ARRAY BIOPHARMA INC
Form 10-K
August 12, 2011

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U.S. SECURITIES AND EXCHANGE COMMISSION
Washington, D.C. 20549

Form 10-K

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

For the fiscal year ended June 30, 2011

OR

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

For the transition period from to

Commission File Number: 001-16633

Array BioPharma Inc.

(Exact Name of Registrant as Specified in Its Charter)

Delaware	**84-1460811**
(State of Incorporation)	(I.R.S. Employer Identification No.)

3200 Walnut Street
Boulder, Colorado 80301
(Address of Principal Executive Offices)

(303) 381-6600
(Registrant's Telephone Number, Including Area Code)

Common Stock, Par Value $.001 per Share
(Securities Registered Pursuant to Section 12(b) of the Act)

The NASDAQ Stock Market LLC (NASDAQ Global Market)
(Name of Exchange on Which Registered)

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

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

Yes o No þ

Indicate by check mark if the registrant is not required to file reports pursuant to Section 13 or 15(d) of the Exchange Act.

Yes o No þ

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 þ No o

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

Yes o No o

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 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. o

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 o	Accelerated filer þ	Non-accelerated filer o (do not check if a smaller reporting company)	Smaller reporting company o

Indicate by check mark whether the registrant is a shell company (as defined in Rule 12b-2 of the Exchange Act).

Yes o No þ

The aggregate market value of voting stock held by non-affiliates of the registrant as of December 31, 2010 (based upon the closing sale price of such shares as of the last trading day of the second fiscal quarter ended December 31, 2010, on the NASDAQ Global Market) was $76,171,933. Shares of the Registrant's common stock held by each executive officer and director and by each entity that owns 5% or more of the Registrant's outstanding common stock have been excluded in that such persons or entities may be deemed to be affiliates. This determination of affiliate status is not necessarily a conclusive determination for other purposes.

Number of shares outstanding of the registrant's class of common stock as of August 5, 2011: 57,020,003.

DOCUMENTS INCORPORATED BY REFERENCE

Portions of the registrant's definitive Proxy Statement to be filed with the Securities and Exchange Commission on Form 14A for the 2011 Annual Meeting of Stockholders are incorporated by reference in Part III of this Annual Report on Form 10-K to the extent stated therein.

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PART I

Array BioPharma Inc., the Array BioPharma Inc. logo and the marks "ARRAY BIOPHARMA THE DISCOVERY RESEARCH COMPANY," "TURNING GENOMICS INTO BREAKTHROUGH DRUGS," "OPTIMER," and "ARRAY DISCOVERY PLATFORM" are trademarks of Array BioPharma Inc. All other brand names or trademarks appearing in this report are the property of their respective holders. Unless the context requires otherwise, references in this report to "Array," "we," "us," and "our" refer to Array BioPharma Inc.

FORWARD-LOOKING STATEMENTS

This Annual Report filed on Form 10-K and other documents we file with the Securities and Exchange Commission contain forward-looking statements within the meaning of the Private Securities Litigation Reform Act of 1995 that involve significant risks and uncertainties. In addition, we may make forward-looking statements in our press releases or in other oral or written communications with the public. These forward-looking statements include, but are not limited to, statements concerning the future drug development plans and projected timelines for the initiation and completion of preclinical and clinical trials by Array or our collaborators; the potential for the results of ongoing preclinical or clinical trials conducted by Array or our collaborators to support regulatory approval or the marketing success of drug candidates; our plans with respect to the timing and scope of the expansion of our clinical and commercialization capabilities; other statements regarding our future product development and regulatory strategies, including with respect to specific indications; the ability of third-party contract manufacturing parties to support our drug development activities; any statements regarding our future financial performance, results of operations or sufficiency of capital resources to fund our operating requirements; and any other statements which are other than statements of historical fact.

Although we believe the assumptions upon which our forward-looking statements are based currently to be reasonable, our actual results could differ materially from those anticipated in these forward-looking statements as a result of many factors. These factors include, but are not limited to, our ability to continue to fund and successfully progress internal research and development efforts and to create effective, commercially viable drugs; our ability to effectively and timely conduct clinical trials in light of increasing costs and difficulties in locating appropriate trial sites and in enrolling patients who meet the criteria for certain clinical trials; the extent to which the pharmaceutical and biotechnology industries are willing to in-license drug candidates for their product pipelines and to collaborate with and fund third parties on their drug discovery activities; our ability to out-license our proprietary candidates on favorable terms; risks associated with our dependence on our collaborators for the clinical development and commercialization of our out-licensed drug candidates; the ability of our collaborators and of Array to meet objectives tied to milestones and royalties; our ability to attract and retain experienced scientists and management; our ability to achieve and maintain profitability; and the risk factors set forth below under the caption Item 1A. Risk Factors. We are providing this information as of the date of this report. We undertake no duty to update any forward-looking statements to reflect the occurrence of events or circumstances after the date of such statements or of anticipated or unanticipated events that alter any assumptions underlying such statements.

ITEM 1 BUSINESS

Our Business

We are a biopharmaceutical company focused on the discovery, development and commercialization of targeted small molecule drugs to treat patients afflicted with cancer and inflammatory diseases. Our proprietary drug development pipeline includes clinical candidates that are designed to regulate

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therapeutically important target pathways. In addition, leading pharmaceutical and biotechnology companies partner with us to discover and develop drugs across a broad range of therapeutic areas.

The five most advanced wholly-owned programs that we are developing internally are:

Program	Indication	Clinical Status
1. ARRY-520	Kinesin spindle protein, or KSP, inhibitor for multiple myeloma	Phase 2
2. ARRY-614	p38/Tie-2 dual inhibitor for myelodysplastic syndrome, or MDS	Phase 1
3. ARRY-380	HER2 inhibitor for breast cancer	Phase 1
4. ARRY-797	p38 inhibitor for pain	Phase 2
5. ARRY-502	CRTh2 antagonist for allergic inflammation	Phase 1

In addition to these development programs, our most advanced partnered drugs in clinical development are:

Drug Candidates	Indication	Partner	Clinical Status
1. Selumetinib and AZD8330	MEK inhibitors for cancer	AstraZeneca, PLC	Phase 2
2. MEK162 and MEK300	MEK inhibitors for cancer	Novartis International Pharmaceutical Ltd.	Phase 2
3. Danoprevir	Hepatitis C virus (HCV) protease inhibitor	InterMune (now being developed by Roche Holding AG)	Phase 2
4. ARRY-543	HER2/EGFR inhibitor for solid tumors	ASLAN Pharmaceuticals Pte Ltd.	Phase 2
5. LY2603618	ChK-1 inhibitor for cancer	Eli Lilly and Company	Phase 2
6. AMG 151	Glucokinase activator for Type 2 diabetes	Amgen Inc.	Phase 1b
7. GDC-0068	AKT inhibitor for cancer	Genentech Inc.	Phase 1b
8. VTX-2337	Toll-like receptor for cancer	VentiRx Pharmaceuticals, Inc.	Phase 1b
9. VTX-1463	Toll-like receptor for allergy	VentiRx	Phase 1b

		Pharmaceuticals, Inc.	
10. ARRY-382	cFMS inhibitor for cancer	Celgene Corporation	Phase 1
11. ARRY-575 and GDC-0425	ChK-1 inhibitor for cancer	Genentech Inc.	Phase 1

Any information we report about the development plans or the progress or results of clinical trials or other development activities of our partners is based on information that is publicly disclosed.

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Our significant and / or recent collaborators under our partnered programs include:

Amgen We entered into a worldwide strategic collaboration with Amgen in December 2009 to develop and commercialize our glucokinase activator, AMG 151, and to discover potential back-up compounds for AMG 151.

ASLAN Pharmaceuticals We entered into a collaboration and license agreement with ASLAN Pharmaceuticals in July 2011 to develop Array's HER2 / EGFR inhibitor, ARRY-543, which is currently entering Phase 2 development for solid tumors.

AstraZeneca In December 2003, we entered into a collaboration and license agreement with AstraZeneca under which AstraZeneca received a license to three of our MEK inhibitors for cancer, including selumetinib, which is currently in multiple Phase 2 clinical trials.

Celgene We entered into a worldwide strategic collaboration agreement with Celgene in September 2007 focused on the discovery, development and commercialization of novel therapeutics in cancer and inflammation. The most advanced drug is ARRY-382, a cFMS inhibitor for cancer, which is currently in a Phase 1 clinical trial.

Genentech We entered into a worldwide strategic collaboration agreement with Genentech in January 2003, which was expanded in 2005, 2008, and 2009, and is focused on the discovery, development and commercialization of novel therapeutics. The most advanced drug is GDC-0068, an AKT inhibitor for cancer currently in a Phase 1b trial. The other programs under this collaboration are in preclinical development. In August 2011, we entered into an oncology partnership with Genentech for the development of each company's small-molecule Checkpoint kinase 1 (ChK-1) program. The programs include Genentech's compound GDC-0425 (RG7602), currently in Phase 1, and Array's compound, ARRY-575, which is being prepared for an investigational new drug application to initiate a Phase 1 trial in cancer patients.

Novartis We entered into a worldwide strategic collaboration with Novartis in April 2010 to develop and commercialize our MEK inhibitor, MEK162, and other MEK inhibitors identified in the agreement.

InterMune (program acquired by Roche) We entered into a collaboration with InterMune in 2002, which resulted in the joint discovery of danoprevir, a novel small molecule inhibitor of the Hepatitis C Virus NS3/4A protease. Roche Holding AG acquired danoprevir from InterMune in 2010. Danoprevir is currently in Phase 2b clinical trials.

Under our partnered drug discovery programs, we are generally entitled to receive payments upon achievement of clinical development and commercialization milestones and royalties based on sales of any resulting drugs. Under our existing partnered program agreements, we have the potential to earn over $3.5 billion in additional milestone payments if we or our collaborators achieve the drug discovery, development and commercialization objectives detailed in those agreements. We also have the potential to earn royalties on any resulting product sales or share in the proceeds from development or commercialization arrangements resulting from 12 drug research and development programs.

Additionally, we have a portfolio of proprietary and partnered drug discovery programs generated by our internal discovery efforts. Our internal drug discovery programs include inhibitors that target Trk receptors for the treatment of pain and G-protein-coupled receptor 119, or GPR-119 for the treatment of diabetes. We may choose to out-license select promising candidates through research partnerships.

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Business History

We have built our clinical and discovery pipeline programs through spending $464.1 million from our inception in 1998 through June 30, 2011. In fiscal 2011, we spent $63.5 million in research and development expenses for proprietary drug discovery, compared to $72.5 million and $89.6 million for fiscal years 2010 and 2009, respectively. Over the past 20 months through the date of filing this Annual Report, we signed strategic collaborations with Amgen, Genentech and Novartis. Together these collaborations resulted in $133 million in initial payments, and entitle Array to receive up to over $2.2 billion in potential milestone payments if all clinical and commercialization milestones under the agreements are achieved, double digit royalties and/or commercial co-detailing rights. We have received a total of $523.3 million in research funding and in up-front and milestone payments from our collaboration partners since inception through June 30, 2011.

Our Strategy

We are building a fully integrated, commercial-stage biopharmaceutical company that discovers, develops and markets safe and effective small molecule drugs to treat patients afflicted with cancer and inflammatory diseases. We intend to accomplish this through the following strategies:

Invent targeted small molecule drugs that are either first-in-class or second generation drugs that demonstrate a competitive advantage over drugs currently on the market or in clinical development;

Partner our drugs for co-development and commercialization, selectively retaining U.S. commercial and/or co-promotion rights for drugs that can be distributed through a therapeutically specialized sales force;

Partner select early-stage programs for continued research and development to receive research funding plus significant milestone payments and royalties; and

Build a commercial capability to position our drugs to maximize their overall value. As our first drug nears approval, we plan to build a U.S.-based therapeutically-focused sales force to commercialize or co-promote our drugs.

Our out-license and collaboration agreements with our partners typically provide for up-front payments, research funding, success-based milestone payments, co-detailing rights and/or royalties on product sales. These agreements may also be structured to share in the proceeds received from a collaborator resulting from the further development or commercialization of resulting drugs.

We also have a large number of research and development programs and are partnering certain of these programs with collaborators to provide funding, development, manufacturing and commercial resources. These partnering activities are central to our strategy over the next several years and may include co-development or co-commercialization and may be worldwide or limited to certain geographic areas. We plan to advance our most promising development assets internally at least through clinical proof-of-concept before partnering them, which we believe will maximize their value. We are also identifying certain programs to partner earlier during discovery or preclinical development with the goal of optimizing the potential return for Array on these programs.

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Discovery and Development Programs

We have collaborations with leading pharmaceutical and biotechnology companies under which we have out-licensed certain proprietary drug programs for further research, development and commercialization. Our largest or most advanced collaborations include our agreements with Amgen, ASLAN Pharmaceuticals, AstraZeneca, Celgene, Genentech, Roche and Novartis. Under some of these collaborations, such as with Novartis for MEK162, we continue development work that is funded all or in part by our collaborators. Under some of our other partnered programs, our involvement in the development or research phase has ended but we retain the right to receive clinical and commercialization milestones and/or royalties on sales of any products covered by the collaboration. We also have research partnerships with leading pharmaceutical and biotechnology companies, for which we design, create and optimize drug candidates and conduct preclinical testing across a broad range of therapeutic areas, on targets selected by our partners. In certain of these partnerships, we also perform process research and development, perform clinical development and manufacture clinical supplies.

Information about our collaborators that comprise 10% or more of our total revenue and information about revenue we receive within and outside the U.S. can be found in *Note 2 Segments, Geographical Information and Significant Collaborators* to the accompanying audited financial statements included elsewhere in this Annual Report.

Development Programs

Below is a description of the five most advanced programs that we are developing, their stage in the drug development process and our expected future development plans. Each of these programs is wholly-owned by Array.

