior to and/or during the treatment with approved antitumor anti-metabolites such as 5-FU and capecitabine. Filed on July 29, 2003, the patent (EP #1 545 553), entitled *Use of Alkyl Phosphocholines in Combination with Anti-Tumour Medicaments*, became effective as of July 13, 2011 and will expire on July 28, 2023.

On July 27, 2011, we announced completion of patient recruitment for the ongoing Phase 3 trial with perifosine in refractory advanced colorectal cancer, involving over 465 patients from 65 sites in the United States. This Phase 3 X-PECT (Xeloda® + Perifosine Evaluation in Colorectal Cancer Treatment) trial is a randomized (1:1), double-blind trial comparing the efficacy and safety of perifosine + capecitabine vs. placebo + capecitabine. The primary endpoint is overall survival, with secondary endpoints including overall response rate (complete + partial responses), progression-free survival and safety. Approximately 360 events of death will trigger the unblinding of the study.

On August 31, 2011, we announced that the DSMB for the Phase 3 X-PECT study of perifosine in patients with refractory advanced colorectal cancer had completed a pre-specified interim analysis for safety and futility. The DSMB recommended that the Phase 3 study continue to completion, as planned.

On October 5, 2011, we announced that a manuscript, entitled *Randomized Placebo-Controlled Phase 2 Trial of Perifosine Plus Capecitabine as Second- or Third-Line Therapy in Patients with Metastatic Colorectal Cancer,* had been published in the October 3, 2011 online edition of the *Journal of Clinical Onclogy* (JCO), in which Phase 2 activity of perifosine in the treatment of patients with refractory advanced CRC was reported. The publication highlighted the efficacy and safety data on the 38 CRC patients participating in this Phase 2, randomized, multicenter study, comparing perifosine plus capecitabine (P-CAP) to placebo plus capecitabine. Based on the data, in which the combination of P-CAP demonstrated statistical significance with respect to median overall survival and median time to tumor progression, the investigators concluded that the P-CAP combination showed promising clinical activity compared to single-agent capecitabine, and that the difference in clinical outcome seen with the addition of perifosine was impressive.

Efficacy data from this study had been previously presented in June 2010 at the 46th Annual Meeting of the American Society of Clinical Oncology.

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On October 13, 2011, we announced that the manuscript, entitled *Perifosine Plus Bortezomib and Dexamethasone in patients with Relapsed/Refractory Multiple Myeloma Previously Treated with Bortezomib: Results of a Multicenter Phase 1/2 Trial,* had been published in the October 10, 2011 online edition of the JCO, in which Phase 1/2 combination activity of perifosine in the treatment of advanced multiple myeloma patients was reported. Results showed that the Overall Response Rate was 65% for bortezomib-relapsed patients and 32% for bortezomib-refractory patients. Median PFS was 6.4 months, with a median PFS of 8.8 months in the bortezomib-relapsed population. Therapy was generally well-tolerated, and toxicities, including gastrointestinal side-effects and fatigue, proved manageable. No treatment-related mortality was seen. The investigators concluded the data reported for both safety and efficacy in this patient population were encouraging for the continued study of perifosine. Data from this study had been previously presented at the 2009 ASH conference.

On December 12, 2011, we reported encouraging preclinical data for perifosine in HL. *In vitro* data from HL cell lines showed that perifosine combined with sorafenib induced increased apoptosis, while *in vivo* data for the same combination treatment for HL significantly increased survival in mice. Data were presented during the ASH Annual Meeting and Exposition, in San Diego, California.

The study was conducted to investigate, *in vitro* and *in vivo*, the activity and mechanism(s) of action of perifosine in combination with sorafenib by using three HL cell lines (HD-MyZ, L-540, HDLM-2). In the *in vitro* experiments perifosine/sorafenib treatment resulted in synergistic cell growth inhibition and cell death induction in HD-MyZ and L-540 cell lines, but not in the HDLM-2 cell line. Cell cycle arrest, down-modulating of the MAPK and PI3K/Akt pathways as well as caspase-independent cell death was observed, which was associated with severe mitochondrial dysfunction. Further, expression of genes involved in amino acid metabolism, cell cycle, DNA replication and cell death was shown to be modulated. In addition, overexpression of the tribbles homologue 3 [TRIB3] was observed.

In vivo, perifosine/sorafenib treatment significantly increased survival in the HD-MyZ model (45 vs. 81 days, as compared to controls), with 25% tumor-free mice at the end of the 200-day observation period. In the L-540 model, subcutaneous tumor volume was also reduced as compared to controls (by 42%), perifosine (by 35%) or sorafenib (by 46%) alone. In HD-MyZ and L-540 but not HDLM-2, the combined treatment induced an increase in tumor necrosis (2- to 8-fold, P £.0001) and in tumor apoptosis (2- to 2.5-fold, P £.0001). In 2 of 3 HL cell lines, perifosine/sorafenib combination treatment induced potent antitumor effects. Striking increase of mitochondrial injury and apoptosis, and marked reduction of cell viability was observed in the *in vitro* experiments. *In vivo* combination treatment increased survival and inhibited tumor growth.

On December 13, 2011, we reported encouraging clinical data for an ongoing Phase 2 clinical study in patients with refractory/relapsed HL. Preliminary response data showed that perifosine combined with sorafenib significantly increased median PFS in refractory/relapsed HL patients with high phosphorylation levels of Erk and Akt, as compared to patients with low baseline phosphorylation levels of Erk and Akt. Data were presented during the ASH Annual Meeting and Exposition, in San Diego, California.

The study evaluated phosphorylation levels of Erk (pErk) and Akt (pAkt) in circulating lymphocytes from patients enrolled in two consecutive Phase 2 trials evaluating activity and safety of sorafenib as a single agent or in combination with perifosine in relapsed/refractory HL patients. Four patients were treated with sorafenib alone at a dose level of 400mg BID and twenty-one patients received a 4-week treatment with perifosine alone at a dose level of 50mg BID, followed by a perifosine/sorafenib combination therapy with 50mg BID and 400mg BID, respectively. Circulating lymphocytes were evaluated for their phosphorylation levels of Erk and Akt, in order to assess predictive value of the phosphokinase levels for therapy responses.

Clinical response data showed that baseline pErk and pAkt levels were significantly higher in responsive patients, as compared to unresponsive patients. The pErk and pAkt levels measured after 60 days of therapy with perifosine combined with sorafenib were significantly reduced in responsive patients. The median baseline value of pErk and pAkt efficiently discriminated responsive and unresponsive patients which was associated with a significantly improved median PFS for patients with baseline pErk ³43% and/or pAkt >23%. Based on these data, the correlation of baseline pErk and pAkt levels with objective responses and time to tumor progression will need to be validated in prospective studies.

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On January 3, 2012, we announced that our Japanese partner, Yakult, had initiated a Phase 1/2 trial in Japan to assess the safety and efficacy of perifosine in combination with chemotherapeutic agent, capecitabine, in patients with refractory advanced CRC. The primary endpoint of the Phase 1 portion of the trial is the safety profile of perifosine in combination with capecitabine. The primary endpoint of the Phase 2 portion is efficacy (Disease Control Rate). This trial is sponsored by Yakult and its initiation on December 27, 2011 triggered a milestone payment which is payable in the first quarter of 2012 of approximately 2 million from Yakult to Aeterna Zentaris under the agreement signed in March 2011 for perifosine in Japan described above.

AEZS-108

On September 14, 2011, we presented positive final Phase 2 efficacy and safety data for AEZS-108 (zoptarelin doxorubicin) in advanced endometrial cancer at the European Society of Gynecological Oncology in Milan, Italy. Data showed that AEZS-108, administered as a single agent at a dosage of 267 mg/m^2 every 3 weeks was active, well tolerated and that overall survival is similar to that reported for modern triple combination chemotherapy, but was achieved with lower toxicity. The primary endpoint was the response rate as defined by the Response Evaluation Criteria in Solid Tumors (RECIST). Secondary endpoints included safety, time-to-progression (TTP) and overall survival (OS).

In all, of 43 patients treated with AEZS-108, 39 were evaluable for efficacy. Efficacy confirmed by independent response review included 2 complete responses (CR), 10 partial responses (PR), and 17 patients with disease stabilization (SD). Based on those data, the estimated Overall Response Rate (ORR = CR+PR) was 30.8% and the Clinical Benefit Rate (CBR) (CBR = CR+PR+SD) was 74.4%. Responses in patients previously treated with chemotherapy included 1 CR, 1 PR and 2 SDs in 8 of the patients with prior use of platinum/taxane regimens. Median TTP and OS were 7 months and 13.7 months, respectively.

Overall, tolerability of AEZS-108 was good and commonly allowed retreatment as scheduled. Severe (Grade 3 or 4) toxicity was mainly restricted to rapidly reversible leukopenia and neutropenia, associated with fever in only 1 patient who had been treated only 3 weeks after a surgery. Good tolerability of AEZS-108 was also reflected by a low rate of severe non hematological possibly drug-related adverse events which included single cases each of nausea, diarrhea, fatigue, general health deterioration, creatinine elevation, and blood potassium decrease. No cardiac toxicity was reported.

On September 26, 2011, we announced positive interim data for the Phase 1 portion of our Phase 1/2 trial with AEZS-108 in castration- and taxane-resistant prostate cancer at the ESMO meeting in Stockholm, Sweden. This is a single arm study with a Phase 1 lead-in to a Phase 2 clinical trial. The primary endpoint of the Phase 1 portion is safety. The primary objective of the Phase 2 portion is to evaluate the clinical benefit of AEZS-108 for these patients. Data showed that AEZS-108 was well tolerated at all dose levels and early evidence of antitumor activity was observed even at low dose level. The Phase 2 extension is planned after completion of the toxicity assessment in the final dose level of the Phase 1 portion of the study. Furthermore, data from correlative studies demonstrated for the first time the internalization of AEZS-108 in circulating tumor cells of patients.

On October 25, 2011, we announced that the FDA had completed the review of the IND application filed by Alberto J. Montero, M.D., Assistant Professor, Department of Medicine, Division of Hematology/Oncology, Sylvester Comprehensive Cancer Center at the University of Miami Miller School of Medicine and concluded that Dr. Montero may proceed with the initiation of a randomized Phase 2 trial in chemotherapy refractory triple-negative (ER/PR/HER2-negative) LHRH receptor-positive metastatic breast cancer with AEZS-108. This will be an open-label, randomized, two-arm, multicenter Phase 2 study involving up to 74 patients. The primary study endpoint will be PFS. Secondary endpoints will also include overall response rate, and overall survival. The study will also evaluate AEZS-108 s toxicity profile and patients quality of life relative to conventional cytotoxic chemotherapy used in the control arm of this study.

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On January 5, 2012, we announced that we had entered into a collaboration agreement with Ventana Medical Systems, Inc., a member of the Roche Group, to develop a companion diagnostic for the immunohistochemical determination of LHRH-receptor expression, for AEZS-108. In humans, LHRH receptors are expressed in a significant proportion of endometrial, ovarian, breast, bladder, prostate and pancreatic tumors. AEZS-108 specifically targets LHRH receptors and could, therefore, prove to be more efficient in treating patients with these types of LHRH-receptor positive cancers.

AEZS-120

On July 20, 2011, we announced that we had successfully reached a key milestone in the non-clinical development of our live recombinant prostate cancer vaccine candidate, AEZS-120, for oral administration. The program encompassed the full development of a GMP process, including GMP production and quality testing of a clinical batch, as well as a non-clinical safety and toxicology package, as previously agreed with the regulatory authorities. Subject to a positive review by the regulatory authorities, we aim to start Phase 1 clinical development in 2012. AEZS-120 has been developed through a research collaboration with the Department of Medical Radiation Biology and Cell Research, and the Department of Microbiology of the University of Würzburg, Germany. The collaboration was funded with a total of $890,000 for the Company and $870,000 for the university partner by the German Ministry of Education and Research (BMBF) for a period of three years. As part of the collaboration, a melanoma vaccine based on the recombinant expression of a modified B-Raf protein has been generated.

AEZS-129

On March 22, 2011, we presented preclinical results for AEZS-129 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-129 was identified as a potent inhibitor of class I PI3Ks lacking activity against mTOR. Lack of mTOR activity is considered to potentially lead to a better safety profile. In biochemical and cellular assays, AEZS-129 demonstrated favorable properties in early *in vitro* ADMET screening, including microsomal stability, plasma stability and screening against a safety profile composed of receptors, enzymes and cardiac ion-channels. *In vitro*, the compound was shown to be a selective ATP-competitive inhibitor of PI3K with a broad antiproliferative activity against a broad panel of tumor cell lines. *In vivo*, AEZS-129 showed excellent plasma exposure and significant tumor growth inhibition in several tumor xenografts models, including A-549 (lung), HCT-116 (colon) and Hec1B (endometrium). These data suggest that AEZS-129 is a promising compound for clinical intervention of the PI3K/Akt pathway in human tumors.

AEZS-130

On July 26, 2011, we announced the completion of the Phase 3 study on AEZS-130 as a first oral diagnostic test for AGHD and the decision to meet with the FDA for the future filing of an NDA for the registration of AEZS-130 in the United States.

On August 30, 2011, we announced favorable top-line results of the completed Phase 3 study with AEZS-130 as the first oral diagnostic test for AGHD. The results showed that AEZS-130 reached its primary endpoint demonstrating >90% AUC of the ROC curve, which determines the level of specificity and sensitivity of the product.

The first part of the study, conducted by our former partner, Ardana, was a two-way crossover study involving 42 patients with confirmed AGHD or multiple pituitary hormone deficiencies and a low insulin-like growth factor-I. A 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 (Ar-Gine®, Pfizer) administered intravenously over 30 minutes; the other regimen was a dose of 0.5 mg/kg body weight of AEZS-130 given in an oral solution of 0.5 mg/ml. As a result of the SPA reached with the FDA in order to complete the trial, the second part of the study contained the following revisions/additions to the first protocol:

§ An additional 30 normal control subjects were to be enrolled to match the AGHD patients from the original cohort;

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§ Further, an additional 20 subjects were to be enrolled: 10 AGHD patients and 10 matched normal control subjects;

§ The above brought the database to ~100 subjects;

§ All subjects received a dose of 0.5 mg/kg body weight of AEZS-130;

§ As a secondary endpoint, the protocol required that at least 8 of the 10 newly enrolled AGHD patients be correctly classified by a pre-specified peak GH threshold level.

