CYTRX CORP
Form 10-K
March 15, 2010

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

Form 10-K

(Mark One)

R ANNUAL REPORT PURSUANT TO SECTION 13 OR 15(d) OF THE SECURITIES EXCHANGE ACT OF 1934

For the fiscal year ended December 31, 2009
or

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

For the transition period from _____ to _____

Commission file number 0-15327

CytRx Corporation
(Exact name of Registrant as specified in its charter)

Delaware	58-1642740
(State or other jurisdiction of incorporation or organization)	(I.R.S. Employer Identification No.)
11726 San Vicente Blvd, Suite 650, Los Angeles, California	90049
(Address of principal executive offices)	(Zip Code)

Registrant's telephone number, including area code: (310) 826-5648

_____

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

Title of each class	Name of exchange on which registered

Common Stock, $0.001 par value per share	The NASDAQ Capital Market
Series A Junior Participating Preferred Stock Purchase Rights	

Securities Registered Pursuant to Section 12(g) of the Act:
None

Indicate by check mark if the Registrant is a well-known seasoned issuer (as defined in Securities Act Rule 405). Yes £ No R

Indicate by check mark if the Registrant is not required to file reports pursuant to Section 13 or Section 15(d) of the Securities Exchange Act of 1934.
Yes £ No R

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 and (2) has been subject to such filing requirements for the past 90 days. Yes R No £

Indicate by check mark whether the registrant has submitted electronically and posted on its corporate website, 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 £

Indicate by check mark if disclosure of delinquent filers pursuant to Item 405 of Regulation S-K is not contained herein, and will not be contained, to the best of the Registrant's knowledge, in definitive proxy or information statements incorporated by reference in Part III of this Form 10-K or any amendment to this Form 10-K. R

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

Large accelerated filer £	Accelerated filer R	Non-accelerated filer £ (Do not check if a smaller reporting company)	Smaller reporting company £

Indicate by check mark whether the Registrant is a shell company (as defined in Rule 12b-2 of the Act). Yes £ No R

Based on the closing price as reported on The Nasdaq Capital Market, the aggregate market value of the Registrant's common stock held by non-affiliates on June 30, 2009 (the last business day of the Registrant's most recently completed second fiscal quarter) was approximately $102.0 million. Shares of common stock held by directors and executive officers and their respective affiliates have been excluded from this calculation, because such stockholders may be deemed to be "affiliates" of the Registrant. This is not necessarily determinative of affiliate status for other purposes. The number of outstanding shares of the Registrant's common stock as of March 12, 2010 was 108,908,105, exclusive of treasury shares.

CYTRX CORPORATION
2009 ANNUAL REPORT ON FORM 10-K

TABLE OF CONTENTS

“SAFE HARBOR” STATEMENT

Some of the information contained in this Annual Report may include forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, or the Securities Act, and Section 21E of the Securities Exchange Act of 1934, as amended, or the Exchange Act. We base these forward-looking statements on our current views with respect to our research and development activities, business strategy, business plan, financial performance and other matters, both with respect to us, specifically, and the biotechnology sector, in general. Statements that include the words “expect,” “intend,” “plan,” “believe,” “project,” “estimate,” “may,” “should,” “anticipate,” “will” and simi of a future or forward-looking nature identify forward-looking statements for purposes of the federal securities laws or otherwise, but the absence of these words does not necessarily mean that a statement is not forward-looking.

All forward-looking statements involve inherent risks and uncertainties, and there are or will be important factors that could cause actual results to differ materially from those indicated in these statements. We believe that these factors include, but are not limited to, those factors set forth in the sections entitled “Business,” “Risk Factors,” “Legal Proceedings,” “Management’s Discussion and Analysis of Financial Condition and Results of Operations,” “Quantitative and Qualitative Disclosures About Market Risk” and “Controls and Procedures” in this Annual Report, all of which you should review carefully. Please consider our forward-looking statements in light of those risks as you read this Annual Report. We undertake no obligation to publicly update or review any forward-looking statement, whether as a result of new information, future developments or otherwise, except as required by law.

If one or more of these or other risks or uncertainties materializes, or if our underlying assumptions prove to be incorrect, actual results may vary materially from what we anticipate. All subsequent written and oral forward-looking statements attributable to us or individuals acting on our behalf are expressly qualified in their entirety by the cautionary language above. You should consider carefully all of the factors set forth or referred to in this Annual Report that could cause actual results to differ.

