



XL

2007 Annual Report

XL647

POTENTIAL INDICATIONS

NON-SMALL CELL LUNG CANCER
BREAST CANCER
HEAD & NECK CANCER
GLIOBLASTOMA
TARGETS EGFR, HER2 & VEGFR

XL184

POTENTIAL INDICATIONS

MEDULLARY THYROID CANCER
NON-SMALL CELL LUNG CANCER
GLIOBLASTOMA
TARGETS MET, RET & VEGFR

XL147

STUDIES

ADVANCED SOLID TUMORS
TARGETS PI3K

XL765

STUDIES

ADVANCED SOLID TUMORS
TARGETS PI3K & mTOR

XL880

POTENTIAL INDICATIONS
PAPILLARY RENAL CELL CARCINOMA
GASTRIC CANCER
HEAD & NECK CANCER
TARGETS MET & VEGFR

XL019

POTENTIAL INDICATIONS
MYELOPROLIFERATIVE DISORDERS
CANCER
INFLAMMATION
TARGETS JAK2

XL281

STUDIES
ADVANCED SOLID TUMORS
TARGETS B-RAF & C-RAF, KEY COMPONENTS OF
THE RAS/RAF/MEK/ERK PATHWAY

XL518


STUDIES
ADVANCED SOLID TUMORS
TARGETS MEK, A KEY COMPONENT OF
THE RAS/RAF/MEK/ERK PATHWAY

MATURING PIPELINE

**XL647, XL184 AND XL880
HAVE GENERATED
ENCOURAGING DATA IN
NON-SMALL CELL LUNG
CANCER, THYROID
CANCER AND RENAL
CANCER, RESPECTIVELY**

**ADDITIONAL TRIALS
ARE PLANNED FOR EACH
OF THESE COMPOUNDS
TO EXPLORE ITS
POTENTIAL IN ADDITIONAL
INDICATIONS**

**PIVOTAL TRIALS, DESIGNED
TO GENERATE DATA IN
SUPPORT OF POTENTIAL
FDA APPROVAL, ARE
PLANNED TO BEGIN THIS
YEAR FOR XL647 AND XL184**



**NON-SMALL CELL LUNG
CANCER IS THE LEADING
CAUSE OF CANCER
DEATH IN WOMEN.**

Our later stage compounds are an example of our integrated development strategy. Each of these compounds has demonstrated activity in patients with specific types of tumors, and we are excited about the potential of each of these compounds.

XL647 is designed specifically to inhibit three validated cancer targets simultaneously (EGFR, HER2, and VEGFR). The compound had encouraging results in patients with non-small cell lung cancer (NSCLC) and is currently being studied in two phase 2 trials in this indication. Additional phase 2 trials in breast cancer, glioblastoma, and head and neck cancer are planned, and we are planning to initiate a pivotal trial in NSCLC later this year.

XL184 inhibits RET, MET and VEGFR. All three of these targets may play a role in thyroid cancer, and to date all evaluable patients with medullary thyroid cancer who have been treated with XL184 have had either stable disease or a partial response as their best response. In addition, decreases in the tumor markers calcitonin and carcinoembryonic antigen were seen in all patients. These are exciting observations, and we plan to initiate a pivotal trial in this indication later this year. Additionally, the compound has substantial potential in lung cancer and other indications, and we have initiated a phase 2 trial of XL184 in patients with NSCLC.

XL880 inhibits MET and VEGFR. It was selected by GSK as part of our collaboration in 2007 and is being moved through clinical development by GSK, although Exelixis retains substantial economic interest in the compound. XL880 demonstrated encouraging data in patients with papillary renal cell cancer and is currently in phase 2 trials for that indication as well as for gastric cancer. We are excited about the potential of this compound and will work with our colleagues at GSK to facilitate its rapid development.

ADVANCING XL COMPOUNDS

THE RAPID ADVANCEMENT
OF OUR CLINICAL
PIPELINE MOVES
US CLOSER TO OUR GOAL
OF DEVELOPING
FIRST-IN-CLASS AND
BEST-IN-CLASS THERAPIES



Throughout 2007 we made substantial progress in advancing our clinical pipeline. Numerous trial results make it clear that we have built a successful drug development infrastructure. As we move through 2008, we intend to make additional strides toward our goal of becoming a leading drug development organization. In addition to our near-term pivotal trial opportunities, we also continue to advance the rest of our pipeline, which comprises compounds that target key drivers of cancer development, progression and resistance.