The parameters of the study were achieved as agreed to with the FDA under our SPA. Importantly, the primary efficacy parameters showed that the study achieved both specificity and sensitivity at a level of 90% or greater. In addition, 8 of the 10 newly enrolled AGHD patients were correctly classified by a pre-specified peak GH threshold level. The use of AEZS-130 was shown to be safe and well tolerated overall throughout the completion of this trial.

The Company is planning for the filing of an NDA for the registration of AEZS-130 in the United States as a diagnostic test for AGHD.

On November 28, 2011, we announced that the FDA had completed the review of an IND application filed by Jose M. Garcia, M.D., Ph.D., Assistant Professor, Division of Diabetes Endocrinology and Metabolism, Departments of Medicine and Molecular and Cell Biology, Baylor College of Medicine and the Michael E. DeBakey Veterans Affairs Medical Center, in Houston Texas and concluded that Dr.Garcia may proceed with the initiation of a Phase 2A trial to assess the safety and efficacy of repeated oral administration of AEZS-130 at different doses daily for 1 week in view of developing a treatment for cancer-cachexia. Cachexia, which is characterized by diminished appetite and food intake in cancer patients, is defined as an involuntary weight loss of at least 5% of the pre-illness body weight over the previous six months.

On March 8, 2012, we announced that the Michael E. DeBakey Veterans Affairs Medical Center, in Houston, Texas, initiated a Phase 2A trial assessing the safety and efficacy of repeated doses of AEZS-130 in patients with cancer-cachexia. The study is conducted under a CRADA between the Michael E. DeBakey Veterans Affairs Medical Center, which is funding the study, and us. This is a double-blind, randomized, placebo-controlled Phase 2A trial to test the effects of different doses of the ghrelin agonist, AEZS-130, in 18 to 26 patients with cancer-cachexia. AEZS-130 will be provided by us. The study will involve 3 sequential groups receiving differing doses of AEZS-130. Each dose group will have 6 patients who will receive AEZS-130 and 2-4 patients who will receive a placebo. After analysis of safety and efficacy at each dose level vs. placebo, a decision will be taken either to decrease or increase the dose. If there are major safety issues, the study will be stopped. For this study, adequate efficacy will be defined as a ³0.8 kg of body weight gain or a ³50 ng/mL increase in plasma IGF-1 levels. The primary objective of the study is to evaluate the safety and efficacy of repeated oral administration of AEZS-130 at different doses daily for 1 week in view of developing a treatment for cachexia. The following parameters will be recorded to assess efficacy during the study: change of body weight, change of IGF-1 plasma levels, and change of quality of life score (Anderson Symptom Assessment Scale, FACIT-F). Other secondary objectives will include food intake, and changes in the following: appetite, muscle strength, energy expenditure, reward from food and functional brain connectivity.

AEZS-131

On March 22, 2011, we presented preclinical results for AEZS-131 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-131 was established as a small molecular compound that inhibits Erk in the low nanomolar range and shows an excellent selectivity profile. Further characterization experiments revealed an ATP-competitive mode of action and the potent inhibition of the cellular downstream target Rsk1 in tumor cells. The frontrunner, AEZS-131, produces cell cycle arrest in G1 and results in growth inhibition of cancer cells. Furthermore, the potential of combination therapy of AEZS-131 with inhibitors of the PI3K pathway was

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addressed and the analysis of combination effects on tumor cell proliferation has been presented. These results support the evaluation of selective Erk inhibitors as antiproliferative agents either as monotherapy or in combination with inhibitors of the PI3K/Akt pathway.

On April 5, 2011, we announced that a poster on our highly selective Erk 1/2 inhibitor anticancer compound, AEZS-131, had been presented at the 102nd annual meeting of the AACR. Results showed that AEZS-131 selectively inhibits Erk 1/2 with an IC50 of 4nM, blocks cellular Rsk-1 phosphorylation, modulates downstream cellular substrate activation, arrests tumor cells in G1 and inhibits the growth of multiple human tumor cell lines in the nanomolar range. In *in vivo* pharmacokinetic studies, AEZS-131 showed a favorable PK profile. Antitumor activity was studied in *in vivo* mouse xenograft experiments utilizing the HCT 116 colon cancer model. AEZS-131 significantly inhibited tumor growth and was well tolerated at daily doses up to 120 mg/kg. Focus on inhibition of downstream kinase Erk 1/2 activity as a therapeutic target may be attractive because the pharmacologic inhibition of Erk 1/2 reverses Ras and Raf activation in cells which also demonstrate resistance to common Raf inhibitors, such as PLX-4720/4032.

On August 30, 2011, we announced that a poster on our novel orally active anticancer Erk inhibitor, which includes AEZS-131, was presented at the American Chemical Society National Meeting in Denver, Colorado. The *in vitro* antiproliferative efficacy proved to be excellent in diverse human tumor cell lines. GI50 values in the low nanomolar range were obtained. *In vivo* antitumor activity was studied in a mouse xenograft experiment utilizing the human HCT-116 colon cancer model. Up to 74% inhibition of tumor growth was achieved with daily oral doses of 30 -120 mg/kg. Our medicinal chemistry programs are supported by X-ray crystallography and modeling towards the optimization of pyrido[2,3-b]pyrazines as novel series of kinase inhibitors.

On December 9, 2011, we announced positive preclinical data in TNBC for AEZS-131 at the 34th Annual San Antonio Breast Cancer Symposium. AEZS-131 was tested to check for selectivity, inhibition of Rsk-phosphorylation (cellular substrate of Erk), mode of action and cleavage of PARP. Cytotoxic efficacy was evaluated in a selection of TNBC cell lines, with or without mutations in the MAPK signal transduction pathway, by MTT assay. The study showed that AEZS-131 selectively inhibited ERK with an IC50<4nM. Phosphorylation of Rsk-1 was inhibited with an IC50 of 158 nM. AEZS-131 induced cell cycle arrest in G1 dose-dependently and cleavage of PARP. EC50 values were below 1µM for cell lines with mutations in the MAPK pathway. TNBC cell lines without mutations in the MAPK pathway were less responsive.

AEZS-132

On March 22, 2011, we presented preclinical results for AEZS-132 at the Informa Life Sciences Protein Kinases Congress in Berlin, Germany. AEZS-132 is a unique dual inhibitor of PI3K and Erk in the nanomolar range and exerts high selectivity against other serine threonine and tyrosine kinases. AEZS-132 is also an ATP-competitive inhibitor, with a broad antiproliferative profile *in vitro*, a favorable safety profile and beneficial ADME properties. *In vivo* pharmacokinetic experiments showed plasma profiles expected to result in positive antitumor efficacy, and led to significant antitumor activity in mouse xenograft models, including HCT-116 (colon), A-549 (lung), and Hec 1B (endometrium). Cellular inhibition of the downstream targets p-Akt and p-Rsk was confirmed within the *in vivo* tumor studies. In summary, AEZS-132 is a unique dual kinase inhibitor targeting the PI3K and MAPK pathways, expected to be especially suited to treat tumors with over-activation of both pathways.

AEZS-137

On March 24, 2011, we announced that we had been awarded a $1.5 million grant from the German Ministry of Education and Research to develop, up to the clinical stage, cytotoxic conjugates of the proprietary cytotoxic compound AEZS-137 (disorazol Z) and peptides targeting G-protein coupled receptors, including the LHRH receptors. The compounds being developed will combine the targeting principle successfully employed in Phase 2 with AEZS-108 (doxorubicin and LHRH receptor targeting agent) with the novel cytotoxic disorazol Z. Furthermore, diagnostic tools systematically assessing the receptor expression in tumor specimens will be developed to allow the future selection of patients and tumor types with the highest chance of benefitting from this personalized medicine approach. The grant will be payable as a partial reimbursement of qualifying

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expenditures over a three-year period. The qualified project will be performed with Morphisto GmbH and the Helmholtz Institute in Saarbrücken, Germany, which will receive additional funding of approximately $0.7 million. Researchers from the department of Gynecology and Obstetrics at both the University of Göttingen and Würzburg, Germany, will also be part of the collaboration.

On November 16, 2011, we announced encouraging preclinical data for AEZS-137 from a poster presentation at the AACR-NCI-EORTC International Conference on Molecular Targets and Cancer Therapeutics, in San Francisco. The poster showed that AEZS-137 possesses outstanding cytotoxicity in a highly diverse panel of 60 different tumor cell lines, and also underlined the identification of important aspects of this novel natural compound s mechanism of action. AEZS-137 has been identified as a tubulin binding agent with highly potent antitumor properties. Cell cycle analysis revealed that AEZS-137 arrested cells in the G2/M phase and subsequently induced apoptosis with remarkable potency, as shown by subnanomolar EC50 values.

Corporate Developments

At-the-Market Sales Agreement

On February 22, 2011, we entered into an ATM sales agreement with MLV, under which we were able to sell up to 12.5 million of our common shares through ATM issuances on NASDAQ for aggregate gross proceeds not to exceed $19.8 million. The ATM sales agreement provided that common shares were to be sold at market prices prevailing at the time of sale, and, as a result, prices may have varied. From February 20, 2011 and up to April 29, 2011, we issued a total of 10.0 million of our common shares for aggregate gross proceeds of $19.8 million, less cash transaction costs of $0.6 million and previously deferred transaction costs of $0.2 million.

On June 29, 2011, we entered into a second ATM, the June ATM Sales Agreement with MLV, under which we could, at our discretion, from time to time during the 24-month term of the agreement, sell up to 9.5 million of our common shares through ATM issuances on NASDAQ for aggregate gross proceeds not to exceed $24.0 million. The June ATM Sales Agreement, similar to the February ATM Sales Agreement, provided that common shares were to be sold at market prices prevailing at the time of sale, and, as a result, prices may have varied. From June 29, 2011 and up to December 7, 2011, under the June ATM agreement, we issued a total of 9.5 million of our common shares for aggregate gross proceeds of $17.7 million, less cash transactions costs of $0.6 million and previously deferred transaction costs of $0.2 million.

Gross proceeds raised under both ATM sales agreements totalled $37.5 million for the year ended December 31, 2011.

Cowen Royalty Sales Milestone Payment

On February 28, 2011, we announced that we had received a net sales milestone of $2.5 million from Cowen Healthcare Royalty Partners, L.P. (Cowen). This milestone was payable pursuant to the previously announced sale to Cowen of Aeterna Zentaris rights to royalties on future net sales of Cetrotide®, covered by the Company s license agreement with Merck Serono, and was contingent on 2010 net sales of Cetrotide® reaching a specified level.

Subsequent to Year-End

At-The-Market Issuance Program

On January 23, 2012, the Company, pursuant to its existing ATM sales agreement dated June 29, 2011, with MLV, was commencing a new ATM issuance program (January 2012 ATM Sales Agreement) under which it may, at its discretion, from time to time during the term of the sales agreement, sell up to a maximum of 10.4 million of its common shares through ATM issuances on NASDAQ up to an aggregate amount of $16.0 million.

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From January 23, 2012 through March 15, 2012, the Company issued a total of 3.6 million common shares under the January 2012 ATM Sales Agreement for aggregate gross proceeds of $6.4 million, less cash transaction costs of $0.2 million and previously deferred transaction costs of $56,000.

AEZS-108

On February 3, 2012, we reported positive updated results for the Phase 1 portion of our ongoing Phase 1/2 study in CRPC with AEZS-108. This is a single-arm study with a Phase 1 lead-in portion (testing 3 dose levels) to a Phase 2 clinical trial. The primary endpoint of the Phase 1 portion is safety. The primary objective of the Phase 2 portion is to evaluate the clinical benefit of AEZS-108 for these patients. Data were presented by Jacek Pinski, M.D., Ph.D., Associate Professor of Medicine at the Norris Comprehensive Cancer Center of the University of Southern California, during a poster session at the American Society of Clinical Oncology Genitourinary Cancers Symposium in San Francisco. The trial is being supported by a three-year $1.6 million grant from the National Institutes of Health to Dr. Pinski.

The results were based on 13 patients who have been treated on 3 dose levels: 3 at 160 mg/m^2, 3 at 210 mg/m^2, and 7 at 267 mg/m^2. Overall, AEZS-108 has been well tolerated among this group of heavily pre-treated older patients. To date, there have been 2 dose limiting toxicities; both were cases of asymptomatic Grade 4 neutropenia at the 267 mg/m^2 dose level and both patients fully recovered. The Grade 3 and 4 toxicities were primarily hematologic. There has been minimal non-hematologic toxicity, most frequently fatigue and alopecia.

Despite the low doses of AEZS-108 in the first cohorts, there was some evidence of antitumor activity. One patient received 8 cycles (at 210 mg/m^2) due to continued benefit. Among the 5 evaluable patients with measurable disease, 4 achieved stable disease. At the time of submission of the abstract, a decrease in PSA was noted in 6 patients. Six of 13 (46%) treated patients have received at least 5 cycles of therapy with no evidence of disease progression at 12 weeks. Correlative studies on circulating tumor cells (CTC) have demonstrated the uptake of AEZS-108 into the targeted tumor. After completion of 3 additional patients at the 210 mg/m^2 dose level, the study is expected to be extended into the Phase 2 portion.