Drug Candidates		Current Development Status	Future Development Plan
ARRY-520	KSP	Phase 2 single-agent and Phase 1b combination trials in patients with multiple myeloma.	Complete the Phase 2 and 1b trials.
ARRY-614	p38/Tie2	Phase 1 expansion trial at the maximal administered dose in myelodysplastic syndromes patients and Phase 1 trial with new formulation in healthy volunteers.	Initiate Phase 1 dose escalation in myelodysplastic syndromes patients with the new formulation.
ARRY-380	HER2	Phase 1 expansion trial in patients with metastatic breast cancer at the maximum tolerated dose in cancer patients.	Seek partner for further development.
ARRY-797	p38	Phase 2 randomized, double-blind study in osteoarthritis patients.	Complete the Phase 2 trial and plan Phase 2 acute pain trial.
ARRY-502	CRTh2	Phase 1 multiple ascending dose trial in healthy volunteers.	Initiate 28-day Phase 2a trial in patients with asthma

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1. ARRY-520 KSP Program for Cancer

ARRY-520 inhibits kinesin spindle protein, or KSP, which plays an essential role in mitotic spindle formation. KSP inhibitors induce proliferating cells to die by disrupting mitotic spindle formation during cell division. Unlike other mitosis inhibitors, such as taxanes and vinca alkaloids, KSP inhibitors are not expected to cause certain side effects such as peripheral neuropathy and alopecia.

In preclinical models of acute myeloid leukemia (AML) and multiple myeloma (MM), including MM models that do not respond to Velcade® (bortezomib), treatment with ARRY-520 resulted in significant tumor regression. ARRY-520 also showed synergy in combination with Velcade in several preclinical MM models. This activity was accompanied by a significant increase in apoptosis. ARRY-520 also showed synergy when combined with Revlimid® (lenalidomide) in preclinical MM models.

When administered alone, ARRY-520 has shown promising preliminary clinical activity in patients with relapsed and refractory MM who were previously treated with both an immunomodulatory agent, or IMiD, (such as Revlimid, Thalomid® (thalidomide) or pomalidomide) and a proteasome inhibitor (such as Velcade or carfilzomib). Array presented interim results of a Phase 1 trial of ARRY-520 in patients with MM at the 2010 Annual Meeting of the American Society of Hematology. Among the 30 evaluable patients enrolled in the Phase 1 trial across all dose levels, two partial responses and two minimal responses were reported and eight patients experienced stable disease or better for more than six months. Array plans to report updated Phase 1 and initial Phase 2 data from this study by the end of 2011.

Development Status: Our clinical development activities for ARRY-520 consisted of the following during fiscal 2011:

- Completed a Phase 1 trial in patients with solid tumors
- Completed a Phase 1 trial in patients with AML
- Completed enrollment in a Phase 1 trial in patients with relapsed and refractory MM
- Initiated a Phase 2 expansion cohort of ARRY-520 in combination with dexamethasone in refractory MM
- Initiated a Phase 1b study of ARRY-520 in combination with Velcade and dexamethasone in relapsed and refractory MM
- Continued enrollment in a Phase 2 single agent trial of ARRY-520 in MM

During fiscal 2012, we plan to:

- Complete the ongoing Phase 2 single-agent study, including completing the expansion cohort of ARRY-520 in combination with dexamethasone in Revlimid and Velcade in patients with dual-refractory MM
- Initiate a Phase 1b combination study with Revlimid in patients with relapsed and refractory MM
- Collaborate with M.D. Anderson and Onyx Therapeutics, Inc. on an investigator-sponsored Phase 1b combination study with carfilzomib in patients with refractory MM

2. ARRY 614 p38 /Tie-2 for Myelodysplastic Syndrome Program

ARRY-614, an orally active compound that inhibits both p38 and Tie-2, has been found to block cytokine/chemokine signaling production and attenuate apoptosis. ARRY-614 demonstrates inhibition of inflammation and cytokine-dependent tumor growth in preclinical models.

Myelodysplastic syndromes (MDS) are late onset diseases that are characterized by over-production of myelosuppressive cytokines which leads to aberrant apoptosis in hematological progenitor cells. p38

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MAP kinase (p38) is implicated in dysregulation of apoptosis and myelosuppressive cytokine signaling and production. Tie-2 may affect this process by promoting cytokine production and altering stromal cell senescence. It is hypothesized that disrupting cytokine-driven apoptosis in the normal progenitors and stromal cells may improve hematopoiesis in lower risk MDS patients.

In a Phase 1 clinical trial in healthy volunteers, ARRY-614 demonstrated dose-dependent suppression of IL-1β, PGE2, IL-6 and TNFα, as measured in ex vivo LPS-stimulated whole blood samples. Based on these data, as well as work done on Tie-2 in other experiments, the plasma exposure observed in the volunteers was predicted to result in significant inhibition of p38 and Tie-2 activity in human tissue and plasma.

Development Status: During fiscal 2011, we completed a Phase 1 trial in patients with MDS to determine safety, maximum tolerated dose and pharmacokinetics, and to obtain preliminary efficacy data, of ARRY-614 in this patient population. We also initiated and completed a clinical study in healthy subjects to evaluate a new formulation. Over the next fiscal year, we plan to initiate a second dose-escalation Phase 1 trial in patients with MDS using the improved formulation.

3. ARRY-380 HER2 Program for Cancer

ARRY-380 is an orally active, reversible and selective HER2 inhibitor. HER2, also known as ErbB2, is a receptor tyrosine kinase that is over-expressed in breast cancer and other cancers such as gastric and ovarian cancer. In multiple preclinical tumor models, ARRY-380 was well tolerated and demonstrated significant dose-related tumor growth inhibition that was superior to Herceptin® (trastuzumab) and Tykerb® (lapatinib). Additionally, in these models, ARRY-380 was well tolerated and additive for tumor growth inhibition when dosed in combination with the standard of care therapeutics Herceptin or Taxotere® (docetaxel).

In December 2010, Array presented positive interim results of ARRY-380 in a Phase 1 trial in patients with advanced cancer at the San Antonio Breast Cancer Symposium. Interim results were presented on 19 patients with HER2-positive cancer evaluable for response who were treated with ARRY-380 at doses greater than or equal to 200 mg (twice daily). All of the HER2-positive metastatic breast cancer patients had been previously treated with Herceptin and 81% were previously treated with Tykerb. Thirty two percent of the 19 patients had clinical benefit as measured by a partial response or stable disease for six months or longer. Fifteen of the 19 patients had measurable disease as defined by the Response Evaluation Criteria in Solid Tumors (RECIST); of these patients, seven had regressions in target lesions. Of the four patients with no measurable disease, three had regressions of non-target chest wall lesions. ARRY-380 demonstrated an acceptable safety profile; the predominant treatment-related adverse events have been Grade 1. Because ARRY-380 is selective for HER2 and does not inhibit EGFR, there was, as expected, a low incidence and severity of diarrhea, rash and fatigue. Additionally, there were no Grade 4 treatment related adverse events or treatment related cardiac events reported. The maximum tolerated dose of ARRY-380 established in this Phase 1 trial is 600 mg (twice daily). An expansion cohort in patients with HER2-positive metastatic breast cancer is ongoing to confirm safety and investigate pharmacodynamic markers.

Recently, a sub-population of approximately one-third of HER2+ patients who express high level of truncated HER2 has been identified. This sub-population has been reported to have shorter progression free survival and overall survival from Herceptin-based therapy as compared to patients with low expression of truncated HER2. ARRY-380 targets the intracellular kinase portion of HER2 and retains activity against the truncated HER2, making this population of patients a potentially attractive option for further drug development of ARRY-380.

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Development Status: During fiscal 2011, we completed the dose escalation Phase 1 trial to evaluate the safety, maximum tolerated dose and pharmacokinetics of ARRY-380 in patients with advanced cancer. The drug was shown to have good pharmacokinetics, an acceptable safety profile, and anti-tumor activity in patients who had previously received Herceptin and Tykerb. ARRY-380 is currently in an expansion portion of the Phase 1 trial to confirm safety and the recommended dose for future trials. We are seeking a partner to further advance the program.

4. ARRY-797 p38 Program for Pain

p38 MAPK is an important mediator of pain and inflammation which modulates the production of the pro-inflammatory cytokines TNF, IL-6 and IL-1 as well as the pain mediator PGE2. ARRY-797 is an orally active inhibitor of p38 with unique physical properties: it is highly selective, highly water soluble and has low potential to cross the blood brain barrier. In a Phase 1 clinical trial in healthy volunteers, ARRY-797 demonstrated dose-dependent marked suppression of all three of these cytokines, as measured in ex vivo LPS-stimulated whole blood samples.

To date, in Phase 1 and 2 studies, 417 individuals have received up to twelve weeks of ARRY-797 and the drug has been well tolerated. Among these individuals, pain data are available for 309 patients from four studies. In 2008, Array announced top-line results demonstrating that ARRY-797 achieved its primary endpoints for analgesic efficacy in two Phase 2 acute dental pain studies. ARRY-797 was well tolerated with no serious adverse events. In 2009, post hoc analyses of two studies with a small number of patients: a 28-day rheumatoid arthritis study and a 12-week ankylosing spondylitis study indicated durable pain relief with ARRY-797. Array believes ARRY-797 has an opportunity to address a significant unmet medical need in both acute and chronic pain.

ARRY-797 is currently being evaluated in a 28-day Phase 2 trial of patients with pain associated with osteoarthritis of the knee who are using concomitant nonsteroidal anti-inflammatory drugs. Array anticipates reporting top-line results of this trial during the first quarter of calendar 2012.

5. ARRY-502 CRTh2 Program for Asthma

ARRY-502, an oral CRTh2 antagonist, has the potential to be an effective treatment for patients with asthma, particularly those with severe conditions, and may have advantages over competitor molecules. In various preclinical models of allergic inflammation, ARRY-502 has demonstrated a high level of anti-inflammatory activity. In initial Phase I clinical trials ARRY-502 has been well tolerated and demonstrated pharmacodynamic activity.

Inappropriate inflammatory responses to environmental allergens underlie allergic reactions such as allergic asthma, allergic rhinitis and atopic dermatitis, which collectively affect up to 20% of the United States population. Despite the range of treatments used to treat allergic asthma, there remains a significant need for patients with severe asthma as well as for more convenient and safer therapies for those with mild to moderate asthma. Although severe asthma affects only approximately 10% of the asthmatic population, the condition results in approximately 60% of total healthcare costs associated with asthma. Currently, few treatment options exist for patients with severe asthma.

In severe allergic asthma, there is emerging evidence suggesting that a greater presence of the mediator prostaglandin D2, or PGD2, and an upregulation of CRTh2, a protein receptor for PGD2 that is expressed on inflammatory cells, may play a particularly important role in greater symptoms of asthma such as coughing and difficulty breathing and lower lung function. Indeed, activation of CRTh2 has been shown to result in chemotaxis and activation of inflammatory cells and stimulate the production of cytokines that are thought to drive asthma pathophysiology.

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Based on the role of CRTh2 in mediating the actions of PGD2, selective antagonism of CRTh2 presents an attractive therapeutic approach to the treatment of severe allergic conditions. There are selective antagonists of CRTh2 in various stages of clinical development with compounds currently being evaluated in early Phase 2 studies in allergic rhinitis, asthma and eosinophilic esophagitis.

Development Status: Array initiated a 14-day Phase 1, randomized, double-blind, multiple ascending dose trial with ARRY-502 in healthy volunteers for the evaluation of safety, pharmacokinetics and pharmacodynamics. Array expects to complete enrollment and announce top-line results by year-end. The results to date from the Phase 1 single ascending dose study indicate that ARRY-502 has been well-tolerated at the doses evaluated, has shown excellent exposure and demonstrated good activity in pharmacodynamic assessments. Array expects to initiate a 28-day Phase 2a trial in persistent asthma over the next fiscal year.

Partnered Development Programs

Below are summaries of our most advanced, ongoing partnered development programs. Any information we report about the development plans or the progress or results of clinical trials or other development activities of our partners is based on information that has been reported to us or is otherwise publicly disclosed by our collaboration partners.

1. AstraZeneca Selumetinib and AZD8330 MEK Program

In December 2003, we entered into an out-licensing and collaboration agreement with AstraZeneca to develop our MEK program. Under the agreement, AstraZeneca acquired exclusive worldwide rights to our clinical development candidate, selumetinib (previously known as ARRY-142886), together with two other compounds for oncology indications, including AZD8330, which we invented during the collaboration. We retained the rights to all therapeutic indications for MEK compounds not selected by AstraZeneca for development, subject to the parties' agreement to work exclusively together. In April 2009, the exclusivity of the parties' relationship ended and both companies are now free to independently research, develop and commercialize small molecule MEK inhibitors in the field of oncology. To date, we have earned $21.5 million in up-front and milestone payments. The agreement also provided for research funding, which is now complete, and provides potential additional development milestone payments of approximately $75 million and royalties on product sales.

Under this collaboration, we were responsible for Phase 1 clinical testing, which we completed in 2004, and AstraZeneca is responsible for all future development and commercialization of the compounds under the collaboration.

Development Status. The Phase 1 trial Array conducted in 2004 evaluated tolerability and pharmacokinetics of selumetinib following oral administration to patients with advanced cancer. In addition, the trial examined patients for indications of biological activity as well as pharmacodynamic and tumor biomarkers. Selumetinib inhibited the MEK pathway in tumor tissue at the dose that was later selected for Phase 2 studies and provided prolonged disease stabilization in a number of cancer patients who had previously received numerous other cancer therapies.

In June 2006, AstraZeneca initiated a Phase 2 study for selumetinib in patients with malignant melanoma, resulting in a $3 million milestone payment to us. The trial was a randomized Phase 2 study that compared selumetinib to Temodar® (temozolomide) in the treatment of patients with stage III/IV melanoma. AstraZeneca enrolled approximately 180 patients at 40 centers worldwide in this study. AstraZeneca also initiated additional Phase 2 studies for selumetinib in colorectal, pancreatic and non-small cell lung cancer during 2006.