PART I

Item 1. BUSINESS

In this Annual Report, we sometimes refer to CytRx Corporation as "CytRx," to our former subsidiary, RXi Pharmaceuticals Corporation, as "RXi," and to Innovive Pharmaceuticals, Inc., which we acquired in September 2008, as "Innovive." References in this Annual Report to the "company," "we," "us" or "our" refer to CytRx, alone, unless otherw indicated.

COMPANY OVERVIEW

We are a biopharmaceutical research and development company engaged in the development of high-value human therapeutics, specializing in oncology. Our drug development pipeline includes clinical development of three product candidates for cancer indications, including three planned Phase 2 clinical trials for INNO-206 as a treatment for pancreatic cancer, gastric (stomach) cancer and soft tissue sarcomas, two Phase 2 proof-of-concept clinical trials with bafetinib in patients with high-risk B-cell chronic lymphocytic leukemia, or B-CLL, and patients with glioblastoma multiforme, and a registration study of tamibarotene for the treatment of acute promyelocytic leukemia, or APL. In addition to our core oncology programs, we are developing two drug candidates based on our molecular chaperone regulation technology. Our current business strategy for our molecular chaperone regulation technology is to seek one or more strategic partnerships, or a possible spin-out transaction. Apart from our drug development programs, we currently maintain a 36% equity interest in our former subsidiary, RXi.

We are a Delaware corporation, incorporated in 1985. Our corporate offices are located at 11726 San Vicente Boulevard, Suite 650, Los Angeles, California 90049, and our telephone number is (310) 826-5648.

OUR PRODUCT CANDIDATE PIPELINE

The following tables summarize our product candidates and their current or planned stage of development:

Technology	Product Candidate	Indication(s)	Stage of Development
Doxorubicin prodrug	INNO-2006	Pancreatic cancer	Phase II (1H10)
		Gastric (stomach) cancer	Phase II (2H10)
		Soft tissue sarcomas	Phase II (2H10)
Tyrosine kinase inhibitor	Bafetinib	B-CLL	Phase II (1H10)
		Glioblastoma Multiforme	Phase II (2H10)
Synthetic retinoid	Tamibarotene	APL (acute promyelocytic leukemia)	Pivotal Phase II
Molecular chaperone regulation	Arimoclomol	ALS (amyotrophic lateral sclerosis, or Lou Gehrig's disease)	Phase II
Molecular chaperone regulation	Iroxanadine	Several potential indications	Phase I

OUR CLINICAL DEVELOPMENT PROGRAMS

Our current clinical development programs consist of our efforts to develop INNO-206 for gastric (stomach) cancer, pancreatic cancer and soft tissue sarcomas, bafetinib for B-CLL and glioblastoma multiforme, tamibarotene for APL,

and our molecular chaperone regulation program, which includes a planned Phase II clinical study of arimoclomol in ALS.

INNO-206. INNO-206 (formerly DOXO-EMCH) is a prodrug of the commonly prescribed chemotherapeutic agent doxorubicin. Specifically, it is the (6-Maleimidocaproyl) hydrazone of doxorubicin. Essentially, this chemical is doxorubicin (DOXO) attached to an acid sensitive linker (EMCH).

INNO-206 for the Treatment of Cancer. Anthracyclines are a class of drugs that are among the most commonly used agents in the treatment of cancer. Doxorubicin, the first anthracycline to gain FDA approval, has demonstrated efficacy in a wide variety of cancers including breast cancer, lung cancer, sarcomas, and lymphomas. However, due to the uptake of doxorubicin by various parts of the body, it is associated with side effects such as cumulative cardiotoxicity, myelosuppression (decreased production of blood cells by bone marrow), gastrointestinal disorders, mucositis (inflammation of the mucous membranes lining the digestive tract, including the mouth), stomatitis (inflammation of the mouth's soft tissue), and extravasation (the leakage of intravenous drugs from the vein into the surrounding tissue).

We believe INNO-206 has attributes that improve on native doxorubicin, including reduction of adverse events, improvement in efficacy and the ability to reach the tumor more quickly.