XL019 is an inhibitor of JAK2, a target that plays a pivotal role in the biology of myeloproliferative disorders. This compound showed evidence of clinical activity with either stabilization or shrinkage of spleen size in several of the myelofibrosis patients treated in a phase 1 clinical trial. We currently are optimizing the dosing regimen to achieve activity and tolerability profiles that could potentially position XL019 as a leading selective JAK2 inhibitor. If we complete this optimization during 2008, we intend to initiate a pivotal trial of XL019 in myelofibrosis by year's end. XL019 is currently not part of any collaboration and is entirely owned by Exelixis.

There are several other phase 1 compounds in our pipeline that we believe have potential to

provide substantial benefit to large numbers of cancer patients. Two of these compounds, **XL147** and **XL765**, principally target the phosphoinositide-3-kinase (PI3K) pathway. Two additional compounds, **XL281** and **XL518**, each target distinct components of the RAS/RAF/MEK/ERK pathway. These two pathways are inappropriately activated in the majority of solid tumors, and we are optimistic that our compounds will be substantial additions to the arsenal that we are bringing to the table to combat cancer. In March 2008, Genentech exercised its option to further develop and commercialize XL518. Exelixis retains a significant economic interest in the success of this compound.

In addition, we brought **XL139**, a small molecule inhibitor of the hedgehog signaling pathway into development. Hedgehog signaling is deregulated in a variety of cancers and the pathway is a promising target for novel cancer therapies. In January 2008, Bristol-Myers Squibb (BMS) exercised its option to co-develop XL139 and Exelixis exercised its option to co-develop and co-commercialize XL139 in the United States. Following potential commercialization, the companies will equally share profits in the United States and Exelixis will be entitled to receive double-digit royalties on product sales in the rest of the world.

The clinical pipeline also includes **XL228** which targets IGF1R, SRC and BCR-ABL, **XL820** which targets KIT, PDGFR and VEGFR, and **XL844** which targets the CHK1 and CHK2 kinases. These compounds may play important roles in chronic myelogenous leukemia, gastrointestinal stromal tumors and resistance to DNA damaging therapies, respectively.

By the end of 2008, we expect that several of our compounds will potentially enter pivotal trials, including XL647, XL184 and XL019. In addition, several of the compounds in phase 1 clinical trials, including XL147, XL765 and XL281, are likely to generate encouraging data and move into phase 2 clinical trials. Furthermore, we expect that XL820 will have progressed in phase 2 evaluation and XL844 and XL228 will have progressed in phase 1 evaluation. Also, XL880, XL139 and XL518 now have the resources of GlaxoSmithKline, BMS and Genentech, respectively, to take them forward in development. Consistent with our performance over the past several years, we expect to file at least three new investigational new drug applications in 2008, further enhancing the depth and breadth of our industry-leading clinical pipeline.

