IMMUNOGEN INC
Form 424B5
July 12, 2012

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Registration No. 333-174335

The information in this preliminary prospectus supplement is not complete and may be changed. A registration statement relating to these securities has been declared effective by the Securities and Exchange Commission. This preliminary prospectus supplement and the accompanying prospectus are not an offer to sell these securities and we are not soliciting an offer to buy these securities in any state or other jurisdiction where the offer or sale is not permitted.

SUBJECT TO COMPLETION, DATED JULY 11, 2012

PROSPECTUS SUPPLEMENT
(to Prospectus dated May 19, 2011)

Shares

Common Stock

We are offering shares of our common stock. Our common stock is listed on The NASDAQ Global Select Market under the symbol "IMGN." On July 10, 2012, the last reported sale price of our common stock on The NASDAQ Global Select Market was $17.37 per share.

Investing in our common stock involves a high degree of risk. Please read "Risk Factors" beginning on page S-17 of this prospectus supplement and in the documents incorporated by reference into this prospectus supplement.

Neither the Securities and Exchange Commission nor any state securities commission has approved or disapproved of these securities or determined if this prospectus is truthful or complete. Any representation to the contrary is a criminal offense.

	PER SHARE	TOTAL
Public Offering Price	$	$
Underwriting Discounts and Commissions	$	$
Proceeds to ImmunoGen, before expenses	$	$

Delivery of shares of common stock is expected to be made on or about July , 2012. We have granted the underwriters an option for a period of 30 days to purchase up to an additional shares of our common stock.

Joint Book-Running Managers

Morgan Stanley **Jefferies**

Prospectus Supplement dated July , 2012

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You should rely only on the information contained or incorporated by reference in this prospectus supplement, the accompanying prospectus or any free writing prospectus that we have authorized for use in connection with this offering. Neither we nor the underwriters have authorized anyone to provide you with information different from that contained in this prospectus or any accompanying free writing prospectus. We are offering to sell, and seeking offers to buy, common stock only in jurisdictions where offers and sales are permitted. If anyone provides you with different or inconsistent information, you should not rely on it. We and the underwriters take no responsibility for, and can provide no assurance as to the reliability of, any other information that others may give you. The information contained in this prospectus supplement, the accompanying prospectus and any accompanying free writing prospectus is accurate only as of the date of this prospectus supplement, the accompanying prospectus and any such accompanying free writing prospectus, regardless of the time of delivery of this prospectus supplement, the accompanying prospectus, any such accompanying free writing prospectus or of any sale of our common stock. Our business, financial condition, results

of operations and prospects may have changed since those dates. You should read this prospectus supplement, the accompanying prospectus, the documents incorporated by reference in this prospectus supplement and the accompanying prospectus, and any free writing prospectus that we have authorized for use in connection with this offering, in their entirety before making an investment decision. You should also read and consider the information in the documents to which we have referred you in the sections of this prospectus supplement entitled "Where You Can Find More Information" and "Incorporation of Documents by Reference."

No action is being taken in any jurisdiction outside the United States to permit a public offering of the common stock or possession or distribution of this prospectus supplement, the accompanying prospectus or any free writing prospectus in that jurisdiction. Persons who come into possession of this prospectus supplement, the accompanying prospectus or any free writing prospectus in jurisdictions outside the United States are required to inform themselves about and to observe any restrictions as to this offering and the distribution of this prospectus supplement, the accompanying prospectus and any accompanying free writing prospectus applicable to that jurisdiction.

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About this Prospectus Supplement

On May 19, 2011, we filed with the Securities and Exchange Commission, or SEC, a registration statement on Form S-3 (File No. 333-174335) utilizing a shelf registration process relating to the securities described in this prospectus supplement, which registration statement was automatically effective upon filing. Under this shelf registration process, we may, from time to time, sell common stock and other securities, of which this offering is a part.

This document is in two parts. The first part is this prospectus supplement, which describes the specific terms of this common stock offering and also adds to and updates information contained in the accompanying prospectus and the documents incorporated by reference into the prospectus. The second part, the accompanying prospectus, gives more general information, some of which does not apply to this offering. Generally, when we refer to this prospectus, we are referring to both parts of this document combined.