Our anticipated mechanism of action for INNO-206 is as follows:

- after administration, INNO-206 rapidly binds circulating albumin through the EMCH linker;
- circulating albumin preferentially accumulates in tumors, bypassing uptake by other non-tumor sites, including the heart, bone marrow and the gastrointestinal tract;
- once albumin-bound INNO-206 reaches the tumor, the acidic environment of the tumor causes cleavage of the acid sensitive linker; and
- free doxorubicin is released at the site of the tumor and is taken up by the cancer cells.

Pre-clinical data. In a variety of preclinical models, INNO-206 was superior to doxorubicin in its ability to increase the total doxorubicin dose, antitumor efficacy, and safety, including a reduction in cardiotoxicity. Animal studies conducted by INNO-206 inventor Dr. Felix Kratz, Department of Medical Oncology, Clinical Research, at the Tumor Biology Center in Freiburg, Germany, demonstrated statistically significant efficacy against breast, ovarian, pancreatic and small cell lung cancers growing in immunodeficient mice.

Clinical data. A Phase I study of INNO-206 that demonstrated safety and objective clinical responses in a variety of tumor types was completed in 2005 and presented at the March 2006 Krebskongress meeting in Berlin. In this study, single doses were administered every 3 weeks at up to six times the standard dosing of doxorubicin without an increase in side effects over those historically observed with doxorubicin. Twenty-three of 35 evaluable patients had either an objective clinical (partial) response or stable disease. Objective clinical responses were observed in patients with sarcoma, breast, and lung cancers.

Development Plan. Based on the objective clinical responses seen in the Phase I study, and preclinical data, we intend to initiate three Phase 2 clinical trials in 2010 of INNO-206 in patients with pancreatic cancer, gastric cancer and soft tissue sarcomas.

Bafetinib. Bafetinib (formerly INNO-406) is a novel drug developed by the Japanese pharmaceutical company Nippon Shinyaku, to overcome the limitations of Gleevec and second-line tyrosine kinase inhibitors in resistant chronic myelogenous leukemia, or CML. We have announced plans to develop bafetinib as a treatment for B-cell chronic lymphocytic leukemia (B-CLL) and glioblastoma multiforme (GBM), due to the potent and specific inhibitory properties of bafetinib against Lyn kinase, which is overexpressed in both B-CLL and GBM.

Bafetinib for the Treatment of B-CLL. B-CLL is the most common form of leukemia in adults in Western countries. More than 17,000 new cases of B-CLL are reported in the United States each year; however up to an estimated 40% of cases may not be reported due to under-diagnosis and lack of placement in cancer registries. Virtually all patients are older than 55 years at presentation, with an average age of 70 years. Patients in the high-risk B-CLL classification have a median overall survival of one to five years.

Pre-clinical Data. In mice-leukemia models, bafetinib has been shown to markedly extend the survival of animals implanted with Gleevec-resistant leukemic cells. In toxicology studies done in mice, rats, and dogs, bafetinib appeared to be safe and well-tolerated. A dose described in dogs at which no side effects were seen was used to calculate the starting dose in humans for our recently completed clinical trial.

Phase I Study. In November 2008, we announced that bafetinib demonstrated clinical responses in patients with CML in a Phase I clinical trial conducted in patients with CML and other leukemias that have a certain mutation called the Philadelphia Chromosome (Ph+) and are intolerant of or resistant to Gleevec and, in some cases, second-line tyrosine kinase inhibitors such as dasatinib (Sprycel®) and nilotinib (Tasigna®)). The clinical trial was designed to identify the optimal dose for possible future studies by escalating doses from 30 mg once per day to up to 480 mg twice per day in a total of 56 patients with Ph+ leukemias. Of the patients, 31 had CML in chronic phase (CML-CP), nine were in accelerated phase (CML-AP), seven were in blast phase (CML-BP), and nine had Ph+ acute lymphocytic leukemia. The clinical trial was conducted at seven clinical sites in the US, Germany, and Israel, with Hagop Kantarjian, M.D., Professor & Chairman, Department of Leukemia, The University of Texas, M.D. Anderson Cancer Center, serving as the Principal Investigator. A positive, dramatic decrease in the number of leukemia cells in the bone marrow was seen in 35% of the patients that were randomly chosen to begin their treatment with the optimal bafetinib dose of 240 mg twice per day.