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Consolidated Statements of Comprehensive Loss Information

(in thousands, except share and per share data)	Years ended December 31, **2011** **$**	2010 $	2009* $
Revenues			
Sales and royalties	**31,306**	24,857	20,957
License fees and other	**4,747**	2,846	42,280
	36,053	27,703	63,237
Operating expenses			
Cost of sales	**27,560**	18,700	16,501
Research and development costs, net of refundable tax credits and grants	**24,517**	21,257	43,814
Selling, general and administrative expenses	**16,170**	12,552	16,040
Depreciation and amortization*	*****	*	10,840
	68,247	52,509	87,195
Loss from operations	**(32,194)**	(24,806)	(23,958)
Finance income	**6,239**	1,800	349
Finance costs	**(8)**	(5,445)	(1,115)
Net finance income (costs)	**6,231**	(3,645)	(766)
Loss before income taxes	**(25,963)**	(28,451)	(24,724)
Income tax expense	**(1,104)**	-	-
Net loss	**(27,067)**	(28,451)	(24,724)
Other comprehensive (loss) income:			
Foreign currency translation adjustments	**(789)**	1,001	(1,335)
Actuarial gain (loss) on defined benefit plans	**(1,335)**	191	-
Comprehensive loss	**(29,191)**	(27,259)	(26,059)
Net loss per share			
Basic and diluted	**(0.29)**	(0.38)	(0.43)
Weighted average number of shares outstanding			
Basic and diluted	**94,507,988**	75,659,410	56,864,484

* We adopted IFRS in 2011 with a transition date of January 1, 2010. The selected financial information for the years ended December 31, 2009, 2008 and 2007 is derived from financial statements that were presented in accordance with Canadian GAAP and has not been restated in accordance with IFRS. Consequently, the selected financial information for the years ended December 31, 2009, 2008 and 2007 may not be comparable with the corresponding selected financial information for the years ended December 31, 2011 and 2010. Please refer to Critical Accounting Policies, Estimates and Judgments for the policy differences between Canadian GAAP and IFRS.

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Revenues

Revenues are derived primarily from sales and royalties as well as from license fees. Sales are derived from Cetrotide® (cetrorelix acetate solution for injection), marketed globally (ex-Japan) by ARES Trading S.A. (Merck Serono) for reproductive health assistance for *in vitro* fertilization, as well as from active pharmaceutical ingredients. Royalties are derived indirectly from Merck Serono s net sales of Cetrotide® and represent the periodic amortization, under the units-of-revenue method, of the proceeds received in connection with the 2008 sale to Cowen Healthcare Royalty Partners L.P. of the underlying future royalty stream.

License fees include periodic milestone payments, research and development (R&D) contract fees and the amortization of upfront payments received from our licensing partners.

Sales and royalties were $31.3 million for the year ended December 31, 2011, compared to $24.9 million for the year ended December 31, 2010. This increase is largely related to comparatively higher deliveries of Cetrotide® to Merck Serono.

Despite our original expectations for only slight sales volume increases for the year 2011, our sales and royalties were substantially higher than our original expectations due to an unforeseen increase in sales to Merck Serono.

Sales and royalties in 2012 are expected to stabilize, as compared to the amounts recorded in 2011, since we expect that the volume of Cetrotide® sales will stabilize in 2012.

License fees and other revenues were $4.7 million for the year ended December 31, 2011, compared to $2.8 million for the year ended December 31, 2010. This increase is mainly due to the recording of a milestone payment from Yakult with respect to the initiation of a Phase 1/2 trial with perifosine in CRC in Japan during the last quarter of the year 2011.

During 2012, we expect to provide more R&D services, compared to 2011, related to perifosine to our license partner for North America, Keryx. We expect the results of the ongoing Phase 3 trial of perifosine in CRC in the second quarter of 2012. If the data from the trial is positive, we expect to receive development and regulatory milestone payments from our various license partners. With these additional revenues, we expect that our license fees and other revenues will increase significantly in 2012, as compared to 2011.

Operating Expenses

Cost of sales was $27.6 million for the year ended December 31, 2011, compared to $18.7 million for the year ended December 31, 2010. This increase is largely attributable to the comparative increase in volume of sales of Cetrotide® to Merck Serono, as discussed above. Additionally, cost of sales as a percentage of sales and royalties increased to approximately 88.03% for the year ended December 31, 2011, compared to 75.23% for the year ended December 31, 2010. Our lower margins are largely attributable to a decrease of $3.4 million of royalties in 2011, as compared to 2010.

R&D costs, net of refundable tax credits and grants, were $24.5 million for the year ended December 31, 2011, compared to $21.3 million for the year ended December 31, 2010. The comparative increase is mainly attributable to an increase in third-party costs incurred in connection with the advancement of perifosine, AEZS-130 and Erk/PI3K compounds (AEZS-129, AEZS-131, AEZS-132)-related activities

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

(in thousands)	Years ended December 31, 2011	2010
	$	$
Employee compensation and fringe benefits	**10,028**	9,226
Third-party costs	**10,244**	8,138
Facilities rent and maintenance	**1,835**	1,773
Other costs*	**2,793**	2,807
R&D refundable tax credits and grants	**(383)**	(687)
	24,517	21,257

* including depreciation and amortization charges.

The following table summarizes primary third-party R&D costs, by product candidate, incurred by the Company during the years ended December 31, 2011 and 2010.

(in thousands, except percentages)			Years ended December 31,			
Product	**Status**	**Indication**	**2011**		**2010**	
			$	%	$	%
Perifosine	Phases 2 and 3	Oncology	**3,726**	**36.4**	968	11.9
AEZS-130	Phase 3	Endocrinology (diagnosis of AGHD)	**1,156**	**11.3**	865	10.6
Cetrorelix	Phase 3*	BPH*	**-**	**-**	2,046	25.1
AEZS-108	Phase 2	Oncology	**1,652**	**16.1**	2,089	25.7
AEZS-112	Phase 1	Oncology	**538**	**5.3**	259	3.2
AEZS-129, AEZS-131 and AEZS-132; Erk/PI3K	Preclinical	Oncology	**1,860**	**18.2**	923	11.4
Other		Oncology and endocrinology	**1,312**	**12.7**	988	12.1
			10,244	**100.0**	8,138	100.0

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

We expect net R&D costs for 2012 to increase, compared to 2011, as we continue to focus on the development of perifosine and AEZS-108. In particular, we plan to pursue the ongoing Phase 3 study with perifosine in multiple myeloma, as well as initiate, with AEZS-108, a pivotal trial in endometrial cancer and support the ongoing Phase 1/2 studies in connection with triple-negative breast cancer and other indications.

We expect, excluding the impact of unforeseen foreign exchange rate fluctuations, that net R&D costs will total between $30.0 million and $32.0 million for the twelve-month period ending December 31, 2012, due to the additional expenses discussed above. We note, however, that our R&D estimates may be revised as we continue to advance our development activities and as new information becomes available. We also expect that many perifosine-related development activities will be sponsored by our North American license partner, Keryx, and Japanese partner, Yakult, and that we will continue to seek government grants for our earlier-stage projects.

Selling, general and administrative (SG&A) expenses were $16.2 million for the year ended December 31, 2011, compared to $12.6 million for the year ended December 31, 2010. SG&A expenses were higher during the year 2011 mainly due to the recognition of impairment losses and due to the initiation of pre-launch and marketing efforts related to the potential commercialization of perifosine in Europe.

During the year 2011, we recognized an impairment loss ($1.1 million) following impairment testing that was performed on our Cetrotide® asset. The impairment loss was recognized predominantly to take into account management s lower trend estimates related to the commercialization of Cetrotide®, due to changes in the competitive environment in the Japanese market.

In addition, during the year 2011, following the relocation of one of the Company s offices, we recognized an impairment loss on property plant and equipment (leasehold improvements and furniture and fixtures ($0.3 million)) and an additional onerous lease provision ($0.2 million).

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Furthermore, we initiated our pre-launch and marketing efforts related to the potential marketing by the Company of perifosine in Europe. During the year 2011, we incurred approximately $0.9 million in connection with the preparation of a launch plan, including market research, pricing expectations and forecast calculations.

We expect that our SG&A expenses will slightly decrease in 2012, as compared to 2011, as costs return to more normalized operating levels, despite our expected inclusion of certain investments in perifosine s European pre-launch and marketing efforts.

Net finance income (costs), comprised predominantly of net foreign exchange gains (losses), the change in fair value of our warrant liability and the gain on our short-term investment, for the year ended December 31, 2011, totalled $6.2 million, compared to ($3.6 million) for the year ended December 31, 2010, as presented below.

(in thousands)	**Years ended December 31, 2011 $**	**2010 $**
Finance income		
Net gains due to changes in foreign currency exchange rates	**2,197**	932
Change in fair value of warrant liability	**2,533**	-
Interest income	**231**	181
Gain on held-for-trading financial instrument	**1,278**	687
	6,239	1,800
Finance costs		
Change in fair value of warrant liability	**-**	(5,437)
Unwinding of discount	**(8)**	(8)
	(8)	(5,445)
	6,231	(3,645)

The significant increase in net finance income, as compared to the same period in 2010, is mainly due to the change in fair value of our warrant liability. That change results from the periodic mark-to-market revaluation, via the application of the Black-Scholes option pricing model, of currently outstanding share purchase warrants. The Black-Scholes mark-to-market warrant valuation most notably has been impacted by the market price of our common shares, which, on NASDAQ, has fluctuated from $0.81 as at January 4, 2010 to $1.72 as at December 31, 2010, and $1.54 as at December 31, 2011.

Additionally, our net finance income increased during the year 2011 from the same period in 2010 due to higher foreign exchange gains, which in turn resulted primarily from the overall substantial weakening, during 2011, of the euro against the US dollar, as compared to an overall lower weakening of the euro against the US dollar within the 2010 period. As a result, during the twelve-month period ended December 31, 2011, we recorded foreign exchange gains on transactions and on cash and cash equivalent balances denominated in US dollars.

The increase in our net finance income during the year ended December 31, 2011, as compared to the year ended December 31, 2010, also included gains on our short-term investment, which was sold during the year 2011.

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It can be noted that the gains or losses resulting from the periodic mark-to-market valuation of our share purchase warrants did not result in any cash receipt or cash disbursement during the three-month or twelve-month periods ended December 31, 2011 and 2010.

Income tax expense was $1.1 million for the year ended December 31, 2011, as compared to nil for the same period in 2010. The increase consists of foreign withholding taxes related to the Yakult upfront and milestone license fees revenues discussed above.

Net loss for the year ended December 31, 2011 was $27.1 million, or $0.29 per basic and diluted share, compared to $28.5 million, or $0.38 per basic and diluted share for the year ended December 31, 2010.

The decrease in net loss for the year ended December 31, 2011, as compared to the year ended December 31, 2010, is largely due to the significant increase in license fees revenues, as well as net finance income, partly offset by lower margin contribution from Cetrotide®, higher net R&D costs and SG&A expenses, combined with the recording of income tax expense of $1.1 million in 2011, as discussed above.

Quarterly Consolidated Results of Operations Information

The following is a summary of selected consolidated financial information derived from our unaudited interim period consolidated financial statements for each of the eight most recently completed quarters.

(in thousands, except for per share data)

	Quarters ended			
	December 31,	**September 30,**	**June 30,**	**March 31,**
	2011	**2011**	**2011**	**2011**
	$	**$**	**$**	**$**
Revenues	**12,627**	9,514	6,523	7,389
Loss from operations	**(8,688)**	(8,244)	(7,971)	(7,291)
Net (loss) income	**(7,519)**	1,078	(10,569)	(10,057)
Net (loss) income per share				
Basic and diluted	**(0.07)**	0.01	(0.12)	(0.12)

	Quarters ended			
	December 31,	**September 30,**	**June 30,**	**March 31,**
	2010	**2010**	**2010**	**2010**
	$	**$**	**$**	**$**
Revenues	9,971	5,726	5,584	6,422
Loss from operations	(4,003)	(5,456)	(7,950)	(7,397)
Net loss	(6,610)	(9,920)	(6,176)	(5,745)
Net loss per share*				
Basic and diluted	(0.08)	(0.12)	(0.08)	(0.09)

* Net income (loss) per share 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 income (loss) per share amounts may not equal year-to-date net loss per share.

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In the last eight quarters, revenues have increased, when compared quarter over quarter for each of the corresponding periods in 2011 vs. 2010, mainly due to the higher deliveries of Cetrotide® to Merck Serono, which increase have started in the fourth quarter of 2010, the recording of a contingent payment received from Cowen in the fourth quarter of 2010 as well as the recording of a milestone payment from Yakult in the fourth quarter of 2011.

In the last eight quarters, net (loss) income have been impacted by revenues, as mentioned above, by the increased level of net R&D costs in connection with the advancement of perifosine, AEZS-130 and Erk/PI3K compounds, by the recognition of impairment losses in the third and fourth quarters of 2011, by the initiation of pre-launch and marketing efforts related to the potential commercialization of perifosine in Europe in the third and fourth quarters of 2011, as well as by the foreign exchange gain or loss, the change in fair value of our warrant liability and the gain on our short-term investment.

Fourth Quarter 2011 Results

Revenues

Revenues were $12.6 million for the quarter ended December 31, 2011, compared to $10.0 million for the same quarter in 2010. The increase in revenues is due primarily to comparative higher deliveries of Cetrotide® to Merck Serono ($3.6 million) and to the recording of a milestone payment ($2.6 million) from Yakult with respect to the initiation of a Phase 1/2 trial with perifosine in CRC in Japan, partly offset by the lower royalties in 2011 attributable to the contingent payment of $2.5 million from Cowen recorded in December 2010.

Operating expenses

Cost of sales was $8.1 million for the quarter ended December 31, 2011, compared to $5.4 million for the same quarter in 2010. This increase is largely attributable to the comparative increase in volume of sales of Cetrotide® to Merck Serono, as discussed above. Additionally, cost of sales as a percentage of sales and royalties increased to approximately 88% for the quarter ended December 31, 2011, compared to 63% for the same period in 2010. Our lower margins are largely attributable to a lower contribution of royalties to total sales and royalties in 2011, as compared to 2010, as a result of a $3.4 million royalty milestone payment recorded in 2010 from Cowen.

R&D costs, net of refundable tax credits and grants were $7.8 million for the quarter ended December 31, 2011, compared to $5.4 million for the same quarter in 2010. The comparative increase in R&D expenses primarily results from the increase in third-party costs incurred in connection with the advancement of perifosine, AEZS-108 and Erk/PI3K compounds (AEZS-129, AEZS-131, AEZS-132)-related activities.