If the description of the offering varies between this prospectus supplement and the accompanying prospectus, you should rely on the information contained in this prospectus supplement. However, if any statement in one of these documents is inconsistent with a statement in another document having a later date for example, a document incorporated by reference in this prospectus supplement or the accompanying prospectus the statement in the document having the later date modifies or supersedes the earlier statement.

We further note that the representations, warranties and covenants made by us in any agreement that is filed as an exhibit to any document that is incorporated by reference in the prospectus supplement or the accompanying prospectus were made solely for the benefit of the parties to such agreement, including, in some cases, for the purpose of allocating risk among the parties to such agreements, and should not be deemed to be a representation, warranty or covenant to you. Moreover, such representations, warranties or covenants were accurate only as of the date when made. Accordingly, such representations, warranties and covenants should not be relied on as accurately representing the current state of our affairs.

Unless we have indicated otherwise, or the context otherwise requires, references in this prospectus supplement and the accompanying prospectus to "ImmunoGen," "the Company," "we," "us" and "our" or similar terms are to ImmunoGen, Inc. and its subsidiaries.

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Prospectus Supplement Summary

This summary highlights information contained elsewhere or incorporated by reference in this prospectus supplement and the accompanying prospectus. This summary does not contain all of the information that you should consider before deciding to invest in our common stock. You should read this entire prospectus supplement and the accompanying prospectus carefully, including the "Risk Factors" section contained in this prospectus supplement, our consolidated financial statements and the related notes thereto and the other documents and information incorporated by reference in this prospectus supplement and the accompanying prospectus and the information included in any free writing prospectus that we have authorized for use in connection with this offering.

Company Overview

We develop novel, targeted antibody-based therapeutics for the treatment of cancer using our expertise in cancer biology, monoclonal antibodies, highly potent cytotoxic, or cell-killing, agents, and the design of linkers that enable these agents to remain stably attached to the antibodies while in the blood stream and be released in their fully active form after delivery to a cancer cell. An anticancer compound made using our Targeted Antibody Payload, or TAP, technology consists of a monoclonal antibody that binds specifically to an antigen target found on cancer cells with multiple copies of one of our proprietary cell-killing agents attached using one of our engineered linkers. Its antibody component enables a TAP compound to bind specifically to cancer cells that express its target antigen, the highly potent cytotoxic agent serves to kill the cancer cell and the engineered linker controls the release and activation of the cytotoxic agent inside the cancer cell. With some TAP compounds, the antibody component also has anticancer activity of its own. Our TAP technology is designed to enable the creation of highly effective, well-tolerated anticancer product candidates.

Our TAP technology is providing us with an advancing and expanding product pipeline. There are now ten TAP compounds, three of which are wholly owned by us, in clinical trials from our own programs and from our partnerships with other companies. We also have one non-conjugated, or "naked," antibody in development by one of our partners. We expect to continue to make significant investments in research and development to further advance and expand our product pipeline. As a result, we expect our operating expenses to increase significantly during our fiscal year ending June 30, 2013.

Our current collaborative partners are: Amgen Inc., Bayer HealthCare (a subgroup of Bayer AG), Biotest AG, Genentech, Inc. (a member of the Roche Group), sometimes referred to in this prospectus supplement as Roche, Eli Lilly and Company, or Lilly, Novartis Institutes for BioMedical Research, Inc., or Novartis, and Sanofi.

Our Product Pipeline

There are eleven compounds in clinical trials through our own programs and our collaborations with other companies. In addition to the partner compound already in Phase III clinical testing, we expect up to three additional partner compounds to advance into pivotal clinical testing by the end of 2013. We also expect to submit an Investigational New Drug application, or IND, for our fourth wholly

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owned compound in mid-2013. The following table lists the current stage of development of these compounds:

	Current Stage
Lead Compound in Development through a Collaborative Partner	
Trastuzumab emtansine (T-DM1)	Phase III
Compounds in Development by ImmunoGen	
IMGN901 (lorvotuzumab mertansine)	Phase II
IMGN853	Phase I
IMGN529	Phase I
"IMGN Next"	Preclinical
Other Compounds in Development through Collaborative Partners	
SAR3419	Phase II
BT-062	Phase I
SAR650984*	Phase I
SAR566658	Phase I
BAY 94-9343	Phase I
First Amgen TAP compound ("Amgen 1")	Phase I
Second Amgen TAP compound ("Amgen 2")	Phase I

* Non-conjugated or "naked" antibody therapeutic.