PIPELINE

COMPOUND/TARGET PROFILE	CURRENT TRIALS	ANTICIPATED 2008/2009 ADDITIONAL TRIALS
INHIBITOR OF EGFR, HER2 & VEGFR XL647 ¹	PHASE 2 <ul style="list-style-type: none"> • Non-small cell lung cancer: first-line • Non-small cell lung cancer: progressed after EGFRi 	PHASE 2 <ul style="list-style-type: none"> • Breast cancer PIVOTAL TRIALS <ul style="list-style-type: none"> • Non-small cell lung cancer
INHIBITOR OF RET, MET & VEGFR XL184 ²	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors PHASE 1/2 <ul style="list-style-type: none"> • Non-small cell lung cancer 	PHASE 2 <ul style="list-style-type: none"> • Glioblastoma PIVOTAL TRIAL <ul style="list-style-type: none"> • Medullary thyroid cancer
INHIBITOR OF MET & VEGFR XL880 ³	PHASE 2 <ul style="list-style-type: none"> • Papillary renal cell carcinoma • Gastric cancer • Head and neck cancer 	
INHIBITOR OF JAK2 XL019	PHASE 1 <ul style="list-style-type: none"> • Myeloproliferative disorders 	PHASE 1 <ul style="list-style-type: none"> • Polycythemia vera PIVOTAL TRIAL <ul style="list-style-type: none"> • Myelofibrosis
INHIBITOR OF THE PI3K PATHWAY XL147	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors 	PHASE 2 <ul style="list-style-type: none"> • Single agent PHASE 1B/2 <ul style="list-style-type: none"> • Combined with chemotherapy • Combined with other targeted agents
INHIBITOR OF PI3K & mTOR XL765	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors 	PHASE 2 <ul style="list-style-type: none"> • Single agent PHASE 1B/2 <ul style="list-style-type: none"> • Combined with chemotherapy • Combined with other targeted agents
INHIBITOR OF B-RAF & C-RAF, KEY COMPONENTS OF THE RAS/RAF/MEK/ERK PATHWAY XL281 ²	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors 	PHASE 2 <ul style="list-style-type: none"> • Solid tumors
INHIBITOR OF MEK, A KEY COMPONENT OF THE RAS/RAF/MEK/ERK PATHWAY XL518 ⁴	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors 	
INHIBITOR OF KIT, VEGFR & PDGFR XL820 ²	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors PHASE 2 <ul style="list-style-type: none"> • Gastrointestinal stromal tumors 	
INHIBITOR OF IGF1R, SRC & BCR-ABL XL228 ²	PHASE 1 <ul style="list-style-type: none"> • Chronic myelogenous leukemia • Advanced malignancies 	
INHIBITOR OF CHK1 & CHK2 XL844 ²	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors in combination with gemcitabine 	
INHIBITOR OF THE HEDGEHOG PATHWAY XL139 ⁵	IND ENABLED	PHASE 1 <ul style="list-style-type: none"> • Advanced solid tumors

THE BREADTH OF TARGETING A BROAD SPECTRUM OF CRITICAL CANCER-RELATED PATHWAYS.

THE DEPTH TO SELECT ONLY THOSE COMPOUNDS WITH FIRST- OR BEST-IN-CLASS POTENTIAL.

THE BALANCE TO MANAGE RISK AND REAP THE REWARDS OF OUR EFFORTS.

THE XL COMPOUNDS: WORKING TO BUILD THE BIGGEST NAME IN CANCER THERAPY.

PHASE 2

XL647¹ targets EGFR, HER2 and VEGFR, which are validated targets in the treatment of diverse solid tumors, including non-small cell lung cancer, breast cancer, head and neck cancer and glioblastoma.

XL184² targets RET, which is implicated in several thyroid cancers, MET, which contributes to resistance to EGFR inhibitors in NSCLC, and VEGFR.

XL880³ targets MET and VEGFR. It is the first MET inhibitor to demonstrate proof-of-concept in a phase 2 trial.

XL820² targets KIT, VEGFR and PDGFR. KIT is mutationally activated in gastrointestinal stromal tumors and in some subtypes of melanoma. VEGFR promotes angiogenesis and PDGFR helps to stabilize new blood vessels. PDGFR also contributes to cancer cell proliferation.

PHASE 1

XL019 specifically targets JAK2 without inhibiting other JAK family members. Activating mutations in JAK2 occur frequently in a variety of myeloproliferative disorders.

XL147 targets PI3K. Activation of PI3K is a frequent event in human tumors, promoting cell growth, survival and resistance to chemotherapy and radiotherapy.

XL765 targets PI3K and mTOR. mTOR is a kinase that constitutes an important signaling node downstream of PI3K. mTOR is frequently activated in human tumors and plays a central role in promoting tumor cell growth.

XL281² targets B-RAF and C-RAF, key components of the RAS/RAF/MEK/ERK kinase signaling pathway. Mutations leading to activation of B-RAF occur in many human cancers, including melanoma, colon cancer and thyroid cancer.

XL518⁴ targets MEK, a component of the RAS/RAF/MEK/ERK kinase signaling pathway. Activating mutations in this pathway occur in many human tumors.

XL228² targets IGF1R, SRC and BCR-ABL (including the T315I mutant form), which drive cell proliferation. The T315I mutant form of BCR-ABL is resistant to approved BCR-ABL inhibitors.

XL844² targets the CHK1 and CHK2 kinases, which may reduce the efficacy of cancer therapies that work via DNA damage. XL844 may improve the efficacy of such therapies.