Selling, general and administrative (SG&A) expenses were $5.4 million for the quarter ended December 31, 2011, compared to $3.2 million for the same quarter in 2010. The increase in SG&A expenses is mainly related to the recognition of an impairment loss on property, plant and equipment (leasehold improvements and furniture and fixtures ($0.3 million)) and an additional onerous lease provision ($0.2 million), combined with the pre-launch and marketing efforts related to the potential commercialization of perifosine in Europe ($0.5 million) and with the increase in foreign exchange loss ($0.6 million).

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Net finance income (costs), comprised predominantly of net foreign exchange gains, the change in fair value of our warrant liability and the gain on our short-term investment (2010 only), for the quarter ended December 31, 2011, totalled $1.4 million, compared to ($2.6 million) for the same period in 2010, as presented below.

(in thousands)	**Three months ended December 31, 2011 $**	**Three months ended December 31, 2010 $**
Finance income		
Net gains due to changes in foreign currency exchange rates	**1,118**	276
Change in fair value of warrant liability	**221**	-
Interest income	**95**	70
Gain on held-for-trading financial instrument	**-**	687
	1,434	1,033
Finance costs		
Change in fair value of warrant liability	**-**	(3,638)
Unwinding of discount	**(2)**	(2)
	(2)	(3,640)
	1,432	(2,607)

Net loss amounted to $7.5 million, or $0.07 per basic and diluted share, for the quarter ended December 31, 2011, compared to $6.6 million, or $0.08 per basic and diluted share, for the same quarter in 2010. The increase in net loss is mainly attributable to the higher comparative R&D and SG&A expenses, partly offset by the significant increase in net finance income and by higher gross margin relating to sales of Cetrotide®.

Primarily, given the presence in our 2011 fourth quarter revenues of the $2.6 million milestone receivable from Yakult with respect to the initiation of a Phase 1/2 trial with perifosine in CRC in Japan, and excluding any impact of foreign exchange gains or losses, as well as change in fair value of warrant liability, we expect that the net loss for the first quarter of 2012 will increase, as compared to the fourth quarter of 2011.

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Consolidated Statement of Financial Position Information

(in thousands)	As at December 31, **2011** **$**	2010 $	2009* $
Cash and cash equivalents	**46,881**	31,998	38,100
Short-term investment	**-**	1,934	-
Trade and other receivables and other current assets	**13,258**	9,877	10,913
Restricted cash	**806**	827	878
Property, plant and equipment, net	**2,512**	3,096	4,358
Other non-current assets	**11,912**	13,716	32,013
Total assets	**75,369**	61,448	86,262
Payables and other current liabilities	**17,784**	13,350	19,211
Long-term payable (current and non-current portions)	**88**	150	200
Warrant liability (current and non-current portions)	**9,204**	14,367	-
Non-financial non-current liabilities**	**52,839**	51,156	57,625
Total liabilities	**79,915**	79,023	77,036
Shareholders (deficiency) equity	**(4,546)**	(17,575)	9,226
Total liabilities and shareholders (deficiency) equity	**75,369**	61,448	86,262

* We adopted IFRS in 2011 with a transition date of January 1, 2010. The selected financial information for the years ended December 31, 2009, 2008 and 2007 is derived from financial statements that were presented in accordance with Canadian GAAP and has not been restated in accordance with IFRS. Consequently, the selected financial information for the years ended December 31, 2009, 2008 and 2007 may not be comparable with the corresponding selected financial information for the years ended December 31, 2011 and 2010. Please refer to Critical Accounting Policies, Estimates and Judgments for the policy differences between Canadian GAAP and IFRS.

** Comprised mainly of non-current portion of deferred revenues, employee future benefits and provision.

The increase in cash and cash equivalents as at December 31, 2011, as compared to December 31, 2010, is due to the receipt of net proceeds pursuant to drawdowns made in connection with the February and June ATM Sales Agreements, as discussed above, to the proceeds from the sale of our short-term investment, to the receipt of the upfront license payment in connection with our development, commercialization and licensing agreement entered into with Yakult, as noted above, as well as to the receipt of net proceeds from the exercise of share purchase warrants. These increases were partially offset by recurring disbursements and other variations in components of our working capital.

Trade and other receivables and other current assets increased from December 31, 2010 to December 31, 2011 mainly due to the increase in trade receivable in connection with the higher deliveries of Cetrotide® to Merck Serono for an amount of approximately $2.9 million and due to the perifosine inventory to be sold to our partner Keryx for an amount of approximately $0.7 million which in turn were partly offset by the impact of foreign exchange rate fluctuations.

Other non-current assets decreased from December 31, 2010 to December 31, 2011 primarily due to the net reduction in the carrying value of our identifiable intangible assets, which was negatively impacted by the impairment loss recognized on Cetrotide®, as discussed above.

Payables and other current liabilities increased from December 31, 2010 to December 31, 2011 mainly due to an increase of approximately $2.4 million in trade accounts payable and accrued liabilities in connection with the higher deliveries of Cetrotide® and with the higher level of R&D expenses resulting from the advancement of perifosine, AEZS-130 and Erk/PI3K compounds, which includes an increase due to the perifosine R&D inventory received and payable for an amount of approximately $1.3 million, to the increase in current portion of deferred revenues for an amount of $1.3 million, and to the increase in accrued salaries for an amount of approximately $0.5 million which in turn were partly offset by the impact of foreign exchange rate fluctuations.

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Our warrant liability was lower as at December 31, 2011, as compared to December 31, 2010, predominantly due to the change in fair value pursuant to the periodic mark-to-market revaluation of the underlying outstanding share purchase warrants, as well as following various warrant exercises during the twelve-month period ended December 31, 2011.

Non-financial liabilities were higher as at December 31, 2011, as compared to December 31, 2010, mainly as a result of the deferral of the upfront payment received in connection with our development, commercialization and licensing agreement entered into with Yakult, partially offset by the recurring amortization of deferred revenues.

The net decrease in shareholders deficiency as at December 31, 2011, as compared to December 31, 2010, is predominantly attributable to an increase in share capital following the issuance of common shares pursuant to the aforementioned drawdowns made in connection with the February and June ATM Sales Agreements, partially offset by the increase in our deficit due to the net loss for the year ended December 31, 2011 and by the decrease in accumulated other comprehensive income, which in turn is comprised of cumulative translation adjustments.

Financial Liabilities, Obligations and Commitments

We have certain contractual leasing obligations and purchase obligation commitments. Purchase obligation commitments mainly include R&D services and manufacturing agreements related to the production of Cetrotide® and to other R&D programs. The following tables summarize future cash requirements with respect to these obligations.

Future minimum lease payments and future minimum sublease payments expected to be received under non-cancellable operating leases (subleases), as well as future payments in connection with utility service agreements, as at December 31, 2011 are as follows:

(in thousands)	**Minimum lease payments $**	**Minimum sub-lease payments $**	**Utilities $**
Less than 1 year	1,690	(226)	609
1 3 years	3,128	(451)	793
4 5 years	2,172	(451)	496
More than 5 years	373	(244)	-
Total	**7,363**	**(1,372)**	**1,898**

Service and manufacturing commitments given, which consist of R&D service agreements and manufacturing agreements for Cetrotide®, are as follows:

(in thousands)	**As at December 31, 2011 $**
Less than 1 year	10,760
1 3 years	3,855
4 5 years	-
More than 5 years	-
Total	14,615

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Outstanding Share Data

As at March 27, 2012, there were 108,787,366 common shares issued and outstanding, as well as 7,497,634 stock options outstanding. Share purchase warrants outstanding as at March 27, 2012 represented a total of 8,752,868 equivalent common shares.

Capital Disclosures

Our objective in managing capital, primarily composed of shareholders deficiency and cash and cash equivalents, is to ensure sufficient liquidity to fund our R&D activities, SG&A expenses, working capital and capital expenditures.

Historically, our priority has been to optimize our liquidity by non-dilutive sources, including the sale of non-core assets, investment tax credits and grants, interest income, licensing and related services and royalties. More recently, however, we have raised additional capital via registered direct offerings and drawdowns related to the February and June ATM Sales Agreements, and we expect to continue to raise capital via drawdowns related to the January 2012 ATM Sales Agreement.

At December 31, 2011, cash and cash equivalents amounted to $46.9 million. The Company believes that its cash position will be sufficient to finance its operations and capital needs for at least the next twelve months.

Our capital management objective remains the same as that of previous periods. 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 patterns of expenditures 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 given time and on the availability of funding from investors and prospective commercial partners.

Liquidity, Cash Flows and Capital Resources

Our operations and capital expenditures have been financed mainly through cash flows from operating activities and other non-dilutive activities, except for the registered direct offerings completed during the year ended December 31, 2010 and, more recently, the drawdowns related to our various ATM Sales Agreements, as discussed above.

Our cash and cash equivalents amounted to $46.9 million as at December 31, 2011, as compared to $32.0 million as at December 31, 2010. As at December 31, 2011, cash and cash equivalents, denominated in euro, amounted to 10.3 million.

Based on our assessment, which took into account current cash levels, as well as our strategic plan and corresponding budgets and forecasts, we believe that we have sufficient liquidity and financial resources to fund planned expenditures and other working capital needs for at least, but not limited to, the twelve-month period following the balance sheet date of December 31, 2011.

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.

The variations in our liquidity by activity are explained below.

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Operating Activities

Cash used in operating activities totalled $26.2 million for the year ended December 31, 2011, as compared to $31.7 million for the year ended December 31, 2010. The decrease in cash used in operating activities is due in large part to the receipt, during the year 2011, of nearly $8.4 million in connection with our development, commercialization and licensing agreement entered into with Yakult, as discussed above, as well as to lower trade accounts payable settlements, partially offset by lower trade accounts receivable settlements.

We expect net cash used in operating activities to increase during 2012, as compared to 2011, as we increase our investment in perifosine and in AEZS-108, as discussed above.

Financing Activities

Cash flows provided by financing activities increased to $38.6 million for the year ended December 31, 2011, as compared to $26.0 million for the year ended December 31, 2010. The increase is primarily due to the receipt of higher net proceeds from offerings such as the drawdowns related to the February and June ATM Sales Agreements, discussed above, as well as to the increase in proceeds received following the exercise of share purchase warrants.

Investing Activities

Cash flows provided by investing activities reached $2.5 million for the year ended December 31, 2011, as compared to cash flows used in investing activities of nearly $0.05 million for the year ended December 31, 2010. The increase in cash provided by investing activities is due to the increase in cash proceeds received on the sale of our short-term investment, partly offset by cash disbursements made in connection with the purchases of laboratory and other equipment used in ongoing R&D activities.

Critical Accounting Policies, Estimates and Judgments

As noted above, our consolidated financial statements as at December 31, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010 have been prepared in accordance with IFRS. Note 29 Transition to IFRS to our consolidated financial statements as at December 31, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010 discusses the impact of the transition to IFRS on our reported financial position, financial performance and cash flows, including the nature and effect of significant changes in accounting policies from those used in our previous Canadian GAAP consolidated financial statements as at December 31, 2010 and for the year then ended. Comparative figures for 2010 have been adjusted to give effect to these changes.

These consolidated financial statements were approved by our Board of Directors for issue on March 27, 2012.

Additionally, the preparation of consolidated financial statements in accordance with IFRS often requires management to make estimates about and apply assumptions or subjective judgment to future events and other matters that affect the reported amounts of our assets, liabilities, revenues, expenses and related disclosures. Assumptions, estimates and judgments are based on historical experience, expectations, current trends and other factors that management believes to be relevant at the time at which our consolidated financial statements are prepared. Management reviews, on a regular basis, the Company s accounting policies, assumptions, estimates and judgments in order to ensure that the consolidated financial statements are presented fairly and in accordance with IFRS.

Critical accounting estimates and judgments are those that have a significant risk of causing material adjustment and are often applied to matters or outcomes that are inherently uncertain and subject to change. As such, management cautions that future events often vary from forecasts and expectations and that estimates routinely require adjustment.

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A summary of those areas where we believe critical accounting policies affect the significant judgments and estimates used in the preparation of our consolidated financial statements can be found in note 3 to our consolidated financial statements as at December 31, 2011 and December 31, 2010 and for the years ended December 31, 2011 and 2010.

Accounting Standards Not Yet Adopted

In November 2009 and October 2010, the IASB issued IFRS 9, *Financial Instruments* (IFRS 9), which represents the completion of the first part of a three-part project to replace IAS 39, *Financial Instruments: Recognition and Measurement*, with a new standard. Per the new standard, an entity choosing to measure a liability at fair value will present the portion of the change in its fair value due to changes in the entity s own credit risk in the other comprehensive income or loss section of the entity s statement of comprehensive loss, rather than within profit or loss. Additionally, IFRS 7 Amendment includes revised guidance related to the derecognition of financial instruments. IFRS 9 applies to financial statements for annual periods beginning on or after January 1, 2015, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

In May 2011, the IASB issued IFRS 10, *Consolidated Financial Statements* (IFRS 10), which builds on existing principles by identifying the concept of control as the determining factor in whether an entity should be included within the consolidated financial statements of a parent company. IFRS 10 also provides additional guidance to assist in the determination of control where this is difficult to assess. IFRS 10 applies to financial statements for annual periods beginning on or after January 1, 2013, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

In May 2011, the IASB issued IFRS 11, *Joint Arrangements* (IFRS 11), which enhances accounting for joint arrangements, particularly by focusing on the rights and obligations of the arrangement, rather than the arrangement s legal form. IFRS 11 also addresses inconsistencies in the reporting of joint arrangements by requiring a single method to account for interests in jointly controlled entities and prohibits proportionate consolidation. IFRS 11 applies to financial statements for annual periods beginning on or after January 1, 2013, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

In May 2011, the IASB issued IFRS 12, *Disclosure of Interests in Other Entities* (IFRS 12), which is a comprehensive standard on disclosure requirements for all forms of interests in other entities, including joint arrangements, associates, special purpose vehicles and other off-balance sheet vehicles. IFRS 12 applies to financial statements for annual periods beginning on or after January 1, 2013, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

In May 2011, the IASB issued IFRS 13, *Fair Value* Measurement (IFRS 13), which defines fair value, sets out in a single IFRS a framework for measuring fair value and requires disclosures about fair value measurements. IFRS 13 does not determine when an asset, a liability or an entity s own equity instrument is measured at fair value. Rather, the measurement and disclosure requirements of IFRS 13 apply when another IFRS requires or permits the item to be measured at fair value (with limited exceptions). IFRS 13 applies to financial statements for annual periods beginning on or after January 1, 2013, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

In June 2011, the IASB amended IAS 1, *Presentation of Financial Statements* (IAS 1), to change the disclosure of items presented in other comprehensive income into two groups, based on whether those items may be recycled to profit or loss in the future. The amendments to IAS 1 apply to financial statements for annual periods beginning after July 1, 2012, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

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In June 2011, the IASB issued an amended version of IAS 19, *Employee Benefits* (IAS 19), including the elimination of the option to defer the recognition of actuarial gains and losses (known as the corridor method), the streamlining of the presentation of changes in assets and liabilities arising from defined benefit plans and the enhancement of the disclosure requirements for defined benefit plans, including additional information about the characteristics of defined benefit plans and the risks to which entities are exposed through participation in those plans. The amendments to IAS 19 apply to financial statements for annual periods beginning on or after January 1, 2013, with early adoption permitted. We currently are evaluating any impact that this new guidance may have on our consolidated financial statements.