Trastuzumab Emtansine Most Advanced Compound with Our TAP Technology

Trastuzumab emtansine, often referred to as T-DM1, is the most advanced compound in development using our TAP technology. Roche expects to apply for marketing approval of T-DM1 in 2012 in the United States and Europe based on the findings from its EMILIA Phase III clinical trial. Based on this timing, T-DM1 could potentially be approved in the United States by mid-2013, which would potentially enable us to begin receiving royalties from this compound in our fiscal year ending June 30, 2014.

T-DM1 consists of trastuzumab, which is the active component of Genentech's antibody therapeutic, Herceptin® (trastuzumab), with one of our cell-killing agents, DM1, attached using our non-cleavable SMCC linker. T-DM1 is in global development by Genentech's parent company, Roche, for the treatment of HER2-positive breast cancer under a license with us to use our maytansinoid TAP technology with antibodies binding to HER2. We believe Roche also intends to develop T-DM1 for non-breast cancer uses such as HER2-positive gastric cancer. Roche markets Herceptin, which had global sales of approximately 5.3 billion Swiss francs, or approximately US$5.6 billion, in 2011 based on public reports from Roche and current exchange rates.

Based on the clinical results received to date, we believe that T-DM1 has the potential to be a valuable new therapeutic for the treatment of patients with HER2-positive cancer.

Development for HER2-positive Metastatic Breast Cancer

Roche is evaluating T-DM1 for HER2-positive metastatic breast cancer in three Phase III clinical trials: EMILIA, MARIANNE and TH3RESA.

EMILIA This lead Phase III clinical trial is a randomized 991-patient study comparing T-DM1, used alone, to Tykerb® (lapatinib) used with Xeloda® (capecitabine) to treat patients with HER2-positive metastatic breast cancer who have previously been treated with Herceptin and with a

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taxane. Findings from EMILIA were reported on June 3, 2012 at the American Society of Clinical Oncology, or ASCO, annual meeting. Among the findings reported were:

Treatment with T-DM1 significantly improved progression-free survival, or PFS, compared to treatment with Tykerb plus Xeloda. Median PFS was 9.6 months with T-DM1 compared to 6.4 months with Tykerb plus Xeloda and the hazard ratio was 0.65 ($p<0.0001$).

As expected, overall survival, or OS, data were not mature at the time of this analysis. The statistical plan had called for an interim analysis of OS to be conducted when PFS was mature and for a final analysis of OS to be conducted after approximately 632 deaths had occurred.

While the OS data were not mature, a sufficient number of deaths had occurred in the Tykerb plus Xeloda group to establish median OS for that treatment group as 23.3 months. Longer follow-up is required to determine the median OS for the T-DM1 group. The interim differences between the T-DM1 and the Tykerb plus Xeloda treatment groups had a hazard ratio of 0.621 ($p=0.0005$). This was not statistically significant based on criteria related to the pre-defined "stopping boundary" used to analyze the data available at the analysis point.

The estimated 1-year and 2-year survival rates based on OS data at the time of the data analysis are shown in the figure below.

The EMILIA data reported that the patients randomized to treatment with T-DM1 had a median dose intensity of 99.9%, while the patients randomized to treatment with Tykerb plus Xeloda had a median dose intensity of 93.4% for Tykerb and 77.2% for Xeloda. Dose intensity is a measure of the amount of a therapeutic that a patient received relative to the amount of the therapeutic the patient was scheduled to receive across the course of their treatment period. Dose reduction below the scheduled amount typically occurs because a patient is unable to tolerate the full dose of the therapeutic.

The EMILIA data reported included that fewer T-DM1-treated patients experienced Grade 3 or higher adverse events, which are severe adverse events, than the patients treated with Tykerb plus Xeloda: 40.8% versus 57%, respectively. The most frequently reported Grade 3 and higher

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adverse events for Tykerb plus Xeloda and for T-DM1 are shown in the tables below (the bolded information represents higher frequency of the reported event).