The maximum tolerated dose was determined to be 240 mg given twice per day, based on evidence of increasing potential liver toxicity at higher doses. Common adverse events (observed in greater than 20% of patients in the 240 mg twice per day dose group) were gastrointestinal toxicity, swelling, and fatigue. There was no evidence of fluid accumulating around the lungs, or significant changes in a certain heart rhythm called QTc prolongation, which are serious side effects known to occur in patients treated with approved drugs for this indication. Approximately 13% of patients across all dose groups discontinued dosing due to unacceptable toxicity.

Development Plan. We recently announced plans to initiate in the first half of 2010 a Phase 2 proof-of-concept clinical trial to evaluate the efficacy and safety of bafetinib in patients with B-CLL. In the planned Phase 2 trial, approximately 20 patients who have failed treatment with first-line agents will be administered daily oral doses of bafetinib. Patients will be monitored for clinical response, time to disease progression and cancer progression-free survival. Based on trial results, we plan to conduct a larger comparative trial to further determine efficacy of this agent. We also plan to initiate a clinical Phase 2 proof of concept clinical trial with bafetinib as a treatment for glioblastoma multiforme, a common and aggressive type of primary brain tumor, in the second half of 2010.

Tamibarotene. Tamibarotene is an orally available, synthetic retinoid rationally designed to overcome resistance and avoid toxic side effects of differentiation therapy with all-trans retinoic acid, or ATRA, a component of the current first-line treatment for APL.

Tamibarotene for the treatment of APL. Acute promyelocytic leukemia, or APL, is a specific type of acute myeloid leukemia characterized by the t(15;17) translocation, which fuses the promyelocytic leukemia, or PML, gene on chromosome 15 to the retinoic acid receptor, or RARα gene on chromosome 17. This fusion causes abnormal cell growth.

Differentiation therapy with ATRA, is the basis for the treatment of APL. Differentiation therapy causes leukemic promyelocytes to mature and undergo cell death. Patients typically receive ATRA in combination with chemotherapy as the initial therapy, followed by anthracyline-based consolidation therapy designed to produce complete remission. The majority of patients treated this way generally experience a complete remission of disease. Current National Comprehensive Cancer Network guidelines recommend that patients then undergo one to two years of maintenance therapy with ATRA to prevent a recurrence. ATRA therapy is associated with several toxicities, the most serious of which, retinoic acid syndrome, or RAS. RAS, which occurs in up to 25% of patients treated with ATRA, is a serious and potentially fatal complication characterized by fever, dyspnea (breathing difficulties), weight gain, pulmonary infiltrates (abnormal accumulation in the lungs), and pleural or pericardial effusions (excess fluid around the lungs or heart).

Patients that initially respond to front-line therapy with ATRA plus chemotherapy sometimes relapse, and some of these patients fail to respond to a second course of treatment with ATRA. Currently, patients who fail ATRA-based therapy are treated with arsenic trioxide, a compound administered intravenously and associated with significant toxicity, including irregular heartbeat. There currently is no standard of care for patients who do not respond to ATRA and arsenic trioxide, or who respond but subsequently relapse. In 2007, the FDA granted Orphan Drug Designation and Fast Track Designation for the use of tamibarotene in patients with relapsed or refractory APL following treatment with ATRA and arsenic trioxide.

Tamibarotene was developed to overcome resistance to ATRA. In vitro, tamibarotene is approximately ten times more potent than ATRA at causing APL cells to differentiate and die. In addition, tamibarotene has a lower affinity for cellular retinoic acid binding protein, or CRABP, which we believe should allow for sustained plasma levels during administration. This may enhance tamibarotene's potential efficacy, because patients may be able to experience benefits from the drug over a longer period of time. Tamibarotene does not bind the RAR-γ receptor, the major retinoic acid receptor in the dermal epithelium, which should lessen the occurrence of skin toxicities.

Pre-clinical data. In preclinical models, tamibarotene was superior to ATRA in its ability to cause APL cells to differentiate and die. In the clinical setting, in vitro response to tamibarotene appeared predictive of clinical response, including activity in patients who had a poor response to ATRA.

Clinical data. Tamibarotene is approved in Japan under the brand name Amnolake for use in relapsed or refractory APL. The approval was based on data from two studies in Japanese patients. In the pivotal study, the effectiveness of orally administered tamibarotene was administered to 42 patients with APL, 39 of whom were evaluable for response. Patients included individuals who had never received treatment for APL and patients who had been previously treated with ATRA. Tamibarotene was administered orally at a dose of 6 mg/m2/day for eight weeks. The overall complete response rate in these patients was 61.5%. In patients who had a recurrence of APL following ATRA therapy, the response rate was 81%. RAS was reported in three patients, or 7.3% of the patient group.