Outlook for 2012

Perifosine

Our primary focus continues to be on the advancement of the ongoing Phase 3 registration studies in both refractory advanced colorectal cancer and multiple myeloma. We expect the results of the ongoing Phase 3 trial of perifosine in CRC in the second quarter of 2012.

We expect to continue to invest in the preparation of marketing and pre-launch activities of perifosine in Europe.

AEZS-108

We expect to initiate a multi-center pivotal study in endometrial cancer, as well as a multi-center Phase 2 study in triple-negative breast cancer. We also expect to pursue our Phase 1/2 studies in castration- and taxane-resistant prostate cancer with the University of Southern California, and in refractory bladder cancer with the University of Miami.

AEZS-130

Following the completion and positive Phase 3 results for AEZS-130 as a diagnostic for AGHD in the United States, we expect, after a successful receipt of pre-NDA meeting with the FDA, to file an NDA.

We also expect to pursue our ongoing Phase 2A study with AEZS-130 as a treatment for cancer-cachexia.

Revenue Expectations

Revenues are expected to increase in 2012, as compared to 2011, given our expectation for potential increased license fees revenues from our partners and despite the expected stabilization of our sales and royalties, as discussed above.

Operating Expense Expectations

During 2012, we expect to continue to focus our R&D efforts on our later-stage compounds, including perifosine and AEZS-108. With our focused strategy, and given our expectations related to R&D investments made on behalf of Keryx, we now expect our R&D expenses to total between $30 million and $32 million for the whole of 2012, as compared to $24.5 million in 2011. However, certain perfosine-related R&D expenses will be reimbursed by our partner, Keryx.

We expect that our overall operating burn in 2012 will increase, as compared to 2011, due most notably to the receipt in 2011 of $8.4 million in connection with our licensing agreement entered into with Yakult and our expected increase in R&D investments, as discussed above, partly offset by increased license fees from our partners.

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Financial Risk Factors and Other Instruments

Fair Value

The change in fair value of our warrant liability results from the periodic mark-to-market revaluation, via the application of the Black-Scholes option pricing model, of currently outstanding share purchase warrants. The Black-Scholes valuation is impacted, among other inputs, by the market price of our common shares. As a result, the change in fair value of the warrant liability, which is reported as finance income (costs) in our consolidated statements of comprehensive loss, has been and may continue in future periods to be materially affected most notably by changes in our common share price, which has ranged from $1.43 to $2.58 on NASDAQ during the year ended December 31, 2011.

Assuming the following variations of the market price of our common shares over a 12-month period:

Market price variations of -10% and +10%

If these variations were to occur, the impact on our net loss for warrant liability held at December 31, 2011 would be as follows:

(in thousands)	**Carrying amount $**	**-10% $**	**+10% $**
Warrant liability*	9,204	1,155	(1,174)
Total impact on net loss decrease/(increase)		1,155	(1,174)

* Includes current and non-current portions.

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

The following variations are reasonably possible over a 12-month period:

Foreign exchange rate variations of -5% (depreciation of the EUR) and +5% (appreciation of the EUR) against the US$, from a year-end rate of EUR1 = US$1.2972.

If these variations were to occur, the impact on the Company s net loss for each category of financial instruments held at December 31, 2011 would be as follows:

		Balances denominated in US$	
(in thousands)	**Carrying amount $**	**-5% $**	**+5% $**
Cash and cash equivalents	33,669	1,683	(1,683)
Warrant liability*	9,204	(460)	460

Total impact on net loss	**decrease/(increase)**	1,223	(1,223)

* Includes current and non-current portions.

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For the year ended December 31, 2011, we were not a party to any forward-exchange contracts, and no forward-exchange contracts were outstanding as at March 27, 2012.

Liquidity Risk

Liquidity risk is the risk that we will not be able to meet our financial obligations as they become due. As indicated in the Capital Disclosures section above, we manage this risk through the management of our capital structure. We also manage liquidity risk by continuously monitoring actual and projected cash flows. The Board of Directors reviews and approves our operating and capital budgets, as well as any material transactions out of the ordinary course of business. We have adopted an investment policy in respect of the safety and preservation of our capital to ensure our liquidity needs are met. The instruments are selected with regard to the expected timing of expenditures and prevailing interest rates.

Credit Risk

Credit risk is the risk of an unexpected loss if a customer or counterparty to a financial instrument fails to meet its contractual obligations. We regularly monitor credit risk exposure and take steps to mitigate the likelihood of this exposure resulting in losses. Our exposure to credit risk currently relates to cash and cash equivalents, to trade and other receivables and to restricted cash. We invest our available cash in amounts that are readily convertible to known amounts of cash and deposit our cash balances with financial institutions that have a minimum rating of A3 .

As at December 31, 2011, trade accounts receivable for an amount of approximately $7,415,000 were with two external customers or partners.

As at December 31, 2011, no trade accounts receivable were past due or impaired.

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, 2011.

As at December 31, 2011, 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 27, 2012.

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
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.

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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 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 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 Inc., Investissement Québec and Chairman of the Board of Foster Wheeler Énergie Inc. He holds a law degree from the University of Ottawa and is a member of the *Barreau du Québec*.

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 operating 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

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Pharmaceutical Council USA and was a Board member of the Pharmaceutical Manufacturers Association of Canada, where he 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., Board member of Algorithme Pharma Inc. and Insy s Therapeutics Inc., a Phoenix Arizona based specialty pharma Co. 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, at the request of the Board of the University of Montreal, has participated in the selection of the Dean of the Faculty of Medicine in 2011. Mr. Limoges is a board member or trustee and chairman of the audit committees of the following public companies: Aeterna Zentaris Inc. (TSX and NASDAQ), Atrium Innovations Inc. (TSX), Hartco Inc. (TSX) and Hart Stores Inc. (TSXV). He is also a board member of various private companies and charities. Mr. Limoges became an FCA in 1984 and received the Order of Canada in 2002.

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 Norton Rose Canada 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. 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.

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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 Norton Rose Canada 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

Our executive officers are generally paid in their home country s currency. Unless otherwise indicated, all directors and executive compensation information included in this document is presented in US dollars and, to the extent a director or officer has been paid in a currency other than US dollars (Canadian dollars or euros), the amounts have been converted from such person s home country currency to US dollars based on the following average exchange rates: for the financial year ended December 31, 2011: 1.000 = US$1.3919 and CAN$1.000 = US$1.0111; for the financial year ended December 31, 2010: 1.000 = US$1.326 and CAN$1.000 = US$0.970; and for the financial year ended December 31, 2009: 1.000 = US$1.388 and CAN$1.000 = US$0.876.

1. 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). The Governance Committee is composed of three (3) directors, each of whom is independent, namely Messrs. José P. Dorais (Chair), Juergen Ernst and Gérard Limoges. 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, their respective chairs and individual directors and (iv) making recommendations to the Board with respect to directors compensation.

We did not employ the services of any external compensation consultant in or with respect to the financial year ended December 31, 2011.

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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 2011 (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.

Outstanding Option-Based Awards and Share-Based Awards

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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, 2011:

	Option-based Awards					Share-based Awards		
Name	Issuance Date (mm-dd-yyyy)	Number of Securities Underlying Unexercised Options[1] (#)	Option Exercise Price (CAN$ or US$)	Option Expiration Date (mm-dd-yyyy)	Value of Unexercised In-the-money Options[2] (CAN$ or US$)	Issuance Date (mm-dd-yyyy)	Number of Shares or Units of Shares that have Not Vested (#)	Market or Payout Value of Share-based Awards that have Not Vested ($)
Aubut, Marcel	12-16-2002	15,000	CAN$3.68	12-15-2012				
	12-11-2003	30,000	CAN$1.74	12-10-2013				
	12-14-2004	15,000	CAN$5.83	12-13-2014				
	12-13-2005	15,000	CAN$3.53	12-12-2015				
	01-04-2007	5,000	CAN$4.65	01-03-2017				
	12-11-2007	25,000	CAN$1.82	12-10-2017				
	12-08-2008	15,000	CAN$0.55	12-08-2018	CAN$15,150			
	12-09-2009	20,000	CAN$0.95	12-08-2019	CAN$12,200			
	12-08-2010	30,000	CAN$1.52	12-07-2020	CAN$1,200			
	12-07-2011	50,000	US$1.74	12-06-2021				
Dorais, José P.	12-08-2010	30,000	CAN$1.52	12-07-2020	CAN$1,200			
	12-07-2011	50,000	US$1.74	12-06-2021				
Ernst, Juergen	02-25-2005	15,000	CAN$5.09	02-24-2015				
	12-13-2005	15,000	CAN$3.53	12-12-2015				
	01-04-2007	5,000	CAN$4.65	01-03-2017				
	12-11-2007	25,000	CAN$1.82	12-10-2017				
	11-14-2008	100,000	CAN$0.65	11-13-2018	CAN$91,000			
	12-08-2008	15,000	CAN$0.55	12-08-2018	CAN$15,150			
	12-09-2009	20,000	CAN$0.95	12-08-2019	CAN$12,200			
	12-08-2010	30,000	CAN$1.52	12-07-2020	CAN$1,200			
	12-07-2011	50,000	US$1.74	12-06-2021				
Lapalme, Pierre	12-09-2009	20,000	CAN$0.95	12-08-2019	CAN$12,200			
	12-08-2010	30,000	CAN$1.52	12-07-2020	CAN$1,200			
	12-07-2011	50,000	US$1.74	12-06-2021				
Limoges, Gérard	12-14-2004	15,000	CAN$5.83	12-13-2014				
	12-13-2005	15,000	CAN$3.53	12-12-2015				
	01-04-2007	5,000	CAN$4.65	01-03-2017				
	12-11-2007	25,000	CAN$1.82	12-10-2017				
	12-08-2008	15,000	CAN$0.55	12-08-2018	CAN$15,150			
	12-09-2009	20,000	CAN$0.95	12-08-2019	CAN$12,200			
	12-08-2010	30,000	CAN$1.52	12-07-2020	CAN$1,200			
	12-07-2011	50,000	US$1.74	12-06-2021				
Meyers, Michael	05-27-2011	20,000	US$2.36	05-26-2021				
	12-07-2011	40,000	US$1.74	12-06-2021				

(1) The number of securities underlying unexercised options represent all awards outstanding as at December 31, 2011.

(2) Value of unexercised in-the-money options at financial year-end is calculated based on the difference between the closing prices of the common shares on the TSX or NASDAQ, as applicable, on the last trading day of the fiscal year (December 30, 2011) of CAN$1.56 and US$1.54, respectively, 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).

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Total Compensation of Outside Directors

The table below summarizes the total compensation earned by the Outside Directors during the financial year ended December 31, 2011 (all amounts are in US dollars):

Name	Fees earned ($)		Share-based Awards	Option-based Awards[2]	Non-Equity Incentive Plan Compensation	Pension Value	All Other Compensation[3]	Total
	Retainer[1]	Attendance[1]	($)	($)	($)	($)	($)	($)
Aubut, Marcel	15,166	4,044		63,000				82,210
Dorais, José P.[4]	27,298	8,088		63,000			506	98,892
Ernst, Juergen	86,299	9,743		63,000			5,568	164,610
Lapalme, Pierre	19,210	9,099		63,000				91,309
Limoges, Gérard[5]	32,354	11,122		63,000				106,476
MacDonald, Pierre[6]	11,883	3,033					506	15,422
Meyers, Michael[7]	13,250	4,500		85,800			500	104,050

(1) These amounts represent the portion paid in cash to the Outside Directors and are paid in each director s home country currency.
(2) The value of option-based awards represents the closing price of the common shares on NASDAQ at the date of grant (US$1.74 for options granted on December 7, 2011 and US$2.36 for options granted to Michael Meyers on May 27, 2011) multiplied by the Black-Scholes factor as at such date (72.41% for options granted on December 7, 2011 and 75.00% for options granted to Michael Meyers on May 27, 2011) and the number of stock options granted on such date.
(3) These amounts represent fees paid in cash for special tasks or overseas travelling and are also paid in each director s home country currency.
(4) José P. Dorais was appointed Chairman of the Governance Committee on May 18, 2011.
(5) Gérard Limoges has been a member of the Governance Committee since May 18, 2011.
(6) Pierre MacDonald was a director of the Company from 2001 to May 18, 2011. He did not stand for re-election as director at the annual and special meeting of shareholders held on May 18, 2011 and therefore ceased to be a director as of such date.
(7) Michael Meyers was appointed director on March 22, 2011 and has been a member of the Audit Committee since May 18, 2011.

During the financial year ended December 31, 2011, the Company paid an aggregate amount of $262,169 to all of its Outside Directors for services rendered in their capacity as directors, excluding reimbursement of out-of-pocket expenses and the value of option-based awards granted in 2011.