	Tyberb plus Xeloda		T-DM1	
Non-Hematologic	**Any Grade**	**Grade ≥ 3**	**Any Grade**	**Grade ≥ 3**
Most frequently reported with Tykerb plus Xeloda				
Diarrhea	79.7%	**20.7%**	23.3%	1.6%
Hand-foot syndrome	58.0	**16.4**	1.2	0.0
Vomiting	29.3	**4.5**	19.0	0.8
Hypokalemia (low potassium)	8.6	**4.1**	8.6	2.2
Most frequently reported with T-DM1				
Increased AST (type of liver enzyme)	9.4	0.8	22.4	**4.3**
Increased ALT (type of liver enzyme)	8.8	1.4	16.9	**2.9**

	Tykerb plus Xeloda			T-DM1		
Hematologic	**Any Grade**	**Gr 3**	**Gr 4**	**Any Grade**	**Gr 3**	**Gr 4**
Most frequently reported with Tykerb plus Xeloda						
Neutropenia (low white blood cells)	8.6%	**3.5%**	**0.8%**	5.9%	1.6%	0.4%
Febrile neutropenia	1.0	**0.4**	**0.6**	0.0	0.0	0.0
Most frequently reported with T-DM1						
Anemia (low red blood cells)	8.0	1.6	0.0	10.4	**2.7**	**0.0**
Thrombocytopenia (low platelets)	2.5	0.0	0.2	28.0	**10.4**	**2.4**

Roche has noted that it plans to use the data from EMILIA to apply in 2012 for marketing approval of T-DM1 in the United States and Europe. This submission timing would enable us to begin receiving royalties on T-DM1 sales in our fiscal year ending June 30, 2014, assuming the marketing application is successfully accepted and approved. Marketing approval, if granted, is expected to be for use of T-DM1 to treat the cancer treated in the trial, which was HER2-positive metastatic breast cancer in patients that had previously been treated with Herceptin and with a taxane in any setting. Separately, Chugai, another member of the Roche Group, has noted that it expects to apply in 2013 for marketing approval of T-DM1 in Japan.

MARIANNE This Phase III clinical trial assesses both T-DM1 used alone and T-DM1 used together with pertuzumab for first-line treatment of HER2-positive metastatic breast cancer and compares these two treatment arms to Herceptin used together with a taxane, which is standard first-line treatment for this cancer. Roche has noted that it intends to apply in 2014 for marketing approval both for T-DM1 used alone and for T-DM1 used with pertuzumab as first-line treatments for this cancer using data from MARIANNE, assuming the findings are favorable. Roche has indicated that patient enrollment in MARIANNE has been completed. The primary endpoint of MARIANNE is PFS. We expect data from this clinical trial to be reported in 2013.

In a separate 137-patient Phase II clinical trial comparing T-DM1 used alone to Herceptin used with a taxane for first-line treatment of HER2-positive metastatic breast cancer, T-DM1 demonstrated significant improvement in PFS as compared to the Herceptin plus a taxane treatment arm and the hazard ratio was 0.59 (p=0.0353).

	T-DM1	**Herceptin plus taxane**	**Hazard ratio**
Progression-free survival (median)	14.2 months	9.2 months	0.59 (p=0.0353)

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Also, fewer Grade 3 or greater adverse events were reported by the patients treated with T-DM1 than those treated with Herceptin plus the taxane, and far fewer T-DM1-treated patients reported hair loss.

	T-DM1	Herceptin plus taxane
Incidence of Grade ≥ 3 adverse events	46%	89%
Incidence of alopecia (hair loss)	4%	67%

TH3RESA This Phase III clinical trial evaluates T-DM1 as a treatment for HER2-positive metastatic breast cancer that was previously treated with Herceptin and Tykerb. This clinical trial started in 2011 and compares T-DM1 to the physician's choice of treatment, since there are no standard treatments for this cancer.

Development for Early Stage HER2-positive Breast Cancer

On June 3, 2012, Roche presented its three-pronged approach to developing T-DM1 for the treatment of early stage HER2-positive breast cancer: development for neoadjuvant use, for adjuvant use, and for patients with residual invasive disease following surgery. Roche has announced that it plans to conduct clinical trials that may be used to support registration of T-DM1 in each of these settings.