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In 2008, AstraZeneca presented Phase 1 clinical trial results at the annual meeting of the American Society of Clinical Oncology, or ASCO, of a new selumetinib capsule formulation that replaces the mix/drink formulation used in all prior trials to that time. AstraZeneca reported that the new capsule's maximum tolerated dose was 25% lower yet provided, on average, higher exposure than historical values for the mix/drink formulation. AstraZeneca also reported a complete response in one of the patients. AstraZeneca also presented at ASCO the following Phase 2 clinical trial results of selumetinib using the prior formulation:

- Selumetinib compared to Alimta® (pemetrexed) in 84 non-small cell lung cancer, or NSCLC, patients: neither of these drugs demonstrated superior efficacy.

- Selumetinib compared to Temodar in patients with advanced melanoma: there was no difference between the two treatment arms in the overall population comparing the safety and tolerability profile for selumetinib and these results were consistent with the results reported from the Phase 1 trial.

- Selumetinib compared to Xeloda® (capecitabine) in patients with metastatic colorectal cancer: results showed that selumetinib was generally well tolerated, with neither of these drugs demonstrating superior efficacy.

AstraZeneca also reported that, in patients suffering from melanomas with RAF mutations in clinical trials, selumetinib provided partial responses in two out of 14 patients using the Phase 2 mix/drink formulation and a complete response in one out of eight patients using the Phase 1 new capsule formulation.

AstraZeneca presented at the 2009 American Association for Cancer Research annual meeting results on a Phase 2 trial of selumetinib that showed a 12% overall response rate among patients with biliary cancer.

In 2010, AstraZeneca presented at the ASCO annual meeting results of a Phase 1 clinical trial using the selumetinib capsule formulation in melanoma patients. This study evaluated two doses of selumetinib (50 mg twice daily and 75 mg twice daily) in combination with four different chemotherapies: DTIC® (dacarbazine) (1000 mg/m^2), Taxotere (75 mg/m^2), Tarceva® (erlotinib) (100 mg daily) or Torisel® (temsirolimus) (25 mg weekly). The study enrolled 25 melanoma patients, 18 of whom had evaluable tumors. Fourteen out of the 18 patients were treated with selumetinib plus DTIC, three with selumetinib plus Taxotere and one with selumetinib plus Torisel. Sixty-seven percent of these patients had previously failed at least one prior systemic treatment. Of the 18 patients, nine had BRAF mutations. Of those patients with BRAF mutations, five had a partial response, four had stable disease with a median time-to-progression of 31 weeks. The other nine patients had wild-type BRAF, five of whom had stable disease and four of whom had progressive disease with a median time-to-progression of eight weeks. The median time to progression difference between BRAF mutant and wild type BRAF was statistically significant (p=0.01, Wilcoxon rank-sum test). Selumetinib plus chemotherapy had a 56% response rate in patients with BRAF mutations, whereas no responses were observed in patients with wild-type BRAF. While the number of patients analyzed is small, the trend toward clinical benefit in patients with BRAF mutation is inferred. This is the first disclosed efficacy data with the new formulation of selumetinib, which provides twice the drug exposure at the preferred dose.

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During 2010, AstraZeneca completed enrollment in two Phase 2 trials with selumetinib, which are the first two randomized Phase 2 combination trials with a MEK inhibitor:

- Selumetinib in combination with DTIC compared with DTIC alone in first-line melanoma patients with BRAF- mutation. The trial completed enrollment of 91 patients in March 2010 with the primary end-point of overall survival.

- Selumetinib in combination with Taxotere compared with Taxotere alone in second-line non-small cell lung cancer patients with KRAS-mutation. The trial completed enrollment of approximately 80 patients in July 2010 with the primary end-point of overall survival.

Selumetinib is currently the subject of the following select additional Phase 2 trials:

- Selumetinib or Temodar in patients with uveal melanoma. One hundred fifty nine patients are anticipated to enroll in this trial.

- Selumetinib in combination with irinotecan in second-line patients with KRAS or BRAF mutation positive advanced or metastatic colorectal cancer. Fifty-seven patients are anticipated to enroll in this trial.

- Selumetinib in combination with Tarceva in non-small cell lung cancer patients with KRAS or KRAS wild-type mutations. One hundred patients are anticipated to enroll in this trial.

- Selumetinib in combination with Nexavar® (sorafenib) in patients with advanced hepatocellular cancer. One hundred patients are anticipated to enroll in this trial.

- Selumetinib in combination with MK-2206 in patients with advanced colorectal cancer. Thirty eight patients are anticipated to enroll in this trial.

In addition to the selumetinib trials described above, AstraZeneca has an ongoing Phase 1 clinical trial with ASD8330 in patients with solid tumors. In March 2007, AstraZeneca reported that it had dosed its first cancer patient in a Phase 1 clinical trial with AZD8330, triggering a $2 million milestone payment to us.

2. Novartis MEK162 and MEK300 MEK Inhibitor Program

In April 2010, we granted Novartis under a License Agreement the exclusive worldwide right to develop and commercialize MEK162, which is currently in multiple Phase 1 and Phase 2 cancer trials. Also included in the agreement were ARRY-300 (also known as MEK300) and other specified MEK inhibitors. Under the agreement, we are responsible for completing the on-going Phase 1 clinical trial of MEK162 and may conduct further development of MEK162 in a specific cancer. Novartis is responsible for all other development activities. Novartis is also responsible for the commercialization of products under the agreement, subject to our option to co-detail approved drugs in the U.S.

In connection with signing the agreement, Novartis paid us $45 million, comprising an upfront fee and an initial milestone payment. In April 2011, we received a $10 million clinical research milestone from Novartis after Novartis had its first patient visit in a Phase 2 clinical trial. We are also eligible under the agreement to receive up to approximately $412 million in aggregate milestone payments if all clinical, regulatory and commercial milestones specified in the agreement are achieved for MEK162 and additional commercial milestone payments for MEK300 and other MEK inhibitors Novartis elects to develop under the agreement. The agreement provides Array with double-digit royalties on worldwide sales of any approved drugs, with royalties on U.S. sales at a significantly higher level. We will pay a

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percentage of development costs up to a maximum amount with annual caps. We may opt out of paying such development costs with respect to one or more products; in which case the U.S. royalty rate would then be reduced for any such product based on a specified formula, subject to a minimum that equals the royalty rate on sales outside the U.S. and we would no longer have the right to develop or detail such product.

The agreement with Novartis will be in effect on a product-by-product and country-by-country basis until no further payments are due with respect to the applicable product in the applicable country, unless terminated earlier. Either party may terminate the agreement in the event of a material breach of a material obligation under the agreement by the other party that remains uncured after 90 days prior notice. Novartis may terminate portions of the agreement following a change in control of Array and may terminate the agreement in its entirety or on a product-by-product basis with 180 days prior notice. Array and Novartis have each further agreed to indemnify the other party for manufacturing or commercialization activities conducted by it under the agreement, negligence or willful misconduct or breach of covenants, warranties or representations made by it under the agreement.

Research suggests that the MEK pathway acts as an important axis in the proliferation of some common human tumors including melanoma, non-small cell lung, head, neck and pancreatic cancers. Increasing evidence suggests that MEK inhibition, either alone or in combination with other agents, may become an important therapeutic strategy in treating cancer. We believe MEK162 will be most effective in selected populations of cancer patients, such as those with tumors having $BRAF^{V600E}$ or KRAS mutations as well as in targeted combinations. MEK162 has been administered to more than 300 patients/volunteers in clinical trials for either safety assessment or the treatment of oncology or inflammatory disease. The drug has demonstrated an acceptable safety profile and has demonstrated significant pharmacodynamic responses in the completed trials.

Development Status: During fiscal 2011, we completed enrollment of Phase 1 dose expansion cohorts in patients with biliary tract cancer, and patients with KRAS mutant colorectal cancer and initiated a Phase 1 dose expansion cohort in patients with BRAF mutant colorectal cancer. In addition, Novartis initiated a Phase 2 open-label study to assess the safety and efficacy of MEK162 in patients with locally advanced and unresectable or metastatic malignant cutaneous melanoma harboring $BRAF^{V600E}$ or NRAS- mutations. The trial is designed to measure the objective response rate to treatment with MEK162 when administered orally to patients. The trial will also evaluate progression-free survival, safety and tolerability. In addition, Novartis initiated three Phase 1b combination trials over the past three months:

Safety, pharmacokinetics and pharmacodynamics of BEZ235 plus MEK162 in selected advanced solid tumor patients

Safety, pharmacokinetics and pharmacodynamics of BKM120 plus MEK162 in selected advanced solid tumor patients

MEK162 and RAF265 in adult patients with advanced solid tumors harboring RAS or $BRAF^{V600E}$ mutations

3. InterMune (being developed by Roche) Danoprevir Hepatitis C Virus NS3/4 Protease Program

In 2002, we entered into a collaboration with InterMune for the discovery of novel small molecule inhibitors of the Hepatitis C Virus, or HCV, NS3/4A protease. Under the terms of Array's collaboration agreement with InterMune, InterMune funded certain drug discovery efforts, preclinical testing, process development and manufacturing in conformity with current Good Manufacturing Practices, or cGMP.

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InterMune will make milestone payments to us based on the selection and progress of clinical drug candidates, as well as royalties on sales of any products derived from the collaboration. To date, we have received $1.8 million in milestone payments and have the potential to earn an additional $9.0 million if all clinical and commercialization milestones are achieved under the agreement.

Development Status: From 2002 to 2007, scientists from Array and InterMune collaborated on discovery activities that resulted in the joint discovery of danoprevir, which was acquired by Roche in October 2010 for $175 million. During 2008, InterMune advanced danoprevir in a Phase 1b multiple ascending dose clinical trial evaluating danoprevir in combination with standard of care therapies in treatment-naive patients with chronic HCV genotype 1 infection. Results from the trial showed that danoprevir in combination with standard of care resulted in rapid and persistent reductions in HCV RNA in the patients. In addition, viral rebound was not observed in any patients receiving the treatment and danoprevir in combination with standard of care was safe and generally well-tolerated over 14 days.

During 2009, InterMune initiated a Phase 2b trial evaluating danoprevir in combination with standard of care therapies. In April 2010, InterMune announced top-line results from a planned interim analysis of the trial. Danoprevir was administered at either 300 mg three times daily, 600 mg twice daily or 900 mg twice daily for 12 weeks in combination with PEGASYS® (pegylated interferon alfa-2a) and COPEGUS® (ribavirin), compared with placebo for the same duration plus PEGASYS and COPEGUS. In November 2009, InterMune reported that due to a safety signal, dosing in the 900 mg group had been stopped. InterMune reported that results from the study indicate danoprevir plus PEGASYS and COPEGUS are capable of achieving complete early virologic response rates as high as 90% compared to 43% in the placebo group. In addition, InterMune completed a Phase 1b trial (INFORM-1) of danoprevir and a polymerase inhibitor, RG7128.

Development Status: Danoprevir is currently being tested in the following trials:

Phase 2b trial with boosted danoprevir, PEGASYS and COPEGUS in genotype 1 +4, which enrolled 421 patients

Phase 2b trial with boosted danoprevir in triple, quad and interferon-free combinations which expects to enroll 421 patients

4. ASLAN Pharmaceuticals ARRY-543 HER2 /EGFR Program

In July 2011, we entered into a collaboration agreement with ASLAN Pharmaceuticals Pte Ltd to develop Array's HER2 / EGFR inhibitor, ARRY-543, which is currently entering Phase 2 development for solid tumors. Under the agreement, ASLAN will fund and develop ARRY-543 through clinical proof of concept, initially targeting patients with gastric cancer through a development program conducted in Asia. Upon achievement of proof of concept, ASLAN will identify a global partner for Phase 3 development and commercialization. Array will share a significant portion of the proceeds of such partnering transaction.

The agreement with ASLAN will remain in effect for two years after conclusion of the initial development plan, unless ASLAN has entered into a license agreement with a third party for the further development and commercialization of the program, in which case the agreement shall remain in force and effect. Either party may terminate the agreement prior to expiration of the term following breach of the agreement by the other party. ASLAN is responsible for diligently advancing development ARRY-543 under an agreed upon development plan.

ARRY-543 is a novel, selective and oral HER2 / EGFR inhibitor, and has shown clinical activity in both HER2-positive and EGFR-positive tumors. Over 200 patients have received ARRY-543 either as monotherapy or in combination with chemotherapy.

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Gastric cancer is a major public-health problem in East Asia. Patients with locally advanced, metastatic or recurrent disease have a poor prognosis, with an overall median survival of approximately 11 months. EGFR and HER2 receptors are commonly overexpressed together in gastric cancer. Recent data from pivotal studies of Herceptin indicate that the activity of this drug is limited to the subset of patients whose disease has amplified copies of the HER2 gene. We believe ARRY-543 has the potential to augment or supersede the activity of Herceptin in this population, and in the broader population of gastric cancers that co-express both EGFR and HER2 receptors.

In a Phase 1 trial, ARRY-543 produced prolonged stable disease in patients with solid tumors who had previously failed prior treatments. Tablets of ARRY-543 were well-tolerated up to 500 mg twice daily dosing. Systemic concentrations of ARRY-543 increased with escalating doses at all dose levels tested. Sixty percent of patients receiving doses of 200 mg twice daily and higher had prolonged stable disease.

In a Phase 1 expansion cohort in patients with HER2-positive metastatic breast cancer or other ErbB-family cancer, ARRY-543 was generally well tolerated and demonstrated evidence of tumor regression and prolonged stable disease in EGFR- and HER2-expressing cancers. Twenty-one metastatic breast cancer patients were evaluated: of the 12 with available biopsies, eight were confirmed HER2-positive. Of the confirmed patients with HER2-positive metastatic breast cancer in this study, 63% had stable disease. Clinical benefit (measured by tumor regression or stable disease) was demonstrated in five of the eight confirmed HER2 patients and patients with confirmed co-expression of HER2 and EGFR tended to have the best clinical benefit. In a cohort of patients with other cancers shown to over-express HER2 and EGFR, a patient with cholangiocarcinoma experienced a tumor marker response that was accompanied by a 25% regression of target lesions.