2. Compensation of Executive Officers

The mandate of the Governance Committee provides that it is responsible for taking all reasonable measures to ensure that appropriate human resources systems and procedures, such as hiring policies, competency profiles, training policies and compensation structures are in place so that we can attract, motivate and retain the quality of senior management required to meet our business objectives.

The Governance Committee also assists the Board in discharging its responsibilities relating to executive and other human resources hiring, assessment, compensation and succession planning matters.

Thus, the Governance Committee recommends the appointment of senior officers, including the terms and conditions of their appointment and termination, and reviews the evaluation of the performance of such senior officers, including recommending their compensation and overseeing risk identification and management in relation to executive compensation policies and practices. The Board, which includes the members of the Governance Committee, reviews the corporate goals and objectives that are set annually and evaluates the Chief Executive Officer s performance and compensation in light of such goals and objectives.

The Governance Committee recognizes that the industry and competitive environment in which the Company operates requires a balanced level of risk-taking to promote and achieve the performance expectations required to provide its shareholders with a fair return. However, the executive compensation program should not encourage senior executives in taking excessive risk. In this regard, the Governance Committee

recommends the implementation of compensation methods that tie a portion of senior executive compensation to each of the short-term and longer-term performance of the Company and each executive officer and that take into account the advantages and risks associated with such compensation methods. The Governance Committee is also responsible for creating compensation policies that are intended to reward the creation of shareholder value while reflecting a balance between the short-term and longer-term performance of the Company and of each executive officer.

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Compensation Discussion & Analysis

Compensation Philosophy and Objectives

The Company s executive compensation program is designed to attract, motivate and retain high performing senior executives, encourage and reward superior performance and align the executives interests with those of our shareholders by:

- providing the opportunity for an executive to earn compensation that is competitive with the compensation received by executives employed by a group of comparable North American companies;
- providing executives with an equity-based incentive plan, namely a stock option plan;
- aligning employee compensation with company corporate objectives; and
- attracting and retaining highly qualified individuals in key positions.

Risk Assessment of Executive Compensation Program

The Board, through the Governance Committee, oversees the implementation of compensation methods that tie a portion of executive compensation to each of the short-term and longer-term performance of the Company and of each executive officer and that take into account the advantages and risks associated with such compensation methods. In addition, the Board oversees the creation of compensation policies that are intended to reward the creation of shareholder value while reflecting a balance between the short-term and longer-term performance of the Company and of each executive officer.

The Governance Committee has considered in general terms the concept of risk as it relates to the Company s executive compensation program.

Base salaries are fixed in amount to provide a steady income to the executive officers regardless of share price and thus do not encourage or reward risk-taking to the detriment of other important business metrics. The variable compensation elements (annual bonuses and stock options) are designed to reward both short and long-term performance. For short-term performance, an annual bonus is awarded based on the level of attainment of specific operational and corporate goals that the Governance Committee believes to be challenging, yet does not encourage unnecessary or excessive risk-taking (for 2011, such operational and corporate goals are listed in the table below). While the Company s bonus payments are generally based on annual results, their value is generally capped and represents only a portion of each individual s overall total compensation opportunities. Finally, a significant portion of the compensation provided to the Company s executive officers is in the form of equity awards under the long-term incentive compensation plan that further align executives interests with those of shareholders. The Governance Committee believes that these awards do not encourage unnecessary or excessive risk-taking since the ultimate value of the awards is tied to the Company s share price, and since grants under the long-term incentive compensation plan are subject to long-term vesting schedules to help ensure that executives generally have significant value tied to long-term share price performance.

The Governance Committee believes that the variable compensation elements (annual bonuses and stock options) represent a percentage of overall compensation that is sufficient to motivate the Company s executive officers to produce superior short-term and long-term corporate results, while the fixed compensation element (base salary) is also sufficient to discourage executive officers from taking unnecessary or excessive risks. The Governance Committee and the Board also generally have the discretion to adjust annual bonuses and stock option grants based on individual performance and any other factors it may determine to be appropriate in the circumstances.

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Such factors may include, where necessary or appropriate, the level of risk-taking a particular executive officer may have engaged in during the preceding year.

Based on the foregoing, the Governance Committee has not identified any risks associated with the Company s executive compensation program that are reasonably likely to have a material adverse effect on the Company. The Governance Committee believes that the Company s executive compensation program does not encourage or reward any unnecessary or excessive risk-taking behaviour.

The Board, based on the Governance Committee s recommendation, set goals for the Company at the end of 2010, which constitute the performance objectives for the Chief Executive Officer and the Company s other executive officers. The performance objectives are not established for individual executive officers but rather by function(s) exercised within the Company, for many of which the Named Executive Officers are responsible.

In December 2011, the Governance Committee determined that the Company s executive officers met or exceeded each of the objectives set forth in the table below as follows:

Objectives for 2011	**Results for 2011**
Clinical	
Advancement of product pipeline	
Perifosine	Completed recruitment of metastatic colorectal Phase 3 study (468 patients)
	Supported Phase 3 study in MM by opening several centres outside North America
AEZS-130	Reported positive Phase 3 data as diagnostic in AGHD
AEZS-108	Reported positive final Phase 2 data in endometrial cancer
	Initiated Phase 1/2 study in refractory bladder cancer in the United States
Regulatory	
Optimize regulatory strategy with lead authorities	Obtained parallel scientific advice for a pivotal study for AEZS-108 in endometrial cancer by both the Food and Drug Administration (FDA) and the EMA
Business Development/Alliance Management:	
Expand partner portfolio for Perifosine in non-core territories	Signed license agreements with Yakult (for Japan) and Hikma (for MENA region)
Ensure steady supply of Cetrotide® to Merck Serono	Exceeded pre-defined threshold of Cetrotide® sales triggering further milestone payment by Cowen Healthcare Royalty Partners, L. P.

Manage alliances with existing drug development and commercialization partners actively	Continued to maintain excellent relations with alliance partners
Financial	
Pursue dilutive and non-dilutive financing alternatives to ensure year-end 2011 cash and cash equivalents in excess of $30 million	Completed two successful At-the-Market Financings (ATMs) generating proceeds in excess of $36 million
	Closed further licensing agreements generating significant upfront payments
	Cash and cash equivalents as of December 31, 2011 totalled $46.9 million

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Investor Relations	
Expand North American research analyst coverage	Initiation of coverage by several North American institutions including, among others, Oppenheimer & Co Inc., JMP Securities and Needham & Company LLC.
Continue to present at strategic healthcare conferences	Presented at several strategic healthcare and partnership conferences and continued to build the Company s investor base
Human Resources	
Maintain high level of motivation at all levels (and sites) of the Company	Executed effective HR policy comprising both monetary (compensation) and non-monetary (training, etc.) components to ensure both short- and long-term commitment of personnel while generally aligning employee s and shareholders interests
	Maintained HR-attrition policy while progressing development pipeline (active headcount virtually unchanged as compared to the prior year)

The determination of individual performance does not involve quantitative measures using a mathematical calculation in which each individual performance objective is given a numerical weight. Instead, the Governance Committee s determination of individual performance is a subjective determination as to whether a particular executive officer substantially achieved the stated objectives or over-performed or under-performed with respect to corporate objectives that were deemed to be important to the Company s success.

While the Company has not formally adopted a policy prohibiting or restricting its executive officers and directors from purchasing financial instruments, including, for greater certainty, pre-paid variable forward contracts, equity swaps, collars, or units of exchange funds, which are designed to hedge or offset a decrease in market value of equity securities granted as executive compensation or directors remuneration, the Company s executive officers and directors have not historically engaged in such financial instruments or transactions. In addition, the Company s disclosure and trading policy requires that all reporting insiders , including executive officers and directors, pre-clear with the Company s Corporate Secretary each trade relating to the Company s securities, which would include the entering into of any such financial instrument or transaction, hedge, swap or forward contract.

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Benchmarking

In order to attain our objectives of providing market competitive compensation opportunities, our executive compensation plan, based on a study provided by AON Consulting (and updated annually), is benchmarked against market compensation data gathered from organizations of comparable size and/or stage of development of other companies that the Company competes for executive talent (the Reference Group). We did not, however, pay AON Consulting any fee or other remuneration in 2011. An overview of the characteristics of the Reference Group is provided in the following table:

(In millions of US$)

	Aeterna Zentaris	Survey Reference Group
Location	North America and Europe	North America
Industries	Biopharmaceutical	Biopharmaceutical
Revenues		
	27.70[(1)]	57.02[(2)]
Last fiscal year		
Market Capitalization		
	175.07	290.34
As at October 31, 2011		
Net Loss		
	23.22[(1)]	19.82[(2)]
Last fiscal year		

(1) For the year ended December 31, 2010, as presented in our 2010 audited consolidated financial statements, which were presented in conformity with Canadian GAAP.

(2) The Reference Group for the financial year ended December 31, 2011 was selected in October 2011 and these data are based on their most recently completed fiscal year at such time.

The Reference Group used in respect of the financial year ended December 31, 2011 was composed of the following companies: Acadia Pharmaceuticals Inc.; Acorda Therapeutics Inc.; Array Biopharma Inc.; BioSanté Pharmaceuticals, Inc.; Cell Therapeutics Inc.; Enzon Pharmaceuticals Inc.; Genomic Health Inc.; Ista Pharmaceuticals Inc.; Ligand Pharmaceuticals Inc.; Neurocrine Biosciences Inc.; Nps Pharmaceuticals Inc.; OncoGenex Pharmaceuticals Inc.; Onyx Pharmaceuticals, Inc.; Salix Pharmaceuticals Ltd; Savient Pharmaceuticals Inc.; and Xoma Ltd.

Positioning

The Company s compensation policy is for executive compensation to be generally aligned with the 50th percentile of the Reference Group. The Governance Committee uses discretion and judgment when determining compensation levels as they apply to a specific executive officer. Individual compensation may be positioned above or below median, based on individual experience and performance or other criteria deemed important by the Governance Committee. The total cash target payment for the Company s executive officers generally falls within the 50th percentile competitive range of the Reference Group.

Compensation Elements

An executive compensation policy has been established to acknowledge and reward the contributions of the executive officers to our success and to ensure competitive compensation, in order that we may benefit from the expertise required to pursue our objectives.

Our executive compensation policy is comprised of both fixed and variable components. The variable components include equity and non-equity incentive plans. Each compensation component is intended to serve a different function, but all elements are intended to work in concert to maximize both company and individual performance by establishing specific, competitive operational and corporate goals and by providing financial incentives to employees based on their level of attainment of these goals.

Our current executive compensation program is comprised of the following four basic components:

(i) base salary;

(ii) non-equity incentives consisting of a cash bonus linked to both individual and corporate performance;

(iii) long-term compensation consisting of our stock option plan established for the benefit of our directors, executive officers and employees (the Stock Option Plan); and

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(iv) other elements of compensation consisting of benefits, perquisites and retirement benefits.

Base Salary

Salaries of our executive officers are established based on a comparison with competitive benchmark positions. The starting point to determine executive base salaries is the median of executive salaries in the Reference Group.

In determining individual base salaries, the Governance Committee takes into consideration individual circumstances that may include the scope of an executive s position, the executive s relevant competencies or experience and retention risk. The Governance Committee also takes into consideration the fulfillment of our corporate objectives as well as the individual performance of the executive.

Short-Term Non-Equity Incentive Compensation

The short-term non-equity incentive compensation plan sets out the allocation of incentive awards based on the financial results and the advancement of the Company s product pipeline and strategic objectives.

In the case of executive officers, a program is designed to maximize both corporate and individual performance by establishing specific operational, clinical, regulatory, financial and corporate goals and to provide financial incentives to executive officers based on their level of attainment of these goals. The granting of incentives requires the approval of both the Governance Committee and the Board and is based upon an assessment of each individual s performance, as well as the performance of the Company. The underlying objectives are set at the end of each financial year as part of the annual review of corporate strategies.

For the financial year ended December 31, 2011, the Governance Committee recommended, and the Board approved, in the best interests of the Company, that, to the extent earned, a maximum of 70% of an executive officers maximum bonus be paid in cash in respect of the year 2011 (paid in early 2012), while the remainder of any earned bonus be payable in the form of stock options (as had been done in 2010 as to 50% of any earned bonus and in 2009 as to 100% of any earned bonus), whereby US$1.00 generally equals one stock option, as adjusted based on each senior executive s individual performance, such that, on a dollar-for-dollar basis, the US$ amount of the non-cash bonus was determined to be paid in the form of stock options to vest in equal one-third tranches at six-month intervals, with the first one-third to vest on the six-month anniversary of the date of grant, in order to allow these grants to serve their purpose as partial replacement for annual cash bonuses. For the Company s European executives, the number of options was grossed-up by a multiple of 1.35 to reflect the then prevailing US$ to exchange rate. See Summary of the Stock Option Plan below for more details on the Stock Option Plan.

In making decisions related to the short-term non-equity incentive compensation for the Named Executive Officers, other than the President and Chief Executive Officer, the Governance Committee concluded as follows, based on the goals and results for 2011, as described in Section Risk Assessment of Executive Compensation Program .

Mr. Turpin s 2011 goals were aligned with the Company s overall objectives, with an emphasis on supporting attainment of the financial objectives. Based upon results achieved, the Governance Committee determined that Mr. Turpin s individual performance exceeded expectations as follows: completed two successful At-the-Market Financings (ATMs) generating proceeds in excess of $36 million as a consequence of which the Company completed 2011 with cash and cash equivalents exceeding significantly the budget amount, and contributed significantly to the achievement of the Company s various goals set in the area of Investor Relations. The Compensation Committee determined that Mr. Turpin s contributions to the achievement of the Company s goals merited a cash bonus in an amount of $80,509 and an equity-based bonus in an amount of $34,125 paid through the granting of 34,125 stock options based on 2011 performance.