IND ENABLED

XL139⁵ targets the hedgehog signaling pathway. The pathway is deregulated in basal cell carcinoma, medulloblastoma, glioblastoma, multiple myeloma, pancreatic carcinoma and a variety of other cancers, and may play a key role in the proliferation and drug resistance of cancer stem cells.

¹ Out-licensed to Symphony Evolution Inc. and is subject to a repurchase option.

² Pursuant to a product development and commercialization agreement between Exelixis and GlaxoSmithKline (GSK), GSK has the option to elect and further develop and commercialize up to two additional compounds in our pipeline, which may include XL184, XL281, XL820, XL228 and XL844.

³ GSK has licensed exclusive rights to further develop and commercialize XL880.

⁴ Genentech has exercised its option under the agreement to further develop and commercialize XL518. Exelixis is responsible for the phase 1 clinical trial until the point that a maximum tolerated dose (MTD) is determined. After MTD is achieved, Genentech will be responsible for completing the phase 1 clinical trial and subsequent clinical development. Exelixis has the option to co-promote in the United States and is entitled to receive an initial equal share in profits in the United States, which will decrease as sales increase. The Company will receive royalties on any sales of the product that may be commercialized outside the United States.

⁵ XL139 is exclusively licensed to Bristol-Myers Squibb (BMS) and BMS has primary responsibility for the further development and commercialization of the compound. We exercised our option under our collaboration agreement with BMS to co-develop and co-commercialize XL139 in the United States. As a result, we will be responsible for 35% of all development costs related to clinical trials intended to support regulatory approval in both the United States and the rest of the world, with the remaining 65% to be paid by BMS. We will be entitled to receive double-digit royalties on product sales outside of the United States.

KNOWLEDGE

OUR UNDERSTANDING OF
CANCER BIOLOGY IS
THE FOUNDATION FOR
OUR INNOVATIVE
APPROACHES
TO INHIBITING
CLINICALLY RELEVANT
PATHWAYS THAT DRIVE
CANCER CELL GROWTH,
PROLIFERATION, SURVIVAL
AND RESISTANCE



At Exelixis, we believe that the basis for generating important new drugs is scientific rigor and excellence. The cutting edge technologies we use are tools, not ends in themselves. It is the thoughtful application of these powerful new technologies to answer questions about the molecular basis of disease that is central to our success. Our understanding of the biology of cancer has increased tremendously in recent years, and the increased understanding already is leading to better therapies for many patients. However, most types of cancer remain intractable, and many questions remain to be answered.

At its core, research is the exploration for knowledge and understanding – knowledge that describes how a compound binds to its target, understanding the role that specific targets play in disease, knowledge of which tumors and which patients are likely to benefit from a given approach. We look beyond the manifestation of illness to see its molecular basis, matching the

inhibition profiles of specific diseases in our compounds to the biology of defined patient populations. We recognize that our ability to treat disease is enhanced by elucidating the pathways and signals that regulate fundamental cellular processes.

We seek to integrate the advances made around the world with the particular insights made at Exelixis. We listen to physicians who are intimately familiar with the strengths and weaknesses of today's cancer therapies, to patients who need new treatment options and to the regulators and competitors who help shape the commercial landscape.

Knowledge – of biology, medical need, the market and the challenges of drug development – drives our clinical strategies, our approach to pipeline management and our partnership negotiations. But perhaps the most important thing we know is that our true worth will be measured in the lives we help to preserve and extend.

CREATING VALUE

**OUR CLINICAL PIPELINE,
R&D ENGINE AND QUALITY
PARTNERSHIPS ARE
TODAY'S FOUNDATION
FOR TOMORROW'S
SUCCESS**



We have amassed a portfolio of diverse assets with an eye on enhancing our near-term value while building a world-class commercial oncology franchise for the future.

Our primary asset is our broad and deep clinical pipeline. Each of the compounds in our pipeline has potential as a first-in-class or best-in-class therapy for patients with a variety of cancers. Today there are 13 compounds discovered by Exelixis in clinical development. Most of the compounds in our pipeline are being taken forward by us, and some are being moved through the clinic by partners such as GlaxoSmithKline (GSK), Bristol-Myers Squibb (BMS) and Genentech. Collectively, these compounds comprise a safeguard against the attrition and competition that is inevitable in our industry.