Development Status: During fiscal 2011, we achieved the maximum tolerated dose and completed enrollment in three Phase 1b studies of ARRY-543 in combination with Xeloda, Taxotere and Gemzar® (gemcitabine) in patients with solid tumors. In July 2011, ASLAN began funding further development of ARRY-543 through clinical proof of concept, initially targeting patients with gastric cancer through a development program conducted in Asia.

5. Eli Lilly LY2603618 CHK-1 Inhibitor Program

In 1999 and 2000, Array entered into collaboration agreements involving small molecule ChK-1 inhibitors with ICOS Corporation. IC83 resulted from the collaboration between Array and ICOS. Eli Lilly and Company acquired ICOS in 2007. Array received a $250 thousand milestone payment after the first patient was dosed with IC83, now LY2603618, in a Phase 1 clinical trial in early 2007. The agreements provided research funding, which has now ended. Array is entitled to receive additional milestone payments totaling $3.5 million based on Eli Lilly's achievement of clinical and regulatory milestones with LY2603618.

Development Status: LY2603618 is currently in multiple Phase 1b/2 clinical trials, including four that began during the first half of 2011, in cancers such as non-small cell lung and pancreatic.

6. Amgen AMG 151 Glucokinase Activator for Type 2 Diabetes Program

In December 2009, Array granted Amgen the exclusive worldwide rights to our small molecule glucokinase activator (GKA) program, including AMG 151. Under the Collaboration and License Agreement with Amgen, we were responsible for completing certain Phase 1 clinical trials of AMG 151, which we completed during fiscal 2011. Amgen is also funding an agreed upon number of full-time Array employees as part of the research collaboration intended to identify and advance second-generation GKAs. Amgen is responsible for the further development and commercialization of AMG 151 and any

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resulting second-generation compounds. The agreement also provides Array with an option to co-promote any approved GKAs with Amgen in the U.S. with certain limitations.

In partial consideration for the rights granted to Amgen under the agreement, Amgen paid an up-front fee of $60 million. Array is also entitled to receive up to approximately $666 million in aggregate milestone payments if all clinical and commercialization milestones specified in the agreement for AMG 151 and at least one backup compound are achieved. We will also receive royalties on sales of any approved drugs developed under the agreement.

The agreement with Amgen will remain in effect on a product-by-product and country-by-country basis until no further payments are due under the agreement unless terminated earlier. Either party may terminate the agreement in the event of a material breach of a material obligation under the agreement by the other party that remains uncured after 90 days prior notice. Amgen may terminate the agreement at any time upon notice of 60 or 90 days depending on the development activities going on at the time of such notice. The parties have also agreed to indemnify each other for certain liabilities arising under the agreement.

GKAs, such as AMG 151, represent a promising new class of drugs for the treatment of Type 2 diabetes. Glucokinase is the enzyme that senses glucose in the pancreas. Glucokinase also increases glucose utilization and decreases glucose production in the liver. GKAs regulate glucose levels via a dual mechanism of action - working in both the pancreas and the liver. The activation of glucokinase lowers glucose levels by enhancing the ability of the pancreas to sense glucose, which leads to increased insulin production. Simultaneously, GKAs increase the net uptake of blood glucose by the liver. In multiple well-established preclinical models of Type 2 diabetes, AMG 151 was highly efficacious in controlling both fasting and non-fasting blood glucose, with rapid onset of effect and maximal efficacy within five to eight once daily doses. When combined with existing standard-of-care drugs (metformin, Januvia® (sitagliptin) or Actos® (pioglitazone), AMG 151 provided additional glucose control, which reached maximal efficacy after five to seven days of once-daily dosing. AMG 151 did not increase body weight, plasma triglycerides or total cholesterol, whether used as monotherapy or in combination with other diabetes drugs.

Development Status: During fiscal 2011, Array completed two Phase 1 studies, a multiple ascending dose trial in patients with Type 2 diabetes to evaluate safety, exposure and glucose control over a 10-day period and a relative bioavailability study assessing the effect of food and formulation on exposure. Amgen is responsible for all future development.

7. Genentech GDC-0068, GDC-0425 (RG7602), ARRY-575 and other Oncology Programs

We entered into a licensing and collaboration agreement with Genentech in December 2003 to develop small molecule drugs against multiple therapeutic targets in the field of oncology. We initiated this collaboration to advance two of our proprietary oncology programs into clinical development. These programs included small molecule leads we had developed along with additional, related intellectual property. Under the agreement, Genentech made an up-front payment, provides research funding and has so far paid us milestones for nominating a clinical candidate and advancing it into regulated safety assessment testing and Phase 1. In addition, Genentech has agreed to make additional potential development milestone payments and pay us royalties on any resulting product sales. Genentech is solely responsible for clinical development and commercialization of the resulting products.

In 2005, 2008 and 2009, we expanded our collaboration with Genentech to develop clinical candidates directed against an additional third, fourth and fifth target, respectively. Under the agreement, we receive additional research funding, as well as potential research and development milestone payments and product royalties based on the success of each new program. Genentech has paid Array a total of

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$15.5 million in up-front and milestone payments, and we have the potential to earn an additional $60 million for all programs if Genentech continues development and achieves the remaining clinical milestones set forth in the agreement.

In September 2010, we and Genentech extended the agreement for an additional two years of funded research through January 2013. Genentech may terminate the agreement upon 120 days' notice.

In June 2011, Genentech disclosed that one collaborative drug, GDC-0068, an AKT inhibitor, was advancing to a Phase 1b, open label, dose escalation study of the safety and pharmacology of GDC-0068 in combination with either Taxotere or fluoropyrimidine plus oxaliplatin in patients with advanced solid tumors.

In August 2011, Array and Genentech, a member of the Roche Group, entered into an oncology agreement with for the development of each company's small-molecule Checkpoint kinase 1 (ChK-1) program. The programs include Genentech's compound GDC-0425 (RG7602), currently in Phase 1, and Array's compound ARRY-575, which is being prepared for an investigational new drug application to initiate a Phase 1 trial in cancer patients. Under the terms of the agreement, Genentech is responsible for all clinical development and commercialization activities. Array will receive an upfront payment of $28 million and is eligible to receive clinical and commercial milestone payments up to $685 million and up to double-digit royalties on sales of any resulting drugs. The agreement will remain in effect until Genentech's obligations to make milestone or royalty payments have passed or expired. Either party may terminate the agreement prior to expiration of the term following breach of the agreement by the other party, and Genentech may terminate the agreement upon at least 60 days' prior notice to Array. If Genentech terminates the agreement for breach of the agreement by Array, the license Array granted to Genentech will become irrevocable and the royalty payable to Array will be reduced to a specified percentage. If the agreement is terminated by Genentech for convenience or by Array for breach of the agreement by Genentech, the licenses Array granted to Genentech will terminate, Genentech will continue to be required to pay milestone and royalty payments on any programs for which Genentech had initiated clinical development and Array's exclusivity obligations will continue so long as Genentech is developing or commercializing at least one product subject to the agreement. Array and Genentech have also agreed to indemnify the other party for breaches of representations or warranties made under the agreement and for certain of their respective activities under the agreement.

8. VentiRx VTX-2337 and VTX-1463 /Toll-Like Receptor (TLR) Program

In February 2007, we entered into a licensing and collaboration agreement with the privately held biopharmaceutical company VentiRx, under which we granted VentiRx exclusive worldwide rights to certain molecules from our toll-like receptor, or TLR, program. The program contains a number of compounds targeting TLRs to activate innate immunity. We received equity in VentiRx as well as an up-front payment and the right to receive potential milestone payments and royalties on product sales. To date, we have received $1.1 million in milestone payments and have the potential to earn $57.5 million if VentiRx achieves the remaining clinical and commercial milestones under the agreement. See *Note 5 Equity Investment* to the accompanying audited financial statements included elsewhere in this Annual Report on Form 10-K for a description of the equity interest we received in VentiRx as a result of this agreement.

VentiRx has reported that it completed Phase 1 clinical trials on its first two candidates, VTX-2337 in cancer and VTX-1463 in allergy. VentiRx reported results from both trials at recent scientific meetings. Phase 1 results on VTX-1463 were reported at The American Academy of Allergy & Immunology (AAAAI) 2011 Annual Meeting in March 2011. This clinical trial assessed safety and efficacy of VTX-1463 in a randomized, placebo-controlled study in 80 patients with confirmed allergy to grass pollen. The patients

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were divided into two dosing regimens or received placebo. Group A received ascending doses of 25, 50, 75 and 100 micrograms once weekly for four weeks. Group B received once weekly administrations of 62.5 micrograms for four weeks. Patients underwent grass pollen exposure on Day 24. Both treatment groups demonstrated statistically significant improvement in the primary endpoint: allergy symptoms based on the Total Nasal Symptom Score (TNSS), a sum of scores for nasal congestion, itching, sneezing and rhinorrhea, compared to placebo (p=0.012 for group A; p=0.008 for Group B). TNSS is the key regulatory endpoint for allergic rhinitis. According to the AAAAI, allergic rhinitis, also known as hay fever, affects 60 million people in the U.S. Treatment was generally well-tolerated.

Phase 1 results on VTX-2337 were reported at the 2011 American Society of Clinical Oncology Annual Meeting in June 2011. Overall, VTX-2337 was well-tolerated, with the most common drug-related adverse events being mild to moderate in severity and including injection-site reactions and transient flu-like symptoms. The maximum tolerated dose of VTX-2337 was established to be 3.9 mg/m2. In addition, pharmacodynamic effects as measured by a defined panel of biomarkers identified in preclinical studies provide evidence of the biological activity of VTX-2337 in stimulating an innate immune response in cancer patients. Twenty five percent of patients (N=8) treated with VTX-2337 experienced disease stabilization based on RECIST criteria at eight weeks. Patients with disease stabilization at eight weeks received additional doses of VTX-2337, ranging from 1 to 6 additional cycles (3 to 18 additional doses), until disease progression. One patient with metastatic melanoma demonstrated tumor regression after cessation of VTX-2337 remains disease free at 18 months post-treatment.

VentiRx has also reported that it plans to advance a broad clinical development program for VTX-2337, with four clinical studies targeted to begin in 2011. These trials will evaluate VTX-2337 in multiple oncology indications in combination with a variety of anticancer agents, including chemotherapy, monoclonal antibody therapy and radiation therapy.

9. Celgene ARRY-382 and other Oncology and Inflammation Programs

In September 2007, we entered into a worldwide strategic collaboration with Celgene focused on the discovery, development and commercialization of novel therapeutics in cancer and inflammation. Under the agreement, Celgene made an upfront payment of $40 million to us in part to provide research funding for activities conducted by Array. We are responsible for all discovery and clinical development through Phase 1 or Phase 2a. Celgene has an option to select a limited number of drugs developed under the collaboration that are directed to up to two of four mutually selected discovery targets and will receive exclusive worldwide rights to the drugs, except for limited co-promotional rights in the U.S. Celgene's option may be exercised with respect to drugs directed at any of the four targets at any time until the earlier of completion of Phase 1 or Phase 2a trials for the drug or September 2014. Additionally, we are entitled to receive, for each drug, potential milestone payments of $200 million if certain discovery, development and regulatory milestones are achieved and an additional $300 million if certain commercial milestones are achieved. We will also receive royalties on net sales of any drugs. We retain all rights to the other programs.

In June 2009, the parties amended the agreement to substitute a new discovery target in place of an existing target and Celgene paid Array an up-front fee of $4.5 million in consideration for the amendment. In September 2009, Celgene notified us that it was waiving its rights to one of the programs leaving Celgene the option to select two of the remaining three targets. In April 2010, Celgene announced names of three of our collaborative research programs: cFMS (oncology), TYK2 (inflammation) and PDGFR (fibrosis). In November 2010, Array received a $10 million milestone payment upon filing an IND application for ARRY-382, a cFMS inhibitor, which is currently in a Phase 1 clinical trial.

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Celgene may terminate the agreement in whole, or in part with respect to individual drug development programs for which Celgene has exercised its option, upon six months' written notice to us. In addition, either party may terminate the agreement, following certain cure periods, in the event of a breach by the other party of its obligations under the agreement. Celgene can also choose to terminate its participation in any drug development program for which they have not exercised an option at any time, provided that they must give us prior notice, generally less than 30 days. In this event, all rights to the program remain with Array and we would no longer be entitled to receive milestone payments for further development or regulatory milestones we achieve if we choose to continue development of the program.

Market Opportunity

Our proprietary pipeline is focused on targeted drugs that treat cancer and inflammatory diseases and related pain. We believe there is a substantial opportunity in creating drugs for these diseases that meet the demand from the medical community for targeted therapies that treat both the underlying disease as well as control symptoms more effectively and/or more safely than drugs that are currently available. We believe future patient care will improve with the use of screening to select targeted therapies for more effective disease treatment. Also, clinical trials aimed at well-defined patient populations may show improved response rates and may thereby increase the chances for approval with regulatory agencies such as the U.S. Food and Drug Administration, or FDA. This approach may result in a greater number of marketed drugs each aimed at a smaller subset of patients.