Neoadjuvant Roche's strategy in the neoadjuvant setting, or treatments used in conjunction with surgery, is designed to leverage use of pathological complete response, or pCR, as a surrogate endpoint. In its neo-adjuvant trial, Roche expects to compare treatment with T-DM1 (with and without pertuzumab) to treatment with Herceptin (with and without pertuzumab). Patients will be randomized to one of four treatment groups: (1) Herceptin plus docetaxel plus carboplatin pre-surgery and Herceptin alone post-surgery; (2) Herceptin plus pertuzumab plus docetaxel plus carboplatin pre-surgery and Herceptin plus pertuzumab post-surgery; (3) T-DM1 plus docetaxel pre-surgery and T-DM1 alone post-surgery; and (4) T-DM1 plus pertuzumab plus docetaxel pre-surgery and T-DM1 plus pertuzumab alone post-surgery. Roche expects patient dosing in this clinical trial to begin in the first quarter of 2013 and pCR data to be available in 2015.

Adjuvant In Roche's clinical trial in the adjuvant setting, Roche expects to evaluate T-DM1 used with pertuzumab as a primary treatment for patients with early stage HER2-positive breast cancer compared to Herceptin used with pertuzumab. Consistent with an approved use of Herceptin in the adjuvant setting, the treatments will be used in conjunction with an anthracycline-based regimen. Also consistent with the approved adjuvant use of Herceptin, patients must have HER2-positive breast cancer that has spread into lymph nodes and/or is estrogen receptor-/progesterone receptor-negative. The primary endpoint of the clinical trial is disease-free survival. Roche expects this clinical trial to begin in 2013 and to report data from the trial in 2018.

Residual invasive disease This clinical trial is expected to evaluate T-DM1 compared to Herceptin as a treatment for patients with HER2-positive breast cancer with residual invasive disease following surgery, which Roche believes is a high unmet medical need. The primary endpoint of this clinical trial is 3-year disease-free survival. Roche expects this clinical trial to begin in the first quarter of 2013 and to report data from the trial in 2018.

IMGN901 Our Lead Wholly Owned Compound

Lorvotuzumab mertansine, or IMGN901, is wholly owned by us. We are evaluating this TAP compound in Phase II testing for the first-line treatment of small-cell lung cancer, or SCLC. We also are completing a Phase I clinical trial assessing IMGN901 for the treatment of multiple myeloma, or MM.

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We developed IMGN901 to bind to and kill cancer cells that express CD56, its target. The antibody component of IMGN901 serves to target our attached cytotoxic agent specifically to CD56-expressing cancer cells and has not been shown to have meaningful anticancer activity of its own. We believe IMGN901 is an example of how our TAP technology can be used to create targeted, antibody-based therapies for more types of cancers than have been able to be treated with non-conjugated or "naked" antibodies.

A number of different types of cancers express CD56, including small-cell carcinomas, such as SCLC and Merkel cell carcinoma, or MCC, many cases of OC, carcinoid tumors and other solid tumors of neuroendocrine origin. CD56-expressing cancers also include MM, NK lymphomas and select other liquid tumors.

Based on our studies, we believe that CD56 is expressed on approximately 89% of SCLC cases and approximately 76% of MM cases. Based on American Cancer Society estimates, we believe that approximately 29,400 new cases of SCLC and 21,700 new cases of MM will be diagnosed in the United States in 2012. IMGN901 has received orphan drug designations for SCLC, MCC and for MM in the United States and Europe.

Evaluation for SCLC

IMGN901 is in Phase II clinical testing for the first-line treatment of SCLC. Assuming this clinical trial is successful, we intend to advance IMGN901 into pivotal clinical testing for this indication.

Rationale We chose to focus the development of IMGN901 on first-line treatment of SCLC for several reasons. SCLC almost universally expresses CD56 and is a prevalent and highly aggressive cancer with limited treatment options. Median survival for patients diagnosed with SCLC extensive disease is less than one year. This is in part because current first-line standard-of-care for such SCLC achieves a median PFS of only five to six months and patients typically decline rapidly once they relapse. We believe that use of IMGN901 as a first-line therapy provides access to the largest SCLC market and the best opportunity for IMGN901 to make a meaningful difference for patients with this cancer.

Evidence of activity was seen with IMGN901 in early clinical trials in which it was assessed as a single agent in patients with previously treated SCLC. IMGN901 has also shown evidence of activity against MCC. In an early stage clinical trial that included 21 evaluable patients with MCC, three patients had complete responses following treatment with IMGN901, another patient had an unconfirmed partial response and four other patients had clinically relevant stable disease.