Development Plan. There is currently a Special Protocol Assessment (SPA) in place with the FDA for a Phase 2 registration clinical trial, known as STAR-1, which is evaluating the efficacy and safety of tamibarotene as a third-line treatment for APL. The STAR-1 trial is ongoing and currently includes six clinical sites in the U.S. We recently reported that, of the 11 patients enrolled in the STAR-1 trial to date, three (27%) achieved a hematologic complete response, and four (36%) a morphologic leukemia-free state. Depending on the trial's outcome, this study, in combination with the data from the two Japanese studies, could form the basis of a new drug application, or NDA.

In addition, we have announced plans to develop tamibarotene, in combination with chemotherapy or arsenic trioxide (ATO), as a first-line treatment for APL. We have received favorable reviews on tamibarotene as a potential treatment for APL from key opinion leaders and have been working with notable clinicians in this field on trial design.

Among other preparatory activities, in September 2009, we initiated a dose escalation clinical trial with tamibarotene combined with TRISENOX® (arsenic trioxide) injection (marketed by Cephalon, Inc.) in patients with relapsed APL. An objective of this trial is to determine the appropriate combination therapy dose for evaluation as a potential first-line treatment for APL.

Arimoclomol. Arimoclomol is an orally-administered small-molecule product candidate that we believe functions by stimulating a normal cellular protein repair pathway by amplifying molecular chaperone proteins implicated in neurological disorders.

Arimoclomol for the treatment of ALS. ALS, or Lou Gehrig's disease, is a debilitating and ultimately deadly disease involving the progressive degeneration of motor neurons believed to be caused by toxic mis-folding of proteins. According to the ALS Association, approximately 30,000 people in the U.S. are living with ALS and 5,600 new cases are diagnosed each year. Worldwide, an estimated 120,000 people are living with ALS. According to the ALS Survival Guide, 50% of ALS patients die within 18 months of diagnosis and 80% die within five years of diagnosis.

The following is a summary of our clinical development of arimoclomol for treating ALS:

- in July 2006, we completed an 84-patient, multi-center, double-blind, placebo-controlled, multi-dose Phase IIa clinical trial of safety and tolerability of arimoclomol in volunteers with ALS, which we refer to as the Phase IIa trial;

- in May 2007, we completed an open-label extension of the Phase IIa trial in approximately 70 ALS patients from the trial who were administered the highest investigational dose (100 mg three times daily) of arimoclomol for an additional six months;

- in June 2007, we completed a multiple ascending-dose clinical trial of safety and tolerability involving 40 healthy volunteers; and

- in November 2007, we completed a 28-day safety clinical trial with 400 mg of arimoclomol three times daily involving 16 healthy volunteers.

Phase IIa clinical trial. Participants in the Phase IIa clinical trial of arimoclomol were administered either a placebo capsule, or one of three dosage levels of arimoclomol capsules, three times daily for a period of 12 weeks, immediately followed by a one-month period without the drug. The primary endpoints of the Phase IIa trial were safety and tolerability. Secondary endpoints included a preliminary evaluation of efficacy using two widely accepted disease-progression markers. The first marker, the revised ALS Functional Rating Scale, or ALSFRS-R, is used to determine patients' overall functional capacity and independence in 13 activities. The second marker measures vital capacity, an assessment of lung capacity, which is an important disease indicator since ALS sufferers eventually lose the ability to breathe on their own. The trial was designed to be able to detect only extreme responses in these two markers.

The results from our Phase IIa trial and open-label extension clinical trial indicated that arimoclomol was safe and well tolerated in ALS volunteers, even at the highest administered dose. Arimoclomol was detected in participants' cerebral spinal fluid, demonstrating that it passed the so-called blood:brain barrier, and participants treated with arimoclomol experienced a statistically significant decrease in adverse events of weakness compared with the placebo group.

Phase IIb efficacy trial. In December 2009, the FDA accepted a revised protocol for an ascending dose efficacy trial to evaluate the safety and efficacy in ALS patients at up to 400 mg administered orally three times daily. The clinical trial protocol accepted by the FDA is a tiered, placebo-controlled, double-blind ascending dose study. The study is designed to test progressive groups, each with between 20 and 30 ALS patients over a three-month treatment period. Fifteen patients would receive a combination of arimoclomol at various dose levels and riluzole at a fixed dose of 50 mg twice daily, with between five and 15 ALS patients receiving placebo and riluzole at the same fixed dose. The first group would receive arimoclomol 100 mg capsules three times daily. Every four weeks, another group of ALS patients would begin three-month testing with six patients receiving arimoclomol dosing three times daily at a 75 mg per dose increase from the prior group. The maximum dose in the protocol allows for testing arimoclomol at 400 mg three times daily. An independent safety monitoring board would review safety results prior to initiating each consecutive increase in dosage level.

Other Clinical Development. In February 2009, a Phase II/III adaptive clinical trial commenced to study arimoclomol in a subset of patients with the inherited or familial form ALS. Patients with familial ALS (fALS) who harbor certain mutations in the superoxide dismutase-1 (SOD1) gene suffer from a rapidly progressing form of the disease. The clinical trial is being financially supported by grants from the ALS Association and the U.S. Food and Drug Administration's (FDA's) Office of Orphan Products Development (OOPD), and we are supplying the drug and allowing the sponsor to reference our Investigational New Drug Application for regulatory purposes.

Arimoclomol for recovery from stroke. Stroke results from an acute loss of normal blood flow to the brain caused most often by a blockage in a blood vessel (ischemic) or due to leaking of blood from a vessel (hemorrhagic). According to the American Heart Association: stroke is the third leading cause of death and the number one cause of long-term disability in the U.S.; between 50% and 70% of stroke survivors regain functional independence, but between 15% and 30% are permanently disabled and 20% require institutional care within three months after stroke; and the direct and indirect stroke cost in the U.S. totaled approximately $58 billion in 2006.

After the normal flow of blood is restored to the brain, post-stroke neurological function continues to decline. We believe that this continuing decline in neurological function is the consequence of mis-folded protein aggregates generated as a result of oxygen deprivation during the original event. Preclinical efficacy studies completed by us in April 2007 indicated that arimoclomol accelerated the time to recovery, and improved recovery, in experimental animal models of stroke. These results were obtained even when arimoclomol was administered as long as 48 hours after onset.

Arimoclomol for the treatment of cancer. Through research conducted in our former San Diego laboratory, we have identified preclinical pipeline compounds that inhibit, rather than amplify, molecular chaperone proteins. These early stage compounds are potential oncology therapeutics because cancer cells by their very nature generate large amounts of toxic, misfolded proteins and are far more dependent upon the activity of molecular chaperone proteins for survival than are normal cells. Because several of these compounds have already been shown to selectively kill cancer cells and spare normal cells in tissue culture, they represent a novel approach to cancer therapeutics with the potential of having relatively large therapeutic indices.

Iroxanadine. Iroxanadine also is an orally-administered small-molecule product candidate. We believe it functions by stimulating the molecular chaperone protein response in the endothelium, the thin layer of cells that line the interior

surface of human blood vessels.

Iroxanadine for the treatment of diabetic ulcers. Type 2 diabetes is a major health problem with significant secondary complications. The American Diabetes Association estimates that there are 21 million type 2 diabetes sufferers in the U.S. The World Health Organization estimates that there are more than 162 million cases of type 2 diabetes worldwide. According to the American Diabetes Association, 15% of all diabetics will develop a foot ulcer during their lifetime, and over 82,000 non-traumatic lower-limb amputations were performed on diabetics in the U.S. in 2002 due to ulcers and other complications. We believe there are reasonable data in the scientific literature supporting the concept that diabetic foot ulcers fail to heal efficiently due to the dysfunction of endothelial cells lining the blood vessels caused by protein mis-folding.

Animal studies completed by us in May 2007 indicated that iroxanadine significantly decreased the time it took for wounds to heal in diabetic mice without affecting healing in healthy mice. Wound healing in the diabetic mice, which normally required twice the time to heal as healthy mice, was accelerated to the extent that healing time of diabetic mice treated with iroxanadine was indistinguishable from that in untreated healthy mice.

In Phase I clinical trials in healthy volunteers and Phase II clinical trials in patients with chronic high blood pressure conducted prior to our acquisition of iroxanadine, the drug candidate was shown to be safe and well-tolerated and demonstrated significant improvement in the function of endothelial cells in the brachial artery, a major blood vessel of the upper arm.

Strategic Plans for Molecular Chaperone Assets. We intend to seek a partner for the development of arimoclomol and iroxanadine for all indications on a worldwide basis. We are also exploring the possibility of a spin-out transaction to accelerate the development of our molecular chaperone assets.

Other Technologies

Our other current technologies are CRL-5861, an intravenous agent for treatment of sickle cell disease and other acute vaso-occlusive disorders, and TranzFect, a delivery technology for DNA-based and conventional vaccines and other potential uses. We currently have no plans for development of these technologies.

Our Separation from RXi Pharmaceuticals Corporation

Until early 2008, we owned approximately 85% of the outstanding shares of common stock of RXi and our financial statements, including our financial statements as of and for the year ended December 31, 2007, included the consolidated financial condition and results of operations of RXi. On February 14, 2008, our board of directors declared a dividend of one share of RXi common stock for each approximately 20.05 outstanding shares of our common stock, which was paid on March 11, 2008 and which reduced our ownership of RXi shares to less than 50%. As a result, our financial statements since March 11, 2008 no longer consolidate the financial condition and results of operation of RXi, but instead reflect our ongoing investment in RXi based on the equity method of accounting. As of March 12, 2010, we owned approximately 36% of the outstanding shares of RXi common stock.

We are party to a letter agreement with RXi and some of RXi's current stockholders under which we are entitled to preemptive rights to acquire any "new securities" (as defined) that RXi proposes to sell or issue, so that we may maintain our percentage ownership in RXi. Our preemptive rights will expire on January 8, 2012 or such earlier time at which we own less than 10% of RXi's outstanding common stock.

Under the letter agreement with RXi, we agreed to vote our RXi shares for the election of RXi directors and take other actions to ensure that a majority of the board of directors of RXi are independent of us. We further agreed to approve of actions that may be adopted and recommended by the RXi board of directors to facilitate any future financing by RXi.

Manufacturing

We have no capability to manufacture supplies of any of our products, and rely on third-party manufacturers to produce materials needed for research and clinical trials. We have contracted with various contract manufacturing facilities for supply of our active pharmaceutical ingredient, or API, for our product candidates. Pursuant to our license with TMRC Co., Ltd., or TMRC, relating to tamibarotene, TMRC will provide us with tamibarotene at a fixed price and in a quantity and quality sufficient to meet our clinical and commercial needs.

To be commercialized, our products also must be capable of being manufactured in commercial quantities in compliance with stringent regulatory requirements and at an acceptable cost. We intend to rely on third-party manufacturers to produce commercial quantities of any products for which we are able to obtain marketing approval. We have not commercialized any product, and so we also have not demonstrated that any of our product candidates can be manufactured in commercial quantities in accordance with regulatory requirements or at an acceptable cost.

If our product candidates cannot be manufactured in suitable quantities and in accordance with regulatory standards, our clinical trials, regulatory approvals, and marketing efforts for such products may be delayed. Such delays could adversely affect our competitive position and our chances of generating significant recurring revenues. If our products are not able to be manufactured at an acceptable cost, the commercial success of our products may be adversely affected.

Marketing

Our tentative plan is to establish our own sales force and marketing capability in order to commercialize our oncology drug candidates, including INNO-206, bafetinib and tamibarotene, in the U.S. and to seek a marketing partner for commercialization in other territories.

Patents and Proprietary Technology

We actively seek patent protection for our technologies, processes, uses, and ongoing improvements and consider our patents and other intellectual property to be critical to our business. We acquired patents and patent applications, and have filed several new patent applications, in connection with our molecular chaperone program.

We regularly evaluate the patentability of new inventions and improvements developed by us or our collaborators, and, whenever appropriate, will endeavor to file U.S. and international patent applications to protect these new inventions and improvements. We cannot be certain that any of the current pending patent applications we have filed or licensed, or any new patent applications we may file or license, will ever be issued in the U.S. or any other country. There also is no assurance that any issued patents will be effective to prevent others from using our products or processes. It is also possible that any patents issued to us, as well as those we have licensed or may license in the future, may be held invalid or unenforceable by a court, or third parties could obtain patents that we would need to either license or to design around, which we may be unable to do. Current and future competitors may have licensed or filed patent applications or received patents, and may acquire additional patents and proprietary rights relating to molecular chaperone amplification and other small molecule technology or other compounds, products or processes that may be competitive with ours.