The worldwide market for targeted cancer drugs the cancer drug market's fastest growing segment is forecast to grow from $35.0 billion in 2010 to $71.2 billion in 2016. There remains a large need to address patients with acute or subacute pain, such as postoperative pain and musculoskeletal pain, as well as pain from chronic conditions such as osteoarthritis pain and chronic lower back pain. The worldwide market for key classes of medications used to treat these types of acute, subacute and chronic pain conditions, nonsteroidal anti-inflammatory drugs, or NSAIDs, cyclooxygenase-2, or COX-2, inhibitors, opioids, dual-acting opioids and other non-narcotic analgesics are forecast to grow from $18.6 billion in 2010 to $22.1 billion in 2016. The inflammatory disease market is highly diverse and includes respiratory diseases such as asthma, allergic rhinitis, and chronic obstructive pulmonary disease, or COPD; dermatological conditions such as psoriasis and atopic dermatitis; gastrointestinal disorders such as Crohn's disease and ulcerative colitis; musculoskeletal disorders such as rheumatoid arthritis, systemic lupus erythematosus, or SLE, and gout; and spondyloarthropathies such as psoriatic arthritis and ankylosing spondylitis. The inflammatory disease market is forecast to grow from $70.0 billion in 2010 to $85.8 billion in 2016.

In addition, the pharmaceutical industry has an ongoing need to fill clinical development pipelines with new drugs to drive future revenue growth. Despite increased spending on internal research, the industry has been unable to meet this demand. As a result, it has become increasingly reliant on biotech companies to acquire new drugs. Due to the scarcity of later-stage clinical assets available for in-licensing, these companies have been willing to enter into licensing deals at early stages, including the preclinical stage. However, once a drug has entered clinical development, companies generally require proof-of-concept data, which includes both efficacy and safety data, before they will consider licensing a drug candidate. Accordingly, we believe there is an opportunity to license drugs at several stages during the drug development process.

Cancer Market

Despite a wide range of available cancer therapies, patients' treatment responses remain limited and variable. As a result, oncologists are increasingly using combination therapies and drug dosing regimens tailored for individual tumor types and patients. Targeted therapies are able to specifically target the

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underlying mechanisms of the disease by regulating discrete aspects of cellular function affecting cancer cells to a greater extent than normal cells. As such, they hold the promise of being more efficacious with fewer side effects than cytotoxic chemotherapy drugs. Further, biomarkers are increasingly playing a role in both patient prognosis and drug selection. We believe certain cancers will eventually become chronic diseases, treated with a combination of targeted therapies. Our research strategy in the cancer market is to build a pipeline of targeted therapies.

According to estimates contained in the American Cancer Society, *Cancer Facts and Figures 2011*, in the U.S. there will be an estimated 1.6 million new cases of cancer in 2011 and nearly 600 thousand cancer-related deaths. The five-year relative survival rate for all cancers diagnosed between 1999 and 2006 was 68%. This represents a 50% improvement from 1975 to 1977. Earlier diagnosis and the use of new and/or better treatments have driven this improvement.

The following table shows estimated new cases diagnosed and estimated deaths in the U.S. during 2011 by major cancer types of interest to Array:

	Estimated 2011	
Type of Cancer	**New Cases**	**Deaths**
Lung	221,130	156,940
Breast	232,620	39,970
Colorectal	141,210	49,380
Melanoma	70,230	8,790
Non-Hodgkin Lymphoma	66,360	19,320
Myelodysplastic Syndromes	45,000	unknown
Pancreas	44,030	37,660
Ovarian	21,990	15,460
Stomach	21,520	10,340
Myeloma	20,520	10,610
Acute Myeloid Leukemia	12,950	9,050
Gallbladder and Other Biliary	9,250	3,300
	906,810	360,820

The use of targeted therapies has the potential to change the focus of cancer treatment away from categorization and treatment modality by organ type and towards categorization and treatment modalities by level of gene expression in individual patients, or "personalized medicine." Targeted therapies and personalized medicine hold the promise of increased survival with improved quality of life.

Oncology, both in treating cancer itself and palliative therapy, has been a major therapeutic category for biotechnology companies since the inception of the industry. Recently, major pharmaceutical companies have increased their research and development and in-licensing investment in this market, particularly the targeted cancer therapy market. Some of the targeted therapies currently on the market that have been successful include Avastin®(bevacizumab), Gleevec®(imatinib mesylate), Herceptin and Rituxan®(rituximab).

Multiple Myeloma (ARRY-520 KSP inhibitor)

Multiple Myeloma, or MM, is a hematological cancer in which malignant plasma cells are overproduced in the bone marrow. Normal plasma cells are white blood cells that produce antibodies to fight infection and disease. MM plasma cells replace normal plasma cells that are important to maintaining the immune system.

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MM is the third most common hematologic malignancy, and garners significant sales due to the cost of treatment regimens and relatively long life expectancies among patients. Despite advances in therapy over the last ten years, it remains an incurable, fatal disease in all patients and accounts for approximately 1% of deaths worldwide. It primarily afflicts the elderly with median age at diagnosis of 68 for men and 70 for women in the U.S. The annual incidence of newly diagnosed MM patients is approximately 45 thousand in the seven major global markets (U.S., France, Germany, Italy, Spain, the U.K. and Japan) with approximately 20 thousand in the U.S. Survival has increased in recent years to approximately five years for patients able to undergo stem cell transplant in combination with high-dose targeted drug therapy. There were approximately 65 thousand patients with MM in the U.S. in 2008.

Market growth of therapies that treat MM is expected to be strong, with sales across the seven major pharmaceutical markets forecasted to grow annually by 6.6% from $3.2 billion in 2010 to $5.8 billion in 2019. This growth will be driven by three factors:

1. Increased efficacy of current treatments, notably the leading targeted therapies (the proteasome inhibitor Velcade, and the IMiDs Revlimid and Thalomid, leading to longer life expectancy and allowing for more drug therapy to be administered over the disease course;

2. Increased use of existing and new drug combinations, particularly combinations with Velcade and Revlimid, leading to higher overall regimen costs; and

3. Introduction and uptake of new, higher cost therapies, particularly greater uptake of Revlimid and anticipated launch of premium priced next generation proteasome inhibitors and IMiDs such as carfilzomib and pomalidomide.

Despite progress in treating MM, current treatments do not cure the disease and are accompanied by high toxicity. Patients who have become refractory to both IMiD and proteasome inhibitor therapy have a particularly poor outcome, with a median overall survival of six to nine months. Therefore, opportunities remain for drug therapies with novel mechanisms of action and/or drugs that can treat refractory patients and can act synergistically with existing leading therapies.

ARRY-520, which targets the mitotic kinesin motor protein KSP, has a distinct mechanism of action from current standard of care drugs: it has been found active in preclinical disease models where standard of care drugs have not been effective. In clinical trials, ARRY-520 has shown signs of clinical activity in heavily pre-treated patients as a single agent; it is one of the very few non-IMiD or proteasome inhibitor drugs to show single agent activity in this patient population. ARRY-520 has also shown activity in patients who have been previously treated with Revlimid and Velcade which supports the potential for further development of ARRY-520 in patients refractory to other therapies. Based on this activity, we believe ARRY-520 has potential as a single agent for treating patients with MM who are refractory to both Revlimid and Velcade, and in combination with standard of care therapies in relapsed and refractory MM.

Myelodysplastic Syndromes (ARRY-614 p38/Tie-2 inhibitor)

Formerly known as "pre-leukemia", the myelodysplastic syndromes (MDS) are a spectrum of diseases in which the bone marrow does not make enough normal blood cells. Patients with MDS develop severe anemia, and platelet and neutrophil cytopenias, due to bone marrow failure. As MDS progresses, patients require frequent blood and platelet transfusions, and are prone to severe and fatal infections. Approximately 30% of MDS patients progress to Acute Myeloid Leukemia (AML) which accounted for approximately 9,000 deaths in 2010 in the U.S. MDS primarily afflicts the elderly, with 86% aged 60 years or above.

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According to an article published in the Journal of Clinical Oncology, June 2010, there were 45,000 new cases of Myelodysplastic Syndromes during 2003 in the U.S. This is four to five times greater than official estimates of MDS incidence based on the National Cancer Institute Surveillance, Epidemiology and End Results Program. The analysis also concluded that MDS patients have debilitating comorbidities, with significantly greater frequency than the overall population, such as cardiac complications (73%), dyspnea (49%), diabetes (40%) and severe infections (22%). Further, over a three-year period, 40% of MDS patients died compared with 15% for the overall population of the same age. These findings on the significance of comorbidities have been demonstrated in other studies. Notably, in a recent subpopulation study of "low" grade MDS patients at M.D. Anderson Cancer Center, infections were the most common cause of disease related death (38%), with hemorrhage (13%) also significant. These findings underscore the importance of addressing aspects of the disease such as neutrophil and platelet deficiencies and may support earlier therapeutic interventions.

MDS is forecast to grow rapidly by over 12% annually from 2010 to 2017; total sales of existing therapies are projected to increase from $653 million in 2010 to $1.5 billion in 2017 across the seven major pharmaceutical markets. This forecast does not include additional potential growth resulting from any novel, emerging therapies. Current therapies on the market include Vidaza® (azacitidine), Revlimid and Dacogen® (decitabine). Vidaza and Revlimid will have captured nearly 80% of the market by end of 2011, although a complete response following treatment with these agents is rare. We expect the recent approvals of these agents for MDS to also drive an increase in the overall drug-treated population, because access to these agents will encourage treatment and because there are no other therapeutic drug options currently available.

A number of other therapies which target the underlying biology of the disease are being investigated in MDS. These include p38, MAPK p38 and Tie-2. p38 is well-known for its role in the regulation of cytokine and chemokine signaling and production. There is a growing understanding of the role of p38 in the modulation of apoptosis and survival, particularly in the presence of DNA damage. Tie-2 signaling may promote stromal cell senescence and production of myelosuppressive cytokines leading to inappropriate apoptosis. We believe ARRY-614, a dual p38/Tie-2 inhibitor, may be effective in the treatment of MDS, particularly "low-to-intermediate" grade, by providing clinical benefit through hematological improvement (i.e. an increase in red blood cells, neutrophil cells and platelets), thereby reducing the need for red blood cell and platelet transfusions.

Lung Cancer (MEK162 and selumetinib MEK inhibitors)

Lung cancer is by far the leading cause of cancer-related mortality in the U.S. Lung cancer forms in the tissues of the lung, usually in the cells lining air passages. The two main types are non-small cell lung cancer, or NSCLC, which represents about 80% of lung cancer, and small cell lung cancer, which represents about 17%. In 2011, the estimated new cases and deaths from all lung cancer in the U.S. were 221,130 and 156,940, respectively. The overall five-year relative survival rate for the period of 2001 to 2007 for patients with lung cancer was 15.6%. The five-year relative survival rate varies markedly depending on the stage at diagnosis, from 52% to 24% to 4% for patients with local, regional and distant stage disease, respectively.

Patients with resectable disease may be cured by surgery or surgery with adjuvant chemotherapy. Local control can be achieved with radiation therapy in a large number of patients with unresectable disease, but a cure is seen only in a small number of patients. Patients with locally advanced, unresectable disease may have long-term survival with radiation therapy combined with chemotherapy. Patients with advanced metastatic disease may achieve improved survival and palliation of symptoms with chemotherapy, however metastatic NSCLC remains a fatal disease.

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Market growth of NSCLC drug therapies is expected to be moderate, with sales across the seven major pharmaceutical markets forecasted to grow annually by 5.5% from $4.0 billion in 2010 to $6.5 billion in 2019. This growth will be driven largely by the increased uptake of Alimta and the anticipated introduction of several novel agents.

The need for more effective and less toxic therapies as alternatives to or in combination with chemotherapy has led to the investigation of targeted therapies. In NSCLC, major components of cell signaling pathways such as the Ras-Raf-MEK-MAPK pathway and components of the normal cell cycle are frequently altered, with KRAS mutations in 15% to 20% NSCLC. These provide the rationale for the evaluation of therapies that target these aberrant pathways including MEK inhibitors.

Melanoma (MEK162 and selumetinib MEK inhibitors)

The number of new malignant melanoma cases is increasing at a rate greater than any other human cancer. According to the American Cancer Society, the estimated new cases and deaths from melanoma in the U.S. in 2011 are 70,230 and 8,790, respectively. Melanoma is a malignant tumor of cells that make the pigment melanin and are derived from the neural crest. Although most melanomas arise in the skin, they may also arise from mucosal surfaces or at other sites to which neural crest cells migrate. Melanoma occurs predominantly in adults and more than 50% of the cases arise in apparently normal areas of the skin. Melanoma in women occurs more commonly on the extremities and in men on the trunk or head and neck, but it can arise from any site on the skin surface. Early signs in a nevus or mole that would suggest malignant change into melanoma include darker or variable discoloration, itching, an increase in size, or the development of satellite moles. Ulceration or bleeding are typically later signs.

The optimal treatment for melanoma varies with the stage of the disease. In patients with early disease, surgical excision is the treatment of choice with some of these patients receiving adjuvant therapy with interferon alfa (IFNa). Surgical excision of limited distant metastatic disease can occasionally produce durable benefit, but most patients with distant metastases require systemic therapy. Systemic therapies include chemotherapy and immunotherapy, used either alone or in combination.

Several novel targeted therapies are under study including several that target the Ras-Raf-MEK-MAPK pathway and specific molecular abnormalities such as BRAF mutation, with BRAF mutations in 40% to 45% of melanoma. The BRAF inhibitor, vemurafenib has shown promising late stage trial results in metastatic melanoma. As MEK inhibitors target the Ras-Raf-MEK-MAPK pathway which is activated with BRAF mutation, they may also have the potential for activity in BRAF mutant melanoma.

Pancreatic Cancer (MEK162 MEK inhibitor)

Pancreatic cancer is one of the most lethal forms of cancer. In 2011, there are an estimated 44,030 new cases and 37,660 disease related deaths in the U.S., making it the fourth highest cause of cancer-related death. The failure of numerous agents in this disease over the past decade has only augmented the already considerable unmet need.

Pancreatic cancer is a disease of the elderly, with approximately 67% of patients 65 years or older at diagnosis. However, given the disease's rapid progression and dismal prognosis, the 30% of pancreatic cancer patients between 45 and 64 years old has received considerable attention in the public consciousness. Most patients present with metastatic disease and have a median survival of only four to six months a timeframe that has remained largely unchanged over the last few decades. Underpinning this lack of progress is a poor understanding of the underlying mechanisms of the disease. While some risk factors such as smoking and diabetes have been associated with a heightened risk of the disease,

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the most readily identified factor, genetics, accounts for less than 10% of the cases. Further, no diagnostic or treatment response biomarkers have been validated for pancreatic cancer.