Underpinning our pipeline is our robust drug discovery engine. Combining our library of over four million compounds with state-of-the-art

screening capabilities and exhaustive compound characterization, we continuously generate a pool of high-quality leads. This pool is sufficiently large that, even after applying stringent selection criteria for a wide variety of characteristics including safety, potency and specificity, we have been able to file at least three high-quality investigational new drug applications each year.

Finally, our strategy is to leverage the strength of our extensive data and the broad potential of our compounds to establish quality partnerships that generate near-term revenue, and focus external resources on advancing additional compounds through development, while retaining long-term rights to those compounds that succeed. XL880, XL139 and XL518 now have the resources of GSK, BMS and Genentech, respectively, behind them. In addition, potential cardiovascular and metabolic disease therapies are advancing with Wyeth, Daiichi-Sankyo and BMS.

**TO OUR
STOCKHOLDERS**

**IN 2007, WE ACHIEVED
NOTABLE CLINICAL
SUCCESS, ADVANCED
OUR COMPOUNDS
RAPIDLY THROUGH
DEVELOPMENT, AND
CAREFULLY MANAGED
OUR FINANCES**

**WE ARE POSITIONED
WELL FOR CONTINUED
SUCCESS IN 2008**



Left to right: **Frank L. Karbe** Executive Vice President and Chief Financial Officer **Pamela A. Simonton, JD, LLM** Executive Vice President and General Counsel **Lupe M. Rivera, SPHR, CCP** Senior Vice President, Operations **George A. Scangos, PhD** President and Chief Executive Officer **Gisela M. Schwab, MD** Executive Vice President and Chief Medical Officer **Michael M. Morrissey, PhD** President of Research and Development **Peter Lamb, PhD** Senior Vice President, Discovery Research and Chief Science Officer

Our vision is to build a world-class biotechnology company. We believe that we can do so by insisting on excellence, focus, execution, and efficiency in everything that we do. Our strategy has been different from most biotechnology companies. From early on in our history, we have worked towards building one of the top oncology pipelines in the biotechnology and pharmaceutical industries. I believe that we have accomplished that goal. Presently there are 13 compounds discovered by Exelixis that are in clinical development. We are leading clinical development for the majority of these compounds, while others are being moved through the clinic by partners such as GlaxoSmithKline (GSK), Bristol-Myers Squibb Company (BMS), Wyeth, and Genentech. Furthermore, this pipeline is dynamic. Each year we bring several additional compounds into clinical development, and we constantly evaluate our data to ensure that we put our resources behind only those compounds that have real potential to be first-in-class or best-in-class

compounds – compounds that not only can gain approval, but also provide meaningful improvements in therapy for patients.

At Exelixis, we strive to maximize success, not to minimize failure. Our goal is to work efficiently, make data-driven decisions rapidly, and quickly advance promising compounds. The robust pipeline of high-quality compounds that has arisen from this approach gives us realistic opportunities to achieve substantial medical and commercial success, and the encouraging data generated by many of our compounds gives us confidence that we are well on the road to achieving that objective. Clearly, not all of our compounds will reach the market. Despite the increasing understanding of cancer biology and the increasing sophistication of our predictive models, the behavior of drugs in humans is still subject to many factors that are not yet completely understood. The process of drug development is one of gradually removing the risks imposed by these unknown factors in a series of clinical trials. One of our objectives is to reach critical success milestones early in development, and by using the latest approaches to translational

medicine, I believe that we have been able to do so. In this way we are able to rapidly identify high priority compounds that merit aggressive development and focus the majority of our resources on those compounds.

I believe that several of our compounds have successfully achieved these early milestones, including XL647, XL184, XL880, XL765, XL147, XL281, and XL518. Each of these compounds has generated encouraging clinical data and merits further development. Additionally, there are several other compounds, including XL019, XL844, XL820, and XL228, that are moving through clinical development on the way towards those milestones. This is a very compelling pipeline that gives me confidence that Exelixis will, in fact, be a substantial contributor to the treatment of many types of cancer.

Next I'll discuss highlights for some of our leading compounds, but a more complete description of the pipeline can be found on our web site and by listening to one of our many public presentations.

XL647: This compound inhibits EGFR, HER2, and VEGFR. Interim data from a phase 2 trial in a selected population of first-line non-small cell lung cancer (NSCLC) patients were very encouraging. Data from two additional phase 2 trials will be presented at the 2008 American Society of Clinical Oncology (ASCO) Annual Meeting in June. We are currently planning a program of pivotal studies designed to generate data in support of registration for this compound. We plan to begin at least one pivotal trial in NSCLC in 2008.

XL184: XL184 inhibits RET, MET, and VEGFR, a unique inhibition profile that has the potential to position the compound as a first-in-class therapy for a variety of cancers.

In October 2007, we presented encouraging interim data from an ongoing phase 1 trial of XL184. The results were especially compelling in seven patients with metastatic medullary thyroid cancer (MTC) for whom data were available at the time of the presentation. Six of the seven patients had measurable disease, and all exhibited tumor shrinkage. All seven patients experienced a rapid decrease in plasma biomarkers of MTC. These results are remarkable in this population of MTC patients, whose disease is difficult to manage and who have failed multiple prior therapies. Updated data will be presented in June at the ASCO Annual Meeting, and we intend to initiate a pivotal trial in MTC in the second half of 2008. Our strategy is to leverage the positive findings in MTC to establish a rapid and cost-effective path to market, while aggressively advancing development of the compound in larger indications. Toward this end, in January 2008 we initiated a phase 1/2 trial of XL184 in patients with NSCLC who have had progressive disease while on an erlotinib-containing regimen.

XL880: This compound, which targets MET and VEGFR, generated an excellent data set in a phase 2 trial in patients with papillary renal cell cancer (PRC). Additional data from this trial as well as from a phase 2 trial in patients with gastric

cancer will be presented in June at the 2008 ASCO Annual Meeting. In addition to those two tumor types, we believe that the compound has substantial potential in some of the major tumors, including NSCLC. Based on the data and the potential of the compound, GSK exercised its option to further develop and commercialize XL880 in December 2007.

XL019: XL019 inhibits JAK2, a kinase inappropriately activated in the majority of patients with myeloproliferative disorders (MPDs) and deregulated in a number of solid tumors, as well as certain subtypes of lymphoma. We believe that XL019 is the only investigational compound designed to inhibit JAK2 selectively, without impacting normal signaling through the three other members of the JAK family. This inhibition profile is expected to provide clinical benefit in MPDs without inducing immune suppression and thrombocytopenia (low platelet counts), a serious and potentially treatment-limiting side effect associated with non-selective JAK inhibitors.

In December 2007, we presented preliminary data from an ongoing phase 1 trial of XL019 in patients with myelofibrosis. Investigators observed evidence of XL019 clinical activity in several patients available for evaluation. To date, we have observed reduction or stabilization of spleen size, a reduction in the number of cells with activating JAK2 mutations and pharmacodynamic evidence of on-target activity. Importantly, we have not seen any myelosuppression or thrombocytopenia, which have been observed in trials of other, non-selective JAK inhibitors. We believe this is a critical differentiating factor for XL019, as MPD is a chronic disease that requires treatment over a prolonged period of time.

We also have observed symptoms consistent with neuropathy at all doses tested to date, and the onset and severity of neuropathy appeared to be dose related. Because even the lowest administered dose of XL019 appeared to have beneficial activity in the disease, we believe that lower doses and/or alternative dosing schedules may retain activity while reducing or eliminating the neuropathies we have seen. Currently, we are working to identify a dose and schedule that will support efficacy and

safety profiles that could position XL019 as a leading selective JAK2 inhibitor. Once the optimization is completed, we intend to initiate a pivotal trial in myelofibrosis and further studies in polycythemia vera and other indications. We believe this goal can be accomplished by the end of 2008.

XL147, XL765: XL147 and XL765 target the phosphoinositide-3-kinase (PI3K) pathway at different points. They are the leading PI3K inhibitors – last year we presented the first data from humans demonstrating inhibition of the pathway. Inappropriate activation of this pathway clearly plays a significant role in the proliferation of many types of tumors, and in the ability of those tumors to overcome chemotherapy. We are moving these compounds aggressively forward and are having partnering discussions with pharmaceutical companies who have the resources to develop these compounds in multiple indications simultaneously.

XL281, XL518: XL281 and XL518 each target distinct components of the RAS/RAF/MEK/ERK pathway and have potential advantages over competitor's compounds in terms of potency and tolerability. We are excited about the phase 1 data and are aggressively moving these compounds forward.