Assessment of IMGN901 for first-line treatment of SCLC extensive disease requires that it can safely be used in combination with first-line standard-of-care for this cancer, since such therapy needs to be provided to patients newly diagnosed with this cancer. The tolerability profile of IMGN901 when used as a single agent supported that it should be able to be safely assessed in combination with etoposide/carboplatin, a current first-line standard-of-care for SCLC. We have completed dose assessment of IMGN901 used in combination with etoposide/carboplatin and expect to present the clinical findings at a medical conference in September 2012.

We also believe IMGN901 in combination with etoposide/carboplatin may provide improved efficacy because IMGN901 works by a different mechanism of action than etoposide/carboplatin and, in preclinical studies, IMGN901 has shown substantially increased activity used in combination with other active agents.

NORTH clinical trial We began patient recruitment in our NORTH Phase II clinical trial in March 2012. This clinical trial assesses IMGN901 as first-line treatment for SCLC extensive disease at the dose established in the Phase I dose escalation stage. The NORTH clinical trial is designed to

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evaluate whether the addition of IMGN901 to etoposide/carboplatin can meaningfully improve duration of PFS over that achieved by etoposide/carboplatin alone.

All patients enrolled in NORTH are to receive a standard six cycles of treatment with etoposide/carboplatin. Two out of every three patients enrolled are randomized to receive IMGN901 in addition to the six cycles of etoposide/carboplatin. Once the etoposide/carboplatin cycles are completed, these patients can elect to also continue to receive IMGN901 as a single agent if they are benefiting from treatment.

Our NORTH clinical trial utilizes a Simon Two-Stage Design. This means that after the first 59 patients are enrolled (39 in the IMGN901 plus etoposide/carboplatin group and 20 in the etoposide/carboplatin alone group), these patients will be followed for an interim analysis, assessment of PFS at 6 months, while patient enrollment in the NORTH clinical trial continues. The interim analysis will examine whether the addition of IMGN901 to etoposide/carboplatin achieved a pre-defined level of improvement compared to etoposide/carboplatin used alone. This level of improvement threshold was developed based on historic data and will serve as a basis for us to move forward with certain IMGN901 pivotal development decisions, which we expect to be able to make in 2013.

The full NORTH Phase II clinical trial is designed to include 80 patients in the IMGN901 plus etoposide/carboplatin group and 40 patients in the etoposide/carboplatin alone group. Its primary endpoint is PFS and its secondary endpoints are PFS at 6 months, OS, OS at 12 months, time to progression, and objective response rate.

Evaluation for Multiple Myeloma

IMGN901 is being assessed in a Phase I clinical trial for the treatment of MM in combination with lenalidomide plus dexamethasone, a standard of care for this cancer. Promising data were presented at the ASCO annual meeting in June 2011 from the dose-finding portion of this clinical trial. We expect to report data from an expansion phase of this clinical trial at a medical meeting in late 2012. Based on the findings to date, we believe IMGN901 is a potential treatment for MM. However, because of the number of new therapies for MM and in order to focus our IMGN901 development program on SCLC, we currently have no plans to advance IMGN901 into pivotal testing for the treatment of MM.

IMGN853 Our Folate Receptor-Targeting TAP Compound

Our wholly owned IMGN853 TAP compound targets folate receptor 1, or FOLR1, which is over-expressed on many cases of ovarian cancer, or OC, and other carcinomas, including non-small cell lung cancer, or NSCLC. The expansion phase of our IMGN853 Phase I clinical trial will evaluate the compound specifically in patients with epithelial OC, the most prevalent type of OC, and in patients with adenocarcinoma NSCLC, the most prevalent type of lung cancer. Based on American Cancer Society estimates, we believe that approximately 19,000 new cases of epithelial OC and 90,000 new cases of adenocarcinoma NSCLC will be diagnosed in the United States in 2012.

IMGN853 consists of our FOLR1-targeting antibody with one of our potent cell-killing agents attached using one of our engineered linkers to counteract the multi-drug resistance that many cancers develop. The antibody component of IMGN853 binds to a different site than folate, enabling IMGN853 to avoid competing with dietary folate for binding sites on cancer cells.