Treatment options for pancreatic cancer are limited and have remained largely the same over the past two decades. Surgery is the only potentially curative treatment in early stage patients, with 50-80% of resected patients relapsing following surgery. However, given the vast majority of patients present with late stage disease, palliative chemotherapy, with the leading standard of care, Gemzar, is the main treatment for most patients.

Market growth of pancreatic cancer drug therapies is expected to be modest, with sales across the seven major pharmaceutical markets forecasted to grow annually by 3.4% from $612 million in 2010 to $829 million in 2019. This growth, largely driven by the entry of the only significant new drug expected to be approved, Abraxane® (albumin-bound nanoparticle paclitaxel) and rapid uptake in the multi-agent cytotoxic regimen of FOLIRINOX, will be offset by Gemzar suffering continued price erosion from increased generic competition.

Several novel targeted therapies are under study, including several that target the Ras-Raf-MEK-MAPK pathway. KRAS gene mutations occur in 70-90% of pancreatic cancer patients, and thus, are thought to play a critical role in the progression and maintenance of the disease. As MEK inhibitors target the Ras-Raf-MEK-MAPK pathway, which is activated with KRAS mutation, they may also have the potential for activity in KRAS mutated pancreatic cancer.

Colorectal Cancer (Selumetinib MEK inhibitor)

Colon and rectal cancers, collectively referred to as colorectal cancer, is one of the most common forms of cancer. According to the American Cancer Society, in the U.S. alone in 2011, there are an estimated 141,210 new cases and 49,380 deaths, making this the second highest cause of cancer-related death in the U.S. From 2010 to 2019, the number of newly diagnosed cases of colon cancer is projected to increase by over 16% across the seven major pharmaceutical markets. Also during this period, there is projected to be a 19% growth in the number of all those living with a history of colon cancer. The risk of colorectal cancer increases exponentially with age. For this reason, the aging population in the U.S., Western Europe and Japan may result in an increase in the incidence of colorectal cancer.

Treatment of colorectal cancer is closely linked to disease stage. Treatment modalities include surgery, radiotherapy and chemotherapy. Surgical resection of the primary tumor and regional lymph nodes is the only curative treatment and may cure up to 50% of patients. Pharmaceutical therapies play an important adjunctive and palliative role in most cases of stage III and IV colorectal cancer by helping reduce the incidence of recurrence, prolonging survival and improving quality of life. Three biological agents have been approved for metastatic colorectal cancer Avastin, Erbitux® (cetuximab) and Vectibix®(panitumumab). Use of these biologics is confined to the metastatic setting, although they are being tested in the adjuvant setting.

Market growth of colorectal cancer drug therapies is expected to be nearly flat, with sales across the seven major pharmaceutical markets forecasted to grow annually by less than 1% from $6.3 billion in 2010 to $6.7 billion in 2019. Despite the anticipated introduction of several novel therapies, the entry of generic/bio-similar competition for key agents such as Avastin and Erbitux is expected to result in considerable price erosion of these agents.

The roles of epidermal growth factor receptor, or EGFR, and vascular endothelial growth factor receptor, or VEGF, are well established and the focus of many current pharmaceutical therapies in development for the treatment of colorectal cancer. Treatment of colorectal cancer is becoming more individualized,

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however, following recent data showing that patients with mutated KRAS genes do not respond to anti-EGFR therapy. Consequently, pharmaceutical and biotechnology companies have begun to develop therapies that are based on the testing of the presence of biomarkers, such as KRAS and, to a lesser extent, BRAF mutations, to estimate the efficacy of drugs. Testing for biomarkers is a new paradigm in the treatment of colorectal cancer and we expect biomarkers for drug efficacy to play an ever-increasing role in colorectal cancer treatment. KRAS and BRAF mutations are thought to play a critical role in colorectal cancer progression and maintenance, with approximately 40% of colorectal cancer patients exhibiting KRAS mutations, due to activation of the Ras-Raf-MEK-MAPK pathway. We believe that a therapeutic approach to block this pathway with a MEK inhibitor may provide an effective therapy in patients with colorectal cancers that have these mutations.

Breast Cancer (ARRY-380 HER2 inhibitor)

Breast cancer is the second most common cancer type in the U.S. with estimates of 232,620 new cases and 39,970 deaths in 2011. Approximately 24% of all breast cancer patients are HER2 positive. Herceptin is an intravenously-dosed monoclonal antibody currently on the market for the treatment of breast cancers that over express HER2 and is approved as adjuvant therapy for HER2 positive breast cancer and all lines of HER2 positive metastatic breast cancer.

Market growth of breast cancer drug therapies is expected to be low, with sales across the seven major pharmaceutical markets forecasted to grow annually by 1% from $9.5 billion in 2010 to $10.4 billion in 2019. Market growth will be hindered by generic erosion of several key hormonal drugs, but will be offset by growth from anticipated entry of new non-hormonal therapies such as the second generation Herceptin, trastuzumab-DM1.

Tykerb, a small molecule drug that modulates HER2 and EGFR, was approved in March 2007 for the treatment of patients with metastatic HER2 positive breast cancer whose tumors have failed to respond to Herceptin and chemotherapy in second and third-line treatment. Tykerb in combination with Xeloda is currently being used in approximately 15% of the HER2 positive subpopulation. Tykerb's sales during 2010 were $351 million, with 2011 worldwide sales projected at $460 million.

We believe the broad use of Herceptin in HER2 positive settings and the increasing usage of Tykerb/Xeloda combinations in metastatic HER2 positive settings suggest a high potential value for an orally active drug that regulates HER2 and can be conveniently dosed for extended periods of time. ARRY-380 is an oral, reversible and selective HER2 inhibitor currently in a Phase 1 study expansion study to evaluate the safety, pharmacokinetics and pharmacodynamics in patients with HER2 positive breast cancer. We believe ARRY-380 has the potential to treat this patient population. ARRY-380 is selective for HER2 and does not appear to have the EGFR-related side effects seen with Tykerb.

Pain and Inflammatory Diseases Market

Pain and inflammation are closely interrelated, yet present distinct challenges and opportunities. Pain remains one of the most pressing as well as largest therapeutic areas to address, including a wide spectrum of acute, subacute and chronic pain conditions ranging from acute postoperative pain to chronic osteoarthritis pain. Although well established, the pain field continues to evolve and specialize, as the etiology of pain is recognized as being increasingly complex. A plethora of medications, procedures and devices marketed to address different forms of pain exist, but pain remains an area of significant unmet need. In recent years, with the exception of the introduction of antidepressant drugs such as Cymbalta® (duloxetine) and the antiepileptics Neurontin® (gabapentin) and Lyrica® (pregabalin), drug development in pain has been rather limited. Instead, drug development has focused largely around reformulations and

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alternate delivery mechanisms to provide improved safety/tolerability/drug abuse prevention among the leading existing classes of opioids and nonsteroidal anti-inflammatory drugs, or NSAIDs.

Inflammation is a natural biologic response to injury or infection that, under normal conditions, resolves during healing or clearing. Unregulated inflammation results in a broad range of conditions, most of which are classified by the tissue or organ where the inflammation occurs. These conditions include: respiratory diseases such as asthma, allergic rhinitis, and chronic obstructive pulmonary disease, or COPD; dermatological conditions such as psoriasis and atopic dermatitis; gastrointestinal disorders such as Crohn's disease and ulcerative colitis; musculoskeletal disorders such as rheumatoid arthritis, systemic lupus erythematosus, or SLE, gout, and spondyloarthropathies such as psoriatic arthritis and ankylosing spondylitis. Similar to the pain market, there are a wide range of drug treatment options and delivery mechanisms depending on the specific condition. Yet even in acknowledged "crowded" disease areas such as asthma, there still remains significant unmet need in specific populations such as those with severe, refractory and difficult-to-control asthma.

Pain (ARRY-797 p38 inhibitor)

Patients are treated for almost 320 million cases per year of acute and subacute pain in the U.S. alone. Acute and subacute pain occurs under a broad set of circumstances including bone fractures, postoperative pain in planned surgical or trauma/emergency settings, severe migraine attacks, arthritis flares, and breakthrough cancer pain. For example, surgical patients typically experience moderate to severe pain up to a few weeks after the procedure.

Chronic pain presents perhaps an even more significant burden, with over half of all adults experiencing chronic pain in their lifetime. According to a recent report to the U.S. Department of Health and Human Services by the Institute of Medicine, chronic pain affects an estimated 116 million adults in the U.S. and costs the nation up to $635 billion per year in medical treatment and lost productivity. Chronic pain, variously defined as a pain condition which persists or recurs for a duration of greater than three or greater than six months depending on the specific condition, includes a wide range of conditions including arthritic pain, inflammatory pain, chronic low back pain, fibromyalgia, neuropathic pain (e.g., postherpetic neuralgia, painful diabetic neuropathy), chronic headache, and cancer pain.

The major analgesic pain therapies currently on the market, including opioids, NSAIDs and selective COX-2 inhibitors, have side effect and efficacy issues. Opioids are the most commonly prescribed drug class in the U.S., with 15% to 20% of doctor visits involving an opioid prescription, and four million Americans per year prescribed a long-acting opioid. Opioids are efficacious in the management of pain, but have considerable side effects including nausea, vomiting, constipation, respiratory depression and cognitive dysfunction. Perhaps even more significant, opioid drug abuse is a major concern; second only to car crashes as a cause of accidental death in the U.S., opioid abuse accounted for 12,000 fatalities in 2007. NSAIDs have demonstrated pain reduction which is modest but less efficacious than opioids. Although NSAIDs have overall a more favorable safety profile than opioids, renal toxicity and gastrointestinal bleeding are associated with their use. COX-2 inhibitors though possibly offering less gastrointestinal toxicity than NSAIDs, still result in other side effects associated with NSAIDs, most notably adverse cardiovascular effects. Most drugs in this class have been withdrawn from the market, with the notable exception of Celebrex® (celecoxib).

We believe there is an opportunity for a novel drug with comparable or better efficacy than NSAIDs and COX-2 inhibitors, as well as a desire for an improved safety profile compared to NSAIDs, COX-2 inhibitors or opioids. Further, there is an opportunity in several inflammatory pain conditions affecting large populations, such as RA and ankylosing spondylitis, to offer additional pain relief over and above what current targeted therapeutics, such as tumor necrosis factor alpha, or TNFα, inhibitors, may provide.

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Market growth of drug therapies used in acute, subacute and chronic pain settings is expected to be moderate. Sales across the seven major pharmaceutical markets for acute and subacute pain are forecasted to grow annually by 3% from $17.4 billion in 2010 to $22.1 billion in 2018. Sales for chronic pain are forecasted to grow annually by 4% from $21.1 billion in 2010 to $29.0 billion in 2018.

Few innovative pain therapeutics have successfully emerged from clinical development in recent years and there remains a significant need for safer and more efficacious drugs for the treatment of acute, subacute and chronic pain. p38 is well-known for its role in the regulation of the production of proinflammatory cytokines such as TNF and IL-1, as well as PGE2, a significant pain modifier. Based on preclinical and clinical data, we believe ARRY-797, a p38 inhibitor, has good potential to treat a broad array of pain conditions.

Asthma (ARRY-502 CRTh2 antagonist)

Asthma, a chronic condition of the airways, poses one of the more significant public health burdens today. According to the American Lung Association, in 2008 an estimated 23 million individuals have asthma, resulting in approximately 4,000 deaths per year and nearly $21 billion in medical treatment and lost productivity.

Asthma is a heterogeneous disease, caused by a combination of environmental and genetic factors, which can wax and wane, with varying frequency and severity among individual patients. Most asthma patients suffer from allergic asthma, whereby the patient's immune system produces an exaggerated response to allergens (e.g. pollens, pets, dust). Mast cells, activated by IgE, release histamine and various other mediators that cause immediate bronchospasm, or a constriction of the muscles of the airway walls, and vasoconstriction, or narrowing of bronchial blood vessels, thereby resulting in coughing and difficulty breathing. Mast cells also promote tissue damage in the airways through release of other mediators, notably cytokines, chemokines, and prostanoids such as the mediator prostaglandin D2, or PGD2, which results in the attraction of more inflammatory cells to the lungs. In severe allergic asthma, there is emerging evidence suggesting that a greater presence of PGD2 and an upregulation of CRTh2, a protein receptor that is expressed on T-helper 2, or Th2, lymphocytes, eosinophils and basophils, may play a particularly important role in greater symptoms and impairment of lung function.

Currently, for chronic maintenance treatment of asthma and other respiratory tract diseases, there are a wide range of treatment options with a variety of delivery mechanisms, each of which has drawbacks. Most notably, safety concerns have arisen with inhaled long-acting beta-2 agonists, or LABAs, and the FDA has required producers of LABAs to to describe these concerns on their labels and to conduct further large-scale randomized trials. These concerns impact the leading class of LABA/ICS, or inhaled corticosteroid, combination therapies as well. The most common rescue medications are short-acting beta-2 agonists.

Market growth of asthma drug therapies is expected to be flat-to-slightly-declining, with sales across the seven major pharmaceutical markets forecasted to shrink annually by less than 1% from $13.4 billion in 2010 to $12.7 billion in 2019. This outlook is based on the lack of any new blockbuster drug class entering the market and a potential softening of the use of LABAs in the U.S. in the short term, and ongoing price erosion due to generic entry and competitive pricing in current therapies. This will be offset somewhat by an expected uptake of once-daily LABA/ICS combination agents, once approved, and likely increased insurance coverage and expenditures due to healthcare policy changes in the U.S. from 2014 onward.

There are a number of drug classes being explored for the treatment of asthma. Array is developing a novel oral drug, ARRY-502, which is an antagonist of the CRTh2 receptor. Upregulation of CRTh2 has also been shown to result in chemotaxis and activation of eosinophils, basophils and Th2 lymphocytes, all

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key mediators of asthma and particularly severe asthma. Therefore, ARRY-502 has the potential to be an effective treatment for patients with asthma, particularly those with severe disease, who currently have few, if any, options for effective treatment.

Research and Development for Proprietary Drug Discovery

Our primary research efforts are centered on the treatment of cancer and inflammatory disease. Our research focuses on biologic functions, or pathways, that have been identified as important in the treatment of human disease based on human clinical, genetic or preclinical data. Within these pathways, we seek to create first-in-class drugs regulating important therapeutic targets to treat patients with serious or life-threatening conditions, primarily in cancer, inflammatory disease. In addition, we seek to identify opportunities to improve upon existing therapies or drugs in clinical development by creating clinical candidates with superior, or best-in-class, drug characteristics, including efficacy, tolerability or dosing to provide safer, more effective drugs. During fiscal years 2011, 2010 and 2009, we spent $63.5 million, $72.5 million and $89.6 million, respectively, on research and development for proprietary drug discovery, which consist of costs associated with our proprietary drug programs for, among other things, salaries and benefits for scientific personnel, consulting and outsourced services, laboratory supplies, allocated facilities costs and depreciation.

Drug Discovery and Development Timeline

The drug development process is highly uncertain, is subject to a number of risks that are beyond our control and takes many years to complete. The following table outlines each phase in the drug development process. Completion times are difficult to estimate and can vary greatly based on the drug and indication. Therefore, the duration times shown in the table below are estimates only.

Phase	Objective	Estimated Duration
Discovery	Lead identification and target validation	2 to 4 years
Preclinical	Initial toxicology for preliminary identification of risks for humans; gather early pharmacokinetic data	1 to 2 years
Phase 1	Evaluate the safety and tolerability of the drug in human subjects and find the maximum tolerated dose. The pharmacokinetics of the drug are examined after single and multiple doses, the effects of food on the pharmacokinetics may be evaluated and drug metabolites may be monitored.	1 to 2 years
Phase 2	Establish effectiveness of the drug and its optimal dosage; continue safety evaluation	2 to 4 years
Phase 3	Confirm efficacy, dosage regime and safety profile of the drug; submit New Drug Application	2 to 4 years
FDA Approval	Approval by the FDA to sell and market the drug under approved labeling	6 months to 2 years

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Animal and other non-clinical studies are often conducted during each phase of human clinical studies. Proof-of concept for a drug candidate generally occurs during Phase 2, after initial safety and efficacy data are established.

Our Research and Development Technologies and Expertise

We are continuing to improve our comprehensive research and development capabilities, consisting of four integrated areas of expertise:

Discovery Research Biology, Chemistry and Translational Medicine

Process Research, Development, Formulation and Manufacturing

Clinical Development Clinical Science, Clinical Operations, Translational Medicine, Biostatistics & Data Management, Regulatory Affairs and Program Management

Information Technology

Discovery Research

We have a broad drug discovery platform with all the necessary capabilities to efficiently invent new chemical compounds. We continue to add to our breadth of knowledge, refine our processes and hire key scientists who enhance our current capabilities. We have expanded our translational medicine team, which designs and runs mechanistic studies in cell biology and pharmacology to provide insight into clinical development strategy, product differentiation and biomarker support for clinical development. To date, our average cost to invent a new chemical entity and file an IND application is $15 million, compared to estimates of up to $100 million spent by major pharmaceutical companies. Our discovery group has created high quality clinical candidates with every wholly-owned and to our knowledge, every partnered, drug to reach the clinic to date having been shown to modulate its mechanistic target, as measured by an appropriate clinical biomarker.

Process Research, Development, Formulation and Manufacturing

We have built and we continue to enhance our process research and development and cGMP manufacturing capabilities to accommodate the productivity of our research platform and support our clinical development plans. Our capabilities include formulations, physical form characterization and aspects of clinical supply manufacturing.

Clinical Development

Our current key capabilities within clinical development include clinical science, clinical operations, clinical pharmacology, safety monitoring, biostatistics, programming and data management, regulatory strategy and program management. This group leads the development and implementation of our clinical and regulatory strategies. The clinical group designs, directs and implements all clinical operations, including identifying and selecting clinical investigators, recruiting study subjects to participate in our clinical trials, biostatistics, data management, drug safety evaluation and adverse event reporting. The clinical group also is responsible for ensuring that our development programs are conducted in compliance with applicable regulatory requirements. The group also works closely with the cross functional project and clinical teams to facilitate the appropriate and efficient development of our diverse product pipeline.

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Our near term focus is on bringing our most promising drugs through proof-of-concept clinical trials. Our proof-of-concept strategy is to efficiently conduct studies to demonstrate the value of each program in a therapeutic area so that decisions to continue, modify or cease development of a program can be made early in the development process. We believe that our broad development pipeline and productive discovery platform provide an incentive to design trials for each program with high hurdles to demonstrate the potential of the drug or to "fail early."

Information Technology

We believe that our information technology, or IT, capabilities provide a competitive advantage in each aspect of our business. Our IT capabilities are essential to increasing our productivity through capturing, organizing and providing appropriate information to improve decision-making. Several years ago, we accomplished our goal of creating a paperless discovery research environment, which has empowered our scientists to improve real time decision-making at the bench. Array has recently completed a clinical information system that parallels the comprehensive capabilities of our discovery system, providing company-wide access to real-time information for each clinical trial as well as the entire drug portfolio. In addition to real-time study data, the system's information includes planned and actual screening/enrollment at the site level, budget and actual costs by types of activities, important events and milestones. We believe Array now has one of the most advanced clinical IT systems in the entire drug industry.

Competitors

The pharmaceutical and biotechnology industries are characterized by rapid and continuous technological innovation. We compete with companies worldwide that are engaged in the research and discovery, licensing, development and commercialization of drug candidates, including large pharmaceutical companies with internal discovery and development functions, biotech companies with competing products in the therapeutic areas we are targeting and contract research organizations that perform many of the functions we perform under our collaborations. In addition, we face competition from other pharmaceutical and biotechnology companies seeking to out-license drugs targeting the same disease class or condition as our drug candidates are based on, among other things, patent position, product efficacy, safety, reliability, availability, patient convenience, price and reimbursement potential. Therefore, we may be unable to enter into collaboration, partnering, or out-licensing agreements on terms that are acceptable to us, or at all. We also compete with other clinical trials for patients who are eligible to be enrolled in clinical trials we or our collaborators are conducting, which may limit the number of patients who meet the criteria for enrollment and delay or prevent us or our collaborators from completing trials when anticipated. Because the timing of entry of a drug in the market presents important competitive advantages, the speed with which we are able to complete drug development and clinical trials, obtain regulatory approval and supply commercial quantities of drugs to the market will affect our competitive position. Some of our competitors have a broader range of capabilities and have greater access to financial, technical, scientific, regulatory, business development, recruiting and other resources than we do. Their access to greater resources may allow them to develop processes or products that are more effective, safer or less costly, or gain greater market acceptance, than products we develop or for which they obtain FDA approval more rapidly than we do. We anticipate that we will face increased competition in the future as new companies enter the market and advanced technologies become available.

Government Regulation

Biopharmaceutical companies are subject to substantial regulation by governmental agencies in the U.S. and other countries. Virtually all pharmaceutical products are subject to extensive pre- and post-market regulation by FDA, including regulations that govern the testing, manufacturing, distribution, safety, efficacy, approval, labeling, storage, record keeping, reporting, advertising and promotion of such

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products under the Federal Food, Drug, and Cosmetic Act and its implementing regulations, and by comparable agencies and laws in foreign countries. Prescription drug products are subject to rigorous preclinical and clinical testing and other approval procedures by the FDA and by foreign regulatory agencies. The FDA must approve any new drug, including a new dosage form or new use of a previously approved drug, prior to marketing in the United States. All applications for FDA approval must contain, among other things, information relating to safety and efficacy, pharmaceutical formulation, stability, manufacturing, processing, packaging, labeling and quality control information. The preclinical and clinical testing and approval process requires substantial time, effort and financial resources, and we cannot be certain that the FDA will grant approval for any of our product candidates on a timely basis, if at all. Before an application requesting FDA approval for a new drug product is submitted to the FDA, three phases of human clinical trials are usually conducted to test the safety and effectiveness of the product. Phase 1 clinical trials most typically involve testing the drug on a small number of healthy volunteers to assess the safety profile of the drug at different dosage levels. Phase 2 clinical trials, which may also enroll a relatively small number of patient volunteers, are designed to further evaluate the drug's safety profile and to provide preliminary data as to the drug's effectiveness in humans. Phase 3 clinical trials consist of larger, well-controlled studies that may involve several hundred or even several thousand patient volunteers representing the drug's targeted population. In addition, biopharmaceutical companies may elect to conduct, or be required by the FDA to conduct, Phase 4 clinical trials to further assess the drug's safety or effectiveness after approval. Such post approval trials are typically referred to as Phase 4 clinical trials. During any of these phases, the clinical trial can be placed on clinical hold, or temporarily or permanently stopped for a variety of reasons, principally for safety concerns. In addition, the failure to comply with applicable regulatory requirements in the U.S., including Good Clinical Practices, or GCP, and in other countries in which we conduct development activities could result in failure to obtain approval, as well as a variety of fines and sanctions, such as warning letters, product recalls, product seizures, suspension of operations, fines and civil penalties or criminal prosecution.

Biopharmaceutical companies must submit the results of product development, preclinical studies and clinical trials to the FDA as part of a new drug application, or NDA. NDAs must also contain extensive information relating to the product's pharmacology, chemistry, manufacture, controls and proposed labeling, among other things. The approval process is time-consuming and expensive and there are no assurances that approval will be granted on a timely basis, or at all. Even if regulatory approvals are granted, a marketed product is subject to comprehensive requirements under federal, state and foreign laws and regulations. Post-marketing requirements include reporting adverse events, recordkeeping and compliance with cGMP and marketing requirements. Adverse events reported after marketing of a drug can result in additional restrictions being placed on the use of a drug and, possibly, in withdrawal of the drug from the market. The FDA or similar agencies in other countries may also require labeling changes to products at any time based on new safety information.

If drug candidates we develop are approved for commercial marketing under a New Drug Application, or NDA, by the FDA, they would be subject to the provisions of the Drug Price Competition and Patent Term Restoration Act of 1984, known as the "Hatch-Waxman Act." The Hatch-Waxman Act provides companies with marketing exclusivity for new chemical entities and allows companies to apply to extend patent protection for up to five additional years. It also provides a means for approving generic versions of a drug product once the marketing exclusivity period has ended and all relevant patents have expired (or have been successfully challenged and defeated). The period of exclusive marketing may be shortened, however, by a successful patent challenge. The laws of other key markets likewise create both opportunities for exclusivity periods and patent protections and the possibility of generic competition once such periods or protections either have reached expiry or have been successfully challenged by generic entrants.

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All facilities and manufacturing processes used in the production of Active Pharmaceutical Ingredients for clinical use in the U.S. must be operated in conformity with cGMP as established by the FDA. Our production takes place at a manufacturing facility that complies with cGMP, which allows us to produce cGMP compliant compounds. In our facility, we have the capacity to produce Active Pharmaceutical Ingredients for early clinical testing. We have validated this capability for compliance with FDA regulations and began our first cGMP manufacturing campaign in 2002. Our cGMP facility is subject to periodic regulatory inspections to ensure compliance with cGMP requirements. We could also be required to comply with specific requirements or specifications of other countries or of our collaborators, which may be more stringent than regulatory requirements and which can delay timely progress in our clinical development programs. If we fail to comply with applicable regulations, the FDA could require us to cease ongoing research or disqualify the data submitted to regulatory authorities. Other countries have similar regulatory powers. A finding that we had materially violated cGMP requirements could result in additional regulatory sanctions and, in severe cases, could result in a mandated closing of our cGMP facility, which would materially and adversely affect our business, financial condition and results of operations.

In the course of our business, we handle, store and dispose of chemicals and biological samples. We are subject to various federal, state and local laws and regulations relating to the use, manufacture, storage, handling and disposal of hazardous materials and waste products. These environmental laws generally impose liability regardless of the negligence or fault of a party and may expose us to liability for the conduct of, or conditions caused by, others.

Most health care providers, including research institutions from whom we or our collaborators obtain patient information, are subject to privacy rules under the Health Insurance Portability and Accountability Act of 1996, or HIPAA and the recent amendments to HIPAA. Additionally, strict personal privacy laws in other countries affect pharmaceutical companies' activities in other countries. Such laws include the EU Directive 95/46-EC on the protection of individuals with regard to the processing of personal data as well as individual EU Member States, implementing laws and additional laws. Although our clinical development efforts are not barred by these privacy regulations, we could face substantial criminal penalties if we knowingly receive individually identifiable health information from a health care provider that has not satisfied HIPAA's or the EU's disclosure standards. Failure by EU clinical trial partners to obey requirements of national laws on private personal data, including laws implementing the EU Data Protection Directive, might result in liability and/or adverse publicity. In addition, certain privacy laws and genetic testing laws may apply directly to our operations and/or those of our collaborators and may impose restrictions on the use and dissemination of individuals' health information.

Our clinical development activities involve the production and use of intermediate and bulk active pharmaceutical ingredients, or API. We frequently contract with third-party manufacturers to produce larger quantities of API for us. Some of these manufacturers are located outside the U.S. and may obtain ingredients from suppliers in other foreign countries before shipping the bulk API to Array in the U.S. Cross-border shipments of pharmaceutical ingredients and products are subject to regulation in the U.S. by the FDA and in foreign jurisdictions, including, in the EU, under laws adopted by the EU Member States implementing the Community Code on Medicinal Products Directive 2001/83, as amended. These regulations generally impose various requirements on us and/or our third-party manufacturers. In some cases, for example in the EU, there are cGMP requirements that exceed the requirements of the FDA. In other cases, we must provide confirmation that we are registered with the FDA and have either a Notice of a Claimed Exception for an IND application, an approved New Drug Application or an approved Biologics License Application. Third party manufacturers may lack capacity to meet our needs go out of business or fail to perform. In addition, supplies of raw materials needed for manufacturing or formulation of clinical supplies may not be available or in shorty supply.