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Mr. Seeber s 2011 goals were aligned with the Company s overall objectives, with an emphasis on the achievement of the Company s strategic milestones. Based upon results achieved, the Governance Committee determined that Mr. Seeber s individual performance contributed significantly to the achievement of the Company s various strategic milestones, including in respect of the various goals set in the areas of Business Development/Alliance Management and Human Resources. The Governance Committee determined that Mr. Seeber s contributions to the achievement of the Company s goals merited a cash bonus in an amount of $82,818 and an equity-based bonus in an amount of $34,425 paid through the granting of 34,425 stock options based on 2011 performance.

Dr. Blake s 2011 goals were aligned with the Company s overall objectives, with an emphasis on overseeing and supporting the attainment of the Company s clinical and regulatory objectives. Based upon results achieved, the Governance Committee determined that Mr. Blake s individual performance exceeded expectations, particularly in respect of the advancement of the product pipeline and the optimization of the Company s regulatory strategy with lead authorities. The Governance Committee determined that Mr. Blake s contributions to the achievement of the Company s goals merited a cash bonus in an amount of $89,670 and an equity-based bonus in an amount of $38,430 paid through the granting of 38,430 stock options based on 2011 performance.

Mr. Pellicione s 2011 goals were aligned with the Company s overall objectives, with an emphasis on overseeing and supporting the attainment of the Company s clinical and regulatory objectives. Based upon results achieved, the Governance Committee determined that Mr. Pellicione s individual performance exceeded expectations, particularly in respect of the advancement of the product pipeline and the optimization of the Company s regulatory strategy with lead authorities. The Governance Committee determined that Mr. Pellicione s contributions to the achievement of the Company s goals merited a cash bonus in an amount of $77,739 and an equity-based bonus in an amount of $33,316 paid through the granting of 33,316 stock options based on 2011 performance.

For the financial year ended December 31, 2011, cash bonuses paid to all of our executive officers under our short-term non-equity incentive compensation plan represented 70% of the target payout established by the Governance Committee under such plan. Similarly, stock options granted to our executive officers under the same short-term non-equity incentive compensation plan, in partial replacement of the remainder of any earned cash bonuses, represented 30% of the target payout previously established by the Governance Committee.

Long-Term Equity Compensation Plan of Executive Officers

The long-term component of the compensation of the Company s executive officers is based exclusively on the Stock Option Plan, which permits the award of a number of options based on the contribution of the officers and their responsibilities. To encourage retention and focus management on developing and successfully implementing the continuing growth strategy of the Company, stock options generally vest over a period of three years, with the first one-third to vest on the one-year anniversary of the date of grant; however, as mentioned in the section above, the vesting schedule for certain of the options granted to senior executives in the financial years ended December 2011, 2010 and 2009 was accelerated from three years to 18 months since the grants were intended to serve as a partial replacement for a certain portion of earned cash bonuses. Stock options are usually granted to executive officers in December of each year.

For the financial year ended December 31, 2011, the Governance Committee recommended, and the Board approved, in the best interests of the Company, that stock options be granted to executive officers under the long-term equity compensation plan, such stock options to vest according to the vesting schedule set forth in the Company s Stock Option Plan. The Governance Committee considered the performance of the Company and each executive in determining the appropriate level of equity incentive opportunity granted to each executive officer under the long-term equity compensation program in 2011. The Company and the Governance Committee believe that an equitable granting of stock options to senior executives is an important element of the Company s overall compensation philosophy with a view to ensuring that senior management works to ensure the long-term success and viability of the Company s business, particularly in light of the length of time required for a biopharmaceutical company such as Aeterna Zentaris to advance its product pipeline from pre-clinical to clinical through the commercialization stages of development.

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Based on the foregoing, the Governance Committee approved stock option awards as part of the long-term incentive compensation plan to each NEO on December 7, 2011 as follows:

	Stock Options	
	Number granted	Exercise price
Named Executive Officer		
Engel, Juergen	200,000	US$1.74
Turpin, Dennis	70,000	US$1.74
Seeber, Matthias	70,000	US$1.74
Blake, Paul	70,000	US$1.74
Pelliccione, Nicholas J.	70,000	US$1.74

Summary of the Stock Option Plan

We established the Stock Option Plan in order to attract and retain directors, executive officers and employees, who will be motivated to work towards ensuring the success of the Company. The Board has full and complete authority to interpret the Stock Option Plan, to establish applicable rules and regulations and to make all other determinations it deems necessary or useful for the administration of the Stock Option Plan, provided that such interpretations, rules, regulations and determinations are consistent with the rules of all stock exchanges and quotation systems on which our securities are then traded and with all relevant securities legislation.

Individuals eligible to participate under the Stock Option Plan are determined from time to time by either the Board or the Governance Committee.

Options granted under the Stock Option Plan may be exercised at any time within a maximum period of ten years following the date of their grant (the Outside Expiry Date). The Board or the Governance Committee, as the case may be, designates, at its discretion, the individuals to whom stock options are granted under the Stock Option Plan and determines the number of common shares covered by each of such option grants, the grant date, the exercise price of each option, the expiry date, the vesting schedule and any other matter relating thereto, in each case in accordance with the applicable rules and regulations of the regulatory authorities. The price at which the common shares may be purchased may not be lower than the greater of the closing prices of the common shares on the TSX or NASDAQ, as applicable, on the last trading day preceding the date of grant of the option. Options granted under the Stock Option Plan generally vest in equal tranches over a three-year period (one-third each year, starting on the first anniversary of the grant date) or as otherwise determined by the Board or the Governance Committee, as the case may be, although, as described above, under section Short-Term Non-Equity Incentive Compensation , a certain number of stock options granted to executive officers since 2009 have an accelerated vesting schedule of 18 months.

Unless the Board or the Governance Committee decides otherwise, option holders cease to be entitled to exercise their options under the Stock Option Plan: (i) immediately, in the event an option holder who is an officer or employee resigns or voluntarily leaves his or her employment with the Company or one of its subsidiaries or the employment with the Company or one of its subsidiaries is terminated with cause and, in the case of an optionee who is a non-employee director of the Company or one of its subsidiaries, the date on which such optionee ceases to be a member of the relevant board of directors; (ii) six months following the date on which employment is terminated as a result of the death of an option holder who is an officer or employee and, in the case of an optionee who is a non-employee director of the Company or one of its subsidiaries, six months following the date on which such optionee ceases to be a member of the relevant board of directors by reason of death; (iii) 30 days following the date on which an option holder s employment with the Company or any of its subsidiaries is terminated for a reason other than those mentioned in (i) or (ii) above including, without limitation, upon the disability, long-term illness, retirement or early retirement of the option holder; and (iv) where the option holder is a service supplier, 30 days following the date on which such option holder ceases to act as such, for any cause or reason (each, an Early Expiry Date).

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The Stock Option Plan also provides that, if the expiry date of an option(s) (whether an Early Expiry Date or an Outside Expiry Date) occurs during a blackout period or within the seven business days immediately after a blackout period imposed by the Company, the expiry date will be automatically extended to the date that is seven business days after the last day of the blackout period. For the purposes of the foregoing, blackout period means the period during which trading in the Company s securities is restricted in accordance with its corporate policies.

Option holders may not assign their options (nor any interest therein) other than by will or in accordance with the applicable laws of estates and succession.

In the event that, at any time, an offer to purchase is made to holders of all our common shares, notice of such offer shall be given by the Company to each optionee and all unexercised options will become exercisable immediately at their respective exercise prices, but only to the extent necessary to enable optionees to tender their common shares in response to such offer.

The Stock Option Plan currently provides that the following amendments may be made to the plan upon approval of each of the Board and our shareholders as well as receipt of all required regulatory approvals:

any amendment to Section 3.2 of the Stock Option Plan (which sets forth the limit on the number of options that may be granted to insiders) that would have the effect of permitting, without having to obtain shareholder approval on a disinterested vote at a duly convened shareholders meeting, the grant of any option(s) under the Stock Option Plan otherwise prohibited by Section 3.2;

any amendment to the number of securities issuable under the Stock Option Plan (except for certain permitted adjustments, such as in the case of stock splits, consolidations or reclassifications);

any amendment which would permit any option granted under the Stock Option Plan to be transferable or assignable other than by will or in accordance with the applicable laws of estates and succession;

the addition of a cashless exercise feature, payable in cash or securities, which does not provide for a full deduction of the number of underlying securities from the Stock Option Plan reserve;

the addition of a deferred or restricted share unit component or any other provision which results in employees receiving securities while no cash consideration is received by the Company;

with respect to any option holder whether or not such option holder is an insider and except in respect of certain permitted adjustments, such as in the case of stock splits, consolidations or reclassifications:

- any reduction in the exercise price of any option after the option has been granted, or

- any cancellation of an option and the re-grant of that option under different terms;

any extension to the term of an option beyond its Outside Expiry Date to an option holder who is an insider (except for extensions made in the context of a blackout period);

any amendment to the method of determining the exercise price of an option granted pursuant to the Stock Option Plan;

the addition of any form of financial assistance or any amendment to a financial assistance provision which is more favourable to employees; and

any amendment to the foregoing amending provisions requiring Board, shareholder and regulatory approvals.

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The Stock Option Plan further currently provides that the following amendments may be made to the Stock Option Plan upon approval of the Board and upon receipt of all required regulatory approvals, but without shareholder approval:

amendments of a housekeeping or clerical nature or to clarify the provisions of the Stock Option Plan;

amendments regarding any vesting period of an option;

amendments regarding the extension of an option beyond an Early Expiry Date in respect of any option holder, or the extension of an option beyond the Outside Expiry Date in respect of any option holder who is a non-insider of the Company;

adjustments to the number of issuable common shares underlying, or the exercise price of, outstanding options resulting from a split or a consolidation of the common shares, a reclassification, the payment of a stock dividend, the payment of a special cash or non-cash distribution to our shareholders on a pro rata basis provided such distribution is approved by our shareholders in accordance with applicable law, a recapitalization, a reorganization or any other event which necessitates an equitable adjustment to the outstanding options in proportion with corresponding adjustments made to all outstanding common shares;

discontinuing or terminating the Stock Option Plan; and

any other amendment which does not require shareholder approval under the terms of the Stock Option Plan.

The maximum number of common shares issuable under the Stock Option Plan is fixed at 11.4% of the issued and outstanding common shares at any given time, which, as at March 27, 2012, represented 12,401,760 common shares. There are currently 7,497,634 options outstanding under the Stock Option Plan representing approximately 6.9% of all issued and outstanding common shares. Under the Stock Option Plan, (i) the number of securities issued to insiders, at any time, or issuable within any one-year period, under all of the Company s security-based compensation arrangements, cannot exceed 10% of the Company s issued and outstanding securities and (ii) no single option holder may hold options to purchase, from time to time, more than 5% of the Company s issued and outstanding common shares.

Outstanding Option-Based Awards and Share-Based Awards

The following table shows all awards outstanding to each of our Company s President and Chief Executive Officer, the Chief Financial Officer and our three (3) other most highly compensated executive officers of the Company during the most recently completed financial year (collectively, the Named Executive Officers) as at December 31, 2011:

	Option-based Awards					Share-based Awards		
Name	Issuance Date (mm-dd-yyyy)	Number of Securities Underlying Unexercised Options[1] (#)	Option Exercise Price (CAN$ or US$)	Option Expiration Date (mm-dd-yyyy)	Value of Unexercised In-the-money Options[2] (CAN$ or US$)	Issuance Date	Number of Shares or Units of shares that have Not Vested (#)	Market or Payout Value of Share-based Awards that have Not Vested ($)
Engel, Juergen	02-20-2003	60,000	CAN$2.43	12-31-2012				

12-11-2003	60,000	CAN$1.74	12-10-2013	
12-14-2004	100,000	CAN$5.83	12-13-2014	
12-13-2005	50,000	CAN$3.53	12-12-2015	
01-04-2007	50,000	CAN$4.65	01-03-2017	
12-11-2007	50,000	CAN$1.82	12-10-2017	
11-14-2008	200,000	CAN$0.65	11-13-2018	CAN$182,000
12-08-2008	75,000	CAN$0.55	12-08-2018	CAN$75,750

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Name	Option-based Awards					Share-based Awards		
	Issuance Date (mm-dd-yyyy)	Number of Securities Underlying Unexercised Options[1] (#)	Option Exercise Price (CAN$ or US$)	Option Expiration Date (mm-dd-yyyy)	Value of Unexercised In-the-money Options[2] (CAN$ or US$)	Issuance Date	Number of Shares or Units of shares that have Not Vested (#)	Market or Payout Value of Share-based Awards that have Not Vested ($)
	12-09-2009	165,000	CAN$0.95	12-08-2019	CAN$100,650			
	12-08-2010	222,750	CAN$1.52	12-07-2020	CAN$8,910			
	12-07-2011	267,000	US$1.74	12-06-2021				
Turpin, Dennis	11-01-2002	90,000	CAN$0.94	10-31-2012	CAN$55,800			
	12-16-2002	50,000	CAN$3.68	12-15-2012				
	12-11-2003	60,000	CAN$1.74	12-10-2013				
	12-14-2004	90,000	CAN$5.83	12-13-2014				
	12-13-2005	50,000	CAN$3.53	12-12-2015				
	01-04-2007	50,000	CAN$4.65	01-03-2017				
	12-11-2007	50,000	CAN$1.82	12-10-2017				
	12-09-2009	115,000	CAN$0.95	12-08-2019	CAN$70,150			
	12-08-2010	56,850	CAN$1.52	12-07-2020	CAN$2,274			
	12-07-2011	104,125	US$1.74	12-06-2021				
Blake, Paul	07-27-2007	45,000	US$3.05	07-26-2017				
	12-11-2007	50,000	US$1.82	12-10-2017				
	12-08-2008	50,000	CAN$0.55	12-08-2018	CAN$50,500			
	12-09-2009	110,000	CAN$0.95	12-08-2019	CAN$67,100			
	12-08-2010	64,050	CAN$1.52	12-07-2020	CAN$2,562			
	12-07-2011	108,430	US$1.74	12-06-2021				
Seeber, Matthias	02-20-2003	15,000	CAN$2.43	12-31-2012				
	12-11-2003	45,000	CAN$1.74	12-10-2013				
	12-14-2004	50,000	CAN$5.83	12-13-2014				
	12-13-2005	40,000	CAN$3.53	12-12-2015				
	01-04-2007	30,000	CAN$4.65	01-03-2017				
	12-11-2007	25,000	CAN$1.82	12-10-2017				
	12-08-2008	30,000	CAN$0.55	12-08-2018	CAN$30,300			
	12-09-2009	46,667	CAN$0.95	12-08-2019	CAN$28,467			
	12-08-2010	51,975	CAN$1.52	12-07-2020	CAN$2,079			
	12-07-2011	104,425	US$1.74	12-06-2021				
Pelliccione, Nicholas J.	05-07-2007	25,000	US$3.96	05-06-2017				
	12-11-2007	50,000	US$1.82	12-10-2017				
	12-08-2008	20,000	CAN$0.55	12-08-2018	CAN$20,200			
	12-09-2009	60,000	CAN$0.95	12-08-2019	CAN$36,600			
	12-08-2010	50,000	CAN$1.52	12-07-2020	CAN$2,000			
	12-07-2011	103,316	US$1.74	12-06-2021				

(1) The number of securities underlying unexercised options represents all awards outstanding at December 31, 2011.

(2) Value of unexercised in-the-money options at financial year-end is calculated based on the difference between the closing prices of the common shares on the TSX or NASDAQ, as applicable, on the last trading day of the year (December 30, 2011) of CAN$1.56 and US$1.54, respectively, and the exercise price of the options, multiplied by the number of unexercised options.

There are no vested share-based awards that have not yet been paid out or distributed.

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Incentive Plan Awards Value Vested or Earned During the Year

The following table shows the incentive plan awards value vested or earned for each Named Executive Officer for the financial year ended December 31, 2011.

Name	Option-based awards Value vested during the year[1] ($)	Share-based awards Value vested during the year ($)	Non-equity incentive plan compensation - Value earned during the year ($)
Engel, Juergen	251,776		160,764
Turpin, Dennis	73,461		80,509
Seeber, Matthias	83,158		82,818
Blake, Paul	92,499		89,670
Pelliccione, Nicholas J.	52,840		77,739

(1) The amount represents the aggregate dollar value that would have been realized if the options had been exercised on the vesting date, based on the difference between the closing price of the common shares on the TSX or NASDAQ, as applicable, and the exercise price on such vesting date.

Other Forms of Compensation

Benefits and Perquisites

Our executive employee benefits program also includes life, medical, dental and disability insurance. Perquisites consist of a car allowance and human resources counselling. These benefits and perquisites are designed to be competitive overall with equivalent positions in comparable North American organizations in the life sciences industry.

Pension Plan

One of our Named Executive Officers, namely Dr. Juergen Engel, the President and Chief Executive Officer, participates in a non-contributory defined benefit pension plan. Benefits payable under this plan correspond to 40% of the executive officer s average salary of the last twelve months during the first five working years after initial participation in this plan and increase by 0.4% for each additional year of employment.

As the normal retirement age is 65 years, first payments under the pension plan were made to Dr. Engel as of September 1, 2010. The following table shows total annual pension benefits payable to Dr. Engel pursuant to this plan. Upon the death of a participant, the surviving spouse and/or children of the participant will be entitled to a benefit equal to 60% of the benefits to which such participant was entitled. All benefits payable under this plan are in addition to German governmental social security benefits.

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Defined Benefit Plans Table as at December 31, 2011

Name	Number of years of credited service (#)	Annual benefits payable At year end ($)[1]	Annual benefits payable At age 65 ($)[1]	Accrued obligation at start of year ($)	Compensatory change ($)[1]	Non-compensatory change ($)	Accrued obligation at year end ($)[1][2]
Engel, Juergen	34	202,755	202,755	3,323,028	549,986	(103,977)	3,569,399

(1) By way of exception to other currency conversions in this Circular, all amounts in the above table have been converted from euros to US$ based on the exchange rate on December 31, 2011, which was 1.000 = US$1.2972.

(2) The figure in the column Accrued obligation at year end was further reduced by an amount of $199,638 representing mandatory pension payments made to Dr. Engel during 2011.

Employer Contribution to Employees Retirement Plan

In 2008, the Board approved a plan whereby we would contribute to our employees retirement plans both in Canada (RRSP) and the United States (401(k)) to the extent of 50% of the employee s contribution up to a maximum of $7,750 annually for employees under 50 years old. The plan also includes a contribution for employees over 50 years old up to a maximum of $10,250 for Canadian employees and $11,000 for those in the United States. Employees based in Frankfurt, Germany also benefit from certain employer contributions into the employees pension funds (DUPK/RUK). Our executive officers, including the Named Executive Officers, are eligible to participate in the aforementioned employer-contribution plans to the same extent and in the same manner as all of our other employees.

Summary Compensation Table

The Summary Compensation Table set forth below shows compensation information for the Named Executive Officers for services rendered in all capacities during the financial years ended December 31, 2011, 2010 and 2009.

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SUMMARY COMPENSATION TABLE

Name and principal position	Years	Salary ($)	Share based awards ($)	Option based awards[1] ($)	Non-equity incentive plan compensation Annual incentive plan ($)	Non-equity incentive plan compensation Long-term incentive plans ($)	Pension Value ($)	All other compensation[2] ($)	Total compensation ($)
Engel, Juergen	2011	505,260		336,420	160,764		590,136	214,212[3]	1,806,792
	2010	419,348[4]		265,763	109,395		34,605	68,593[5]	897,704
President and CEO	2009	458,040		79,497			431,110	3,209[6]	971,856
Turpin, Dennis	2011	332,434		131,198	80,509			5,056	549,197
	2010	309,978[4]		67,828	55,169			7,518[7]	440,493
Senior Vice President and CFO	2009	284,700		55,407					340,107
Seeber, Matthias	2011	342,512		131,576	82,818			56,966[6]	613,872
	2010	288,162[4]		62,011	51,051			38,217[6]	439,441
Senior Vice President, Administration and Legal Affairs	2009	306,748		55,407				54,300[6]	416,455
Blake, Paul	2011	370,223		136,622	89,670			11,000[8]	607,515
	2010	359,876[4]		76,418	64,050			11,000[8]	511,344
Senior Vice President and Chief Medical Officer	2009	366,000		52,998				10,250[8]	429,248
Pelliccione, Nicholas J.	2011	321,062		130,178	77,739			11,000[8]	539,979
	2010	311,992[4]		59,655	50,001			11,000[8]	432,648
Senior Vice President Regulatory Affairs and Quality Assurance	2009	317,300		28,908				8,250[8]	354,458

(1) The value of the option-based awards represents the closing price of the common shares on NASDAQ at the date of grant (US$1.74 for options granted on December 7, 2011, CAN$1.52 equivalent to US$1.47 for options granted on December 8, 2010, and CAN$0.95 equivalent to US$0.83 for options granted on December 9, 2009) multiplied by the Black-Scholes factor as at such date (72.414% for options granted on December 7, 2011, 80.921% for options granted on December 8, 2010, and 57.895% for options granted on December 9, 2009) and the number of stock options granted on such dates.

(2) All Other Compensation represents perquisites and other personal benefits which, in the aggregate, amount to $50,000 or more, or are equivalent to 10% or more of a Named Executive Officer s total salary for the financial year ended December 31, 2011. The type and amount of each perquisite, the value of which exceeds 25% of the total value of perquisites, is separately disclosed for each Named Executive Officer, if applicable. In the case of the President and CEO, All Other Compensation also includes mandatory pension payments paid to him commencing in 2010. See note (3) and note (5) below.

(3) Represents mandatory pension payments made to Dr. Engel during 2011.

(4) In 2010, the Named Executive Officers agreed to a voluntary reduction in salary between May 1, 2010 and August 31, 2010.

(5) Represents DUPK/RUK (Germany) employer contributions to Dr. Engel s retirement savings plans from January 1, 2010 to August 31, 2010. The reported amount also includes $67,169 in mandatory pension payments made to Dr. Engel after attaining age 65 commencing on September 1, 2010 for the remainder of 2010. See Section Pension Plan , above.

(6) Represents DUPK/RUK (Germany) employer contributions to Dr. Engel s and Mr. Seeber s retirement savings plans.

(7) Represents RRSP employer contribution to Mr. Turpin s retirement savings plan.

(8) Represents 401(k) employer contributions to Messrs. Blake s and Pelliccione s retirement savings plans.

Compensation of the Chief Executive Officer

The compensation of the President and Chief Executive Officer is governed by the Company s executive compensation policy described in Section Compensation of Executive Officers , and the President and Chief Executive Officer participates together with the other Named Executive Officers in all of the Company s incentive plans.

Dr. Engel s total earned salary for 2011 was $505,260, which places him approximately 1.03 times the 50th percentile in relation to the companies in the Reference Group.

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Based upon the results attained versus established goals for fiscal 2011, as described in Section Short-Term Non-Equity Incentive Compensation the Governance Committee determined that the individual performance of Prof. Dr. Engel exceeded expectations in a number of key strategic areas, including in respect of the various goals set in the areas of Business Development and Investor Relations. See Section Risk Assessment of Executive Compensation Program above for more details on such goals.

Based on the foregoing, the Governance Committee recommended, and the Board approved, in the best interests of the Company given its financial situation, and in light of the Company s and the CEO s performance in 2011 as well as the fact that in the previous two years, the payment of cash bonuses was limited, that a maximum of 70% of the CEO s bonus under the short-term incentive plan be paid in cash in respect of the year 2011 (paid in early 2012), while the remainder of the bonus be payable in the form of stock options based on his individual performance. Thus, Prof. Dr. Engel received 70% of his target cash bonus for 2011, amounting to $160,764, for his performance in the context of the Company s objectives and was awarded a grant of 67,000 stock options on December 7, 2011 (at an exercise price of US$1.74) for his exceptional performance in leading the Company to attain its objectives in 2011. The terms of such stock options grant provide for accelerated vesting conditions, in order to allow these stock options to serve their purpose as a partial replacement for the remaining 30% of the CEO s 2011 bonus that would otherwise have been paid in cash. See Section Short-Term Non-Equity Incentive Compensation above for more details on such grants.

For the financial year ended December 31, 2011, the Governance Committee also recommended, and the Board approved, that 200,000 stock options be granted to the President and Chief Executive Officer under the long-term equity compensation plan, such stock options to vest in accordance with the vesting schedule set forth in the Company s Stock Option Plan. See Section Long-Term Equity Compensation Plan of Executive Officers Summary of the Stock Option Plan , for a complete description of our Stock Option Plan.

C. Board Practices

Our Articles provide that our Board shall be composed of a minimum of five and a maximum of fifteen directors. Directors are elected annually by our shareholders, but the directors may from time to time appoint one or more directors, provided that the total number of directors so appointed does not exceed one-third of the number of directors elected at the last annual meeting of shareholders. Each elected director will remain in office until termination of the next annual meeting of the shareholders or until his or her successor is duly elected or appointed, unless his or her post is vacated earlier. For information regarding Dr. Engel s employment agreement with the Company, which provides for benefits on termination of his employment, see Item 10.C Material Contracts . None of the other directors are party to any directors service contracts with the Company providing for benefits on termination of employment.

Committees of the Board of Directors

Audit Committee

Our Board has established an Audit Committee and a Governance Committee.

The Audit Committee assists the Board in fulfilling its oversight responsibilities. The Audit Committee reviews the financial reporting process, the system of internal control, the audit process, and the Company s process for monitoring compliance with laws and regulations and with our Code of Ethical Conduct. In performing its duties, the Audit Committee will maintain effective working relationships with the Board, management, and the external auditors. To effectively perform his or her role, each committee member will obtain an understanding of the detailed responsibilities of committee membership as well as the Company s business, operations and risks.

The function of the Audit Committee is oversight and while it has the responsibilities and powers set forth in its charter (incorporated by reference to Exhibit 11.2), it is neither the duty of the committee to plan or to conduct audits or to determine that the Company s financial statements are complete, accurate and in accordance with generally accepted accounting principles, nor to maintain internal controls and procedures.

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The current members of the Audit Committee are Pierre Lapalme, Gérard Limoges and Michael Meyers.

Governance Committee

The mandate of the Governance Committee provides that it is responsible for taking all reasonable measures to ensure that appropriate human resources systems and procedures, such as hiring policies, competency profiles, training policies and compensation structures are in place so that the Company can attract, motivate and retain the quality of personnel required to meet its business objectives.

The Governance Committee also assists the Board in discharging its responsibilities relating to executive and other human resources hiring, assessment, compensation and succession planning matters.

Thus, the Governance Committee recommends the appointment of senior officers, including the terms and conditions of their appointment and termination, and reviews the evaluation of the performance of our senior officers, including recommending their compensation and overseeing risk identification and management in relation to executive compensation policies and practices. The Board, which includes the members of the Governance Committee, reviews the Chief Executive Officer s corporate goals and objectives and evaluates his or her performance and compensation in light of such goals and objectives.

The current members of the Governance Committee are Juergen Ernst, José P. Dorais and Gérard Limoges.

D. Employees

As at March 1, 2012, we had a total of 89 Full Time Equivalents (FTE) (as compared to 87.8 at March 1, 2011 and 99 at March 1, 2010), of which 75.3 are based in Frankfurt, Germany, 5 in New Jersey, United States, and 8.7 in Quebec City, Canada. Of these, 53 are involved in discovery, preclinical, clinical and pharmaceutical development, 9 are involved in regulatory affairs, quality assurance and intellectual property, and 27 are involved in business operations, communications, finance, information technology, human resources, project management and legal affairs. We have agreements with all of our employees covering confidentiality and loyalty, non-competition, and assignment to the Company of all intellectual property rights developed during the employment period.

E. Share ownership

The information in the table below is provided as at March 27, 2012:

Name	No. of common shares owned or held	Percent[(1)]	No. of stock options held[(2)]	No. of currently exercisable options
Marcel Aubut	112,500	*	220,000	143,334
Paul Blake	70,350	*	427,480	297,700
José P. Dorais	0	*	80,000	10,000
Juergen Engel				