The IMGN853 Phase I clinical trial is designed to define the path(s) to potential regulatory approval for IMGN853. The dose-escalation portion of the clinical trial, used to establish the maximum-tolerated dose, or MTD, of IMGN853, has features designed to expedite its completion. This phase of the clinical trial is open to patients with any previously treated epithelial malignancy that over-expresses FOLR1 and allows for single-patient cohorts at the initial, lower dose levels. Once the MTD is defined, IMGN853 will be evaluated in three disease-specific expansion cohorts: (1) patients

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with platinum-resistant/refractory epithelial OC; (2) patients with epithelial OC that is relapsed/refractory to conventional treatments; and (3) patients with adenocarcinoma NSCLC that is relapsed/refractory to conventional treatments. We expect to report the first clinical data with IMGN853 in 2013 and expect data from this Phase I clinical trial to enable us to make certain IMGN853 pivotal development decisions in 2013.

IMGN529 TAP Compound with Both Antibody and Cytotoxic Agent Anticancer Activity

Our wholly owned IMGN529 TAP compound is a potential therapy for CD37-expressing liquid tumors including non-Hodgkin's lymphoma, or NHL, and chronic lymphocytic lymphoma, or CLL. Its CD37 target has an expression profile similar to that of CD20, the target of Rituxan® (rituximab) on NHL subtypes.

IMGN529 has a unique profile for a therapy for NHL as it is an ADC, unlike the naked antibody Rituxan or radioactive therapies, and in preclinical testing, its antibody component alone has demonstrated pronounced anticancer activity, unlike other ADCs in development for NHL including SAR3419. In preclinical studies using CD37-expressing cancer cells, the antibody component of IMGN529 provided evidence of strong pro-apoptotic, or direct cell-killing activity, as well as antibody-dependent cellular cytotoxicity, or ADCC, and complement-dependent cytotoxicity, or CDC, activity. In preclinical studies, the antibody component of IMGN529 continued to demonstrate these anticancer properties after our potent cell-killing agent was attached to it, which provides it with an additional, and highly effective, method of killing cancer cells.

In April 2012, we initiated clinical testing of IMGN529. This Phase I, multi-center clinical trial is open to patients with relapsed or relapsed/refractory CD37-expressing NHL. Patients with any of the most common types of NHL are eligible for enrollment, including follicular lymphoma, diffuse large B-cell lymphoma, mantle cell lymphoma, and marginal zone lymphoma.

We expect to report the first clinical data with IMGN529 in 2013, and to be able to use data from this Phase I clinical trial to make certain IMGN529 development-related decisions in 2013.

"IMGN Next"

We are continuing to build our product pipeline and expect to advance our next wholly owned TAP compound to IND stage by mid-2013. We intend to present the first data on this compound, including the first preclinical findings, at a scientific conference in April 2013.

Other Product Candidates in Development through Our Collaborations

SAR3419 is in development by Sanofi. We created this TAP compound, including its antibody component, and licensed it to Sanofi as part of a broader collaboration. SAR3419 targets CD19 and is a potential new treatment for CD19-expressing B-cell malignancies including NHL and B-cell acute lymphoblastic leukemia, or B-ALL. Sanofi initiated Phase II clinical testing of SAR3419 in October 2011 and is evaluating it for both diffuse large B-cell lymphoma and for B-ALL. We believe initial data from one of the Phase II clinical trials underway with SAR3419 will be presented at a medical meeting in late 2012.

In its Phase I assessment, SAR3419 was found to demonstrate activity across an array of NHL histological subtypes and in patients with Rituxan-refractory and -responsive disease. Alternative dosing schedules were evaluated to establish the recommended Phase II schedule. At the recommended Phase II dose and schedule reported at the 2012 ASCO annual meeting, 29% (six out of 21) of patients had an objective response and another 43% (nine out of 21) had stable disease when treated with SAR3419.

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BT-062 was created by Biotest under a license agreement that grants Biotest the exclusive right to use our maytansinoid TAP technology with antibodies that target CD138, an antigen found on MM and certain other cancers. We have opt-in rights with respect to BT-062 in the United States. BT-062 is in clinical testing for the treatment of MM, and Biotest is now also considering evaluating it for the treatment of certain types of CD138-expressing solid tumors.

SAR650894 advanced into Phase I clinical testing in 2010 through our collaboration with Sanofi. It consists of a CD38-targeting antibody that has demonstrated anticancer activity in preclinical testing and does not have a separate cytotoxic agent attached. Sanofi is developing it for the treatment of certain hematological malignancies.