Managing Our Pipeline for Success In 2007 we filed investigational new drug (IND) applications for four new compounds and initiated five new clinical development programs. As a result of our industry-leading productivity, as we enter 2008, there are 13 compounds discovered by Exelixis in clinical development, and we expect to file at least three new INDs this year.

Our R&D productivity is meaningful only if we have a strategy for managing our pipeline over the long-term. Partnerships are an essential pillar of both our development and financing strategies. Over the course of 2007, we made great progress in our existing partnerships while also establishing additional high-value collaborations. We submitted due diligence packages for XL647, XL880, and XL784 to GSK. We retained

rights to XL647, licensed XL880 rights to GSK, and are evaluating potential third-party partnering opportunities for XL784. We earned a \$35.0 million selection milestone from GSK for XL880 which offset a milestone GSK paid to us in 2005, and we are entitled to receive milestone payments and royalties on any sales of XL880. Under our product and commercialization agreement, GSK may select up to two more compounds from XL820, XL184, XL844, XL281, and XL228.

In January 2007, we announced a co-development agreement with Genentech for XL518, a potential cancer therapy, receiving \$40.0 million upon signing the agreement. In March 2008, Genentech exercised its option to further develop XL518. We received an additional payment of \$3.0 million, and are entitled to receive \$7.0 million more when Genentech commences a phase 2 clinical trial with this compound. After completion of the phase 1 clinical trial, Genentech will be responsible for further development of the compound. We have the option to co-promote in the United States and we are entitled to receive royalties on any sales outside the United States.

Our multiple metabolic disease collaborations are enabling us to advance and derive value from additional compounds. In 2007, we extended our collaboration with BMS for compounds targeting the liver X receptor, receiving an additional \$7.5 million in research funding. We also received a \$5.0 million milestone payment from BMS upon the acceptance of an IND, or its foreign equivalent.

Already in 2008, BMS has exercised its option under our oncology collaboration to develop and commercialize XL139, generating a \$20.0 million selection milestone payment to Exelixis. We have exercised our right to co-develop and co-commercialize the compound in the United States, enabling us to retain significant value in the program over the long-term.

In keeping with our commitment to developing first-in-class or best-in-class therapies, we discontinued development of XL418 and XL999, which did not appear to satisfy these criteria. We also halted development of XL784 in the treatment of diabetic nephropathy after it missed its endpoint in a phase 2 clinical trial. Trends toward improvement were noted in a subset of patients, and we are evaluating opportunities to partner the compound with a company that has expertise to pursue development in this indication.

Four Pillars for Financial Success Our four-pillar strategy – executing in our existing partnerships, establishing new, high-value collaborations, utilizing clinical financing vehicles and tapping the equity markets – provides the resources we need to support quality and productivity. In 2007, we generated approximately \$113.5 million in revenue from our various new and existing collaborations. All of our partnerships also provide additional revenue opportunities in the future, through milestone payments, royalties on sales, or profit sharing.

In 2007, we also enhanced our capital resources through the successful completion of a public offering of common stock, raising net proceeds of \$71.9 million. In addition, during 2007, we received \$19.8 million as a result of the sale of 80.1% of our subsidiary Artemis Pharmaceuticals GmbH to Taconic Farms, and we received \$18.0 million for the sale of selected assets of our subsidiary Exelixis Plant Sciences to Dow AgroSciences' affiliate Agrigenetics, Inc. By continuing to execute on our financing strategy, we finished 2007 with \$299.5 million in cash, cash equivalents and short-term and long-term marketable securities¹. These resources, and our proven ability to access and effectively manage capital, should enable us to continue pursuing a better way to better medicine.

Pulling Ahead in 2008 With a solid cash position at the beginning of 2008, multiple high-quality assets available for partnering, and several opt-in decisions expected under our various collaborations, we are well positioned to see our way through a difficult economic environment. We are constantly evaluating our strategies and options in regard to our assets and the changing economic climate, and as in the past, we expect to react nimbly to changes in the environment going forward.

During the year we expect to have numerous presentations at major medical conferences; I believe that the data will be clinically meaningful and will differentiate our compounds from the competition. Additionally, opt-in decisions are expected from several partners, clarifying our pipeline ownership and potentially generating additional revenue. We also will continue to evaluate high-value collaboration opportunities around some of our proprietary programs.

I am proud of all that we have accomplished over the past year, and I want to take this opportunity to recognize the diligent efforts and heartfelt commitment of everyone on the Exelixis team. Each of us is aware that the stakes become higher as we get closer to our goal of bringing ground-breaking therapies to market, and we feel confident in our ability to continue charting our own path to success. Through innovation, pragmatism, and rigor, we believe we have what it takes to make XL the biggest name in oncology.

We thank you, our stockholders, for your continued support. I very much look forward to updating you on our progress in the months ahead.

Sincerely,



George A. Scangos, PhD
President and Chief Executive Officer

¹ This amount includes investments held by Symphony Evolution, Inc. of \$30.9 million and restricted cash and investments of \$7.2 million.

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SEC FORM 10-K

A copy of the Exelixis annual report on Form 10-K filed with the Securities and Exchange Commission is available free of charge from Exelixis' Corporate Communications Department by calling 650.837.7012.

STOCK INFORMATION

The common stock of Exelixis has traded on the Nasdaq Global Select Market under the symbol "EXEL" since April 11, 2000. No dividends have been paid on the common stock since Exelixis' inception.

COMMON STOCK

The following table sets forth, for the periods indicated, the high and low intraday sales prices for the common stock of Exelixis as reported by the Nasdaq Global Select Market:

Quarter Ended	High	Low
December 31, 2007	\$12.29	\$7.82
September 30, 2007	\$12.37	\$9.40
June 30, 2007	\$12.77	\$9.92
March 31, 2007	\$11.74	\$8.67

BOARD OF DIRECTORS

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Senior Vice President, Operations

D. Ry Wagner, PhD
Vice President, Plant Biotechnology, Exelixis Plant Sciences, Inc.

This annual report and the accompanying letter to stockholders contain statements that are forward-looking, including, without limitation: statements relating to the future development and therapeutic and commercial potential of XL647, XL184, XL880, XL019, XL147, XL765, XL281, XL518, XL228, XL820, XL844, XL139 and Exelixis' other compounds; the anticipated timing of the initiation of clinical trials for XL647, XL184, XL019, XL147, XL765, XL281, XL139 and Exelixis' other compounds; the availability of data related to XL647, XL184, XL880 and Exelixis' other compounds; the anticipated timing of the filing of new investigational new drug applications; potential compound selections by Exelixis' partners; the realization of revenue opportunities from Exelixis' partnerships and product candidates; the sufficiency of Exelixis' cash resources and the timing and success of future business development activities. Words such as "plan," "goal," "may," "would," "will" "could," "expect," "should," "anticipate," "suggest," "intend," "potential," "encouraging" and similar expressions are intended to identify forward-looking statements. These statements are only predictions and are based upon Exelixis' current plans, assumptions, beliefs and expectations. Forward-looking statements involve risks and uncertainties. Exelixis' actual results and the timing of events could differ materially from those anticipated in such forward-looking statements as a result of these risks and uncertainties, which include, without limitation, risks related to: Exelixis' need for additional financing, including Exelixis' ability to enter into new collaborations, continue existing collaborations and receive milestones and royalties derived from future products developed from research efforts under collaborative agreements; Exelixis' arrangement with Symphony Evolution, Inc.; the potential failure of XL647, XL184, XL880, XL019, XL147, XL765, XL281, XL518, XL228, XL820, XL844, XL139 or any of Exelixis' other compounds to demonstrate safety and efficacy in clinical testing; Exelixis' dependence on and relationships with GlaxoSmithKline, Bristol-Myers Squibb and Genentech; Exelixis' ability to initiate and complete clinical trials at the referenced times, or at all; and Exelixis' ability to successfully advance and develop additional compounds. These and other risk factors are discussed under "Risk Factors" and elsewhere in Exelixis' Annual Report on Form 10-K for the fiscal year ended December 28, 2007 and Exelixis' other reports filed with the Securities and Exchange Commission. Exelixis expressly disclaims any duty, obligation or undertaking to release publicly any updates or revisions to any forward-looking statements made in this discussion to reflect any change in Exelixis' expectations with regard thereto or any changes in events, conditions or circumstances on which any such statements are based.



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