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We are subject to other regulations, including regulations under the Occupational Safety and Health Act, regulations promulgated by the U.S. Department of Agriculture, or USDA, and regulations under other federal, state and local laws. Violations of any of these requirements could result in penalties being assessed against us.

Intellectual Property

Our success depends in part on our ability to protect our potential drug candidates, other intellectual property rights and our proprietary software technologies. To establish and protect our proprietary technologies and products, we rely on a combination of patent, copyright, trademark and trade secret laws, as well as confidentiality provisions in our contracts with collaborators.

We attempt to protect our trade secrets by entering into confidentiality agreements with our employees, third parties and consultants. Our employees also sign agreements requiring that they assign to us their interests in inventions, original expressions and any corresponding patents and copyrights arising from their work for us. However, it is possible that these agreements may be breached, invalidated or rendered unenforceable and if so, we may not have an adequate remedy available. Despite the measures we have taken to protect our intellectual property, parties to our agreements may breach the confidentiality provisions or infringe or misappropriate our patents, copyrights, trademarks, trade secrets and other proprietary rights. In addition, third parties may independently discover or invent competing technologies or reverse-engineer our trade secrets or other technology. The failure of our employees, our consultants or third parties to maintain secrecy of our drug discovery and development efforts may compromise or prevent our ability to obtain patent coverage for our invention.

Our patent strategy is designed to protect inventions, technology and improvements to inventions that are commercially important to our business. We have numerous U.S. patents and patent applications on file with the U.S. Patent and Trademark Office and around the world. The source code for our proprietary software programs is protected both as a trade secret and as a copyrighted work.

U.S. patents issued from applications filed on or after June 8, 1995, have a term of 20 years from the application filing date or earlier claimed priority. All of our patent applications were filed after June 8, 1995. Patents in most other countries have a term of 20 years from the date of filing of the patent application. Because the time from filing patent applications to issuance of patents is often several years, this process may result in a period of patent protection significantly shorter than 20 years, which may adversely affect our ability to exclude competitors from our markets. Currently, none of our patents covering drugs currently under development will expire prior to 2023. Our success will depend in part upon our ability to develop proprietary products and technologies and to obtain patent coverage for these products and technologies. We intend to continue to file patent applications covering newly developed products and technologies. We may not, however, commercialize the technology underlying any or all of our existing or future patent applications.

Patents provide some degree of protection for our proprietary technology. However, the pursuit and assertion of patent rights, particularly in areas like pharmaceuticals and biotechnology, involve complex legal and factual determinations and, therefore, are characterized by some uncertainty. In addition, the laws governing patentability and the scope of patent coverage continue to evolve, particularly in biotechnology. As a result, patents may not be issued from any of our patent applications or from applications licensed to us. The scope of any of our patents, if issued, may not be sufficiently broad to offer meaningful protection. In addition, our patents or patents licensed to us, if they are issued, may be successfully challenged, invalidated, circumvented or rendered unenforceable so that our patent rights might not create an effective competitive barrier. Moreover, the laws of some foreign countries may not protect our proprietary rights to the same extent as do the laws of the U.S. Any patents issued to us or our

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strategic partners may not provide a legal basis for establishing an exclusive market for our products or provide us with any competitive advantages. Moreover, the patents held by others may adversely affect our ability to do business or to continue to use our technologies freely. In view of these factors, our intellectual property positions bear some degree of uncertainty.

Employees

As of June 30, 2011, we had 259 full-time employees, including 152 scientists and 44 clinical and regulatory employees, of whom 80 have PhDs or MDs. None of our employees are covered by collective bargaining agreements and we consider our employee relations to be good.

Our Corporate Information

Our principal executive offices are located at 3200 Walnut Street, Boulder, Colorado 80301 and our phone number is (303) 381-6600. We were founded in 1998 and became a public company in November 2000. Our stock is listed on the NASDAQ Global Market under the symbol "ARRY."

Available Information

Electronic copies of our Annual Reports on Form 10-K, Quarterly Reports on Form 10-Q, Current Reports on Form 8-K and other documents we file with or furnish to the SEC are available free of charge (i) on the "Investor Relations" section of our website at http://www.arraybiopharma.com or (ii) by sending a written request to Investor Relations at our corporate headquarters. Information on our website is not incorporated by reference into this report.

Additionally, the documents we file or furnish with the SEC are available free of charge at the SEC's Public Reference Room at 100 F Street, NE, Washington D.C. 20549, or can be accessed free of charge on the website maintained by the SEC at http://www.sec.gov. Other information on the operation of the Public Reference Room is available by calling the SEC at (800) SEC-0330.

ITEM 1A. RISK FACTORS

In addition to the other factors discussed elsewhere in this report and in other reports we file with the SEC, the following factors could cause our actual results or events to differ materially from those contained in any forward-looking statements made by us or on our behalf. In addition, other risks and uncertainties not presently known to us or that we currently deem immaterial may impair our business operations. If any of the following risks or such other risks occur, it could adversely affect our business, operating results and financial condition, as well as cause the value of our common stock to decline.

Risks Related to Our Business

If we need but are unable to obtain additional funding to support our operations, we could be required to reduce our research and development activities or curtail our operations and it may lead to uncertainty about our ability to continue to operate as a going concern.

We have expended substantial funds to discover and develop our drug candidates and additional substantial funds will be required for further development, including preclinical testing and clinical trials of any product candidates we develop internally. Additional funds will be required to manufacture and market any products we own or retain rights to that are approved for commercial sale. Because the successful development of our products is uncertain, we are unable to precisely estimate the actual funds we will require to develop and potentially commercialize them.

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We have historically funded our operations through revenue from our collaborations and out-license transactions, the issuance of equity securities and debt financing. We currently believe that our existing cash resources will enable us to continue to fund our current operations for at least the next 12 months. However, we will continue to be dependent upon such sources for the foreseeable future. Our ability to obtain additional funding when needed, changes to our operating plans, our existing and anticipated working capital needs, the acceleration or modification of our planned research and development activities or expenditures, increased expenses or other events may affect our need for additional capital in the future and may require us to seek additional funding sooner than anticipated. Additional funding may include milestone payments under existing collaborations, up-front fees or research funding through new out-licensing transactions, sales of debt or equity securities and/or securing additional credit facilities.

If we are unable to generate enough revenue or secure additional sources of funding and/or reduce our current rate of research and development spending or further reduce our expenses, we may be required to curtail operations significantly, which could prevent us from successfully executing our operating plan and could raise substantial doubt as to our ability to continue as a going concern in future periods. Even if we are able to secure the additional sources of funding, it may not be on terms that are favorable or satisfactory to us and may result in significant dilution to our stockholders. These events may result in an inability to maintain a level of liquidity necessary to continue operating our business and the loss of all or part of the investment of our stockholders in our common stock. In addition, if we are unable to maintain certain levels of cash and marketable securities, our obligations under our credit facilities with Deerfield Private Design Fund, L.P. and Deerfield Private Design International Fund, L.P. (who we refer to collectively as Deerfield) and our loan agreement with Comerica Bank may be accelerated.

We have a history of operating losses and may not achieve or sustain profitability.

We have incurred significant operating and net losses and negative cash flows from operations since our inception. As of June 30, 2011, we had an accumulated deficit of $547.2 million. We had net losses of $56.3 million, $77.6 million and $127.8 million, for the fiscal years ended June 30, 2011, 2010 and 2009, respectively. We expect to incur additional losses and negative cash flows in the future, and these losses may continue or increase in part due to anticipated levels of expenses for research and development, particularly clinical development and expansion of our clinical and scientific capabilities to support ongoing development of our programs. As a result, we may not be able to achieve or maintain profitability.

We may not receive royalty or milestone revenue under our collaboration agreements for several years, or at all.

Much of our current revenue is non-recurring in nature and unpredictable as to timing and amount. Several of our out-license and collaboration agreements provide for royalties on product sales. However, because none of our drug candidates have been approved for commercial sale, our drug candidates are at early stages of development and drug development entails a high risk of failure, we do not expect to receive any royalty revenue for several years, if at all. For the same reasons, we may never realize much of the milestone revenue provided for in our out-license and collaboration agreements. Similarly, drugs we select to commercialize ourselves or partner for later-stage co-development and commercialization may not generate revenue for several years, or at all.

We may not be successful in entering into additional out-license agreements on favorable terms, which may adversely affect our liquidity or require us to change our spending priorities on our proprietary programs.

We are committing significant resources to create our own proprietary drug candidates and to build a commercial-stage biopharmaceutical company. We have built our clinical and discovery programs

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through spending $464.1 million from our inception through June 30, 2011. In fiscal 2011, we spent $63.5 million in research and development for proprietary programs, compared to $72.5 million and $89.6 million for fiscal years 2010 and 2009, respectively. Our proprietary drug discovery programs are in their early stage of development and are unproven. Our ability to continue to fund our planned spending on our proprietary drug programs and in building our commercial capabilities depends to a large degree on up-front fees, milestone payments and other revenue we receive as a result of our partnered programs. To date, we have entered into eight out-licensing agreements for the development and commercialization of our drug candidates, and we plan to continue initiatives during fiscal 2012 to partner select clinical candidates to obtain additional capital.

We may not be successful, however, in entering into additional out-licensing agreements with favorable terms, including up-front, milestone, royalty and/or license payments and the retention of certain valuable commercialization or co-promote rights, as a result of factors, many of which are outside of our control. These factors include:

- our ability to create valuable proprietary drugs targeting large market opportunities;
- research and spending priorities of potential licensing partners;
- willingness of and the resources available to pharmaceutical and biotechnology companies to in-license drug candidates to fill their clinical pipelines;
- the success or failure, and timing, of pre-clinical and clinical trials for our proprietary programs we intend to out-license; or
- our ability or inability to generate proof-of-concept data and to agree with a potential partner on the value of proprietary drug candidates we are seeking to out-license, or on the related terms.

If we are unable to enter into out-licensing agreements and realize milestone, license and/or up-front fees when anticipated, it may adversely affect our liquidity and we may be forced to curtail or delay development of all or some of our proprietary programs, which in turn may harm our business and the value of our stock. In addition, insufficient funds may require us to relinquish greater rights to product candidates at an earlier stage of development or on less favorable terms to us or our stockholders than we would otherwise choose to obtain funding for our operations.

We may not out-license our proprietary programs at the most appropriate time to maximize the total value or return of these programs to us.

A critical aspect of our business strategy is to out-license drug candidates for further development, co-development and/or commercialization to obtain the highest possible value while also evaluating earlier out-licensing opportunities to maximize our risk-adjusted return on our investment in proprietary research. Because the costs and risk of failure of bringing a drug to market are high, the value of out-licensing a drug candidate generally increases as it successfully progresses through clinical trials.

We may choose or be forced to out-license a drug candidate or program on terms that require us to relinquish commercial or market rights or at a point in the research and development process that does not provide as great a value or return than what might have been obtained if we had further developed the candidate or program internally. Likewise, we may decline, or be unable to obtain favorable, early out-licensing opportunities in programs that do not result in a commercially viable drug, which could leave the resulting program with little or no value even though significant resources were invested in its development. Our inability to successfully out-license our programs on favorable terms could materially adversely affect our results of operations and cash flows.

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Our drug candidates are at early stages of development and we may not successfully develop a drug candidate that becomes a commercially viable drug.

The drug discovery and development process is highly uncertain and we have not developed, and may never develop, a drug candidate that ultimately leads to a commercially viable drug. All of our most advanced drug candidates are in the early stages of development, in either Phase 1 or Phase 2, and we do not have any drugs approved for commercial sale. Before a drug product is approved by the FDA, for commercial marketing, it is tested for safety and effectiveness in clinical trials that can take up to six years or longer. Promising results in preclinical development or early clinical trials may not be predictive of results obtained in later clinical trials. A number of pharmaceutical companies have experienced significant setbacks in advanced clinical trials, even after obtaining promising results in earlier preclinical and clinical trials. At any time, we, the FDA or an Institutional Review Board may place a clinical trial on clinical hold, or temporarily or permanently stop the trial, for a variety of reasons, principally for safety concerns. We or our collaborators may experience numerous unforeseen events during, or as a result of, the clinical development process that could delay or prevent our drug candidates from being approved, including:

- failure to achieve clinical trial results that indicate a candidate is effective in treating a specified condition or illness in humans;
- presence of harmful side effects;
- determination by the FDA that the submitted data do not satisfy the criteria for approval;
- lack of commercial viability of the drug;
- failure to acquire, on reasonable terms, intellectual property rights necessary for commercialization; and
- existence of therapeutics that are more effective.

We or our collaborators may choose not to commercialize a drug candidate at any time during development, which would reduce or eliminate our potential return on investment for that drug.

At any time, we or our collaborators may decide to discontinue the development of a drug candidate or not to commercialize a candidate. If we terminate a program in which we have invested significant resources, we will not receive any return on our investment and we will have missed the opportunity to have allocated those resources to potentially more productive uses. Even if one of our drug candidates receives regulatory approval for marketing, physicians or consumers may not find that its effectiveness, ease of use, side effect profile, cost or other factors make it effective in treating disease or more beneficial than or preferable to other drugs on the market. Additionally, third-party payors, such as government health plans and health insurance plans or maintenance organizations, may choose not to include our drugs on their formulary lists for reimbursement. As a result, our drugs may not be used or may be used only for restricted applications.

Our capital requirements could significantly increase if we choose to develop more of our proprietary programs internally.

We believe that the maximum value for certain proprietary drug candidates is best achieved by retaining the rights to develop and commercialize the candidat