SAR566658 also advanced into clinical testing in 2010 through our collaboration with Sanofi. It targets CA6, which is found on breast, ovarian, cervical, lung and pancreatic tumors. SAR566658 is in Phase I clinical testing for the treatment of solid tumors that express this antigen.

The TAP compound, *BAY 94-9343*, advanced into clinical testing in 2011 through our collaboration with Bayer HealthCare. It targets mesothelin and is in Phase I clinical testing for the treatment of solid tumors that express this antigen. Also, two TAP compounds that we refer to as *Amgen 1* and *Amgen 2* advanced into clinical testing in early 2012 through our collaboration with Amgen.

We believe that as many as three of these partner compounds could advance into pivotal clinical testing by the end of 2013 based on communications with our collaborative partners.

In addition to Roche, Sanofi, Biotest, Bayer HealthCare and Amgen, we also have active partnerships with Novartis and Lilly. These collaborations were established in late 2010 and 2011, respectively, and consequently the product programs are at earlier stages of development than those of earlier collaborations.

Clinical Experience with T-DM1 and Other Compounds Utilizing Our TAP Technology

Clinical findings have been reported with a number of TAP compounds. While Phase II and Phase III clinical trial data has only been reported with T-DM1 to date, findings from Phase I clinical trials have been reported with other TAP compounds including IMGN901, SAR3419 and BT-062. The findings reported support the lack of a single, or "class", toxicity with use of our technology, such as cross-product reports of clinically significant neutropenia, peripheral neuropathy, and/or gastrointestinal toxicities. Rather, the toxicity reported has varied depending on the TAP compound and its target, as would be expected with the technology performing as intended, with a notable lack of clinically significant bone marrow suppression.

The tolerability profiles of TAP compounds support their assessment in combination with other anticancer agents, which can further enhance efficacy and can also enable evaluation of the compounds in earlier lines of therapy. One or more clinical trials have been initiated assessing T-DM1, IMGN901 and SAR3419 as part of combination regimens.

TAP compounds have been safely administered at dose levels at which a naked antibody can have anticancer activity. For example, in an early clinical trial, T-DM1 was safely dosed at 2.4 mg/kg per week, which is greater than the approved 2 mg/kg weekly dose of Herceptin, the same trastuzumab antibody used in T-DM1. This is believed to broaden the utility of our technology as it can be used with antibodies that have anticancer properties with the expectation that the MTD achieved with the resulting TAP compounds will be at a level at which the antibody component can contribute meaningful anticancer activity in addition to that provided by the attached cell-killing agent.

Two antibody-drug conjugates, or ADCs, have gained approval to treat hematological malignancies, although one (Mylotarg®) has since been withdrawn from the market. To date, T-DM1 is the only ADC to have demonstrated significant efficacy in a solid tumor indication in a controlled clinical trial. We believe the effectiveness of our TAP technology in a solid tumor indication is important as solid tumors

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are the most prevalent type of cancers. Of the 1.6 million cases of cancer projected to be diagnosed in the United States in 2012, 90% are for solid tumors.

Out-licenses and Collaborations

We selectively out-license restricted access to our TAP technology to other companies to provide us with cash to fund our own product programs and to expand the utilization of our technology. These agreements typically provide the licensee with rights to use our TAP technology with any of its antibodies to develop products to a defined target. The licensee is generally responsible for the development, clinical testing, manufacturing, registration and commercialization of any resulting product candidate. As part of these agreements, we are generally entitled to receive upfront fees, and also potential milestone payments, royalties on the commercial sales of any resulting products and research and development funding based on activities performed at our collaborative partner's request. We are also compensated for preclinical and clinical materials supplied to our partners.

We will not receive royalty payments from a TAP technology out-license until a product candidate developed under the license is approved for marketing and commercialized, nor do we expect to receive significant individual milestone payments under our existing collaborations prior to product approval. Achievement of product approval requires, at a minimum, favorable completion of preclinical development and evaluation, assessment of early-stage clinical trials, advancement into pivotal Phase II and/or Phase III clinical testing, completion of this later-stage clinical testing with favorable results, and completion of regulatory submissions and review. The only collaboration that may provide us with royalty revenue and significant milestone payments in the foreseeable future is our collaboration with Roche relating to T-DM1. Below is a table setting forth our active collaborations, the number of targets licensed and current status of the product candidates being developed thereunder: