

IMMUNOGEN INC
Form 8-K
May 07, 2010

UNITED STATES

SECURITIES AND EXCHANGE COMMISSION

Washington, D.C. 20549

FORM 8-K

CURRENT REPORT

**PURSUANT TO SECTION 13 OR 15(d)
OF THE SECURITIES EXCHANGE ACT OF 1934**

Date of Report (Date of earliest event reported): **May 6, 2010**

ImmunoGen, Inc.

(Exact name of registrant as specified in its charter)

Massachusetts
(State or other
jurisdiction of
incorporation)

0-17999
(Commission File
Number)

04-2726691
(IRS Employer
Identification No.)

830 Winter Street, Waltham, MA 02451

(Address of principal executive offices) (Zip Code)

Registrant's telephone number, including area code: **(781) 895-0600**

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Check the appropriate box below if the Form 8-K filing is intended to simultaneously satisfy the filing obligation of the registrant under any of the following provisions (*see* General Instruction A.2. below):

- Written communications pursuant to Rule 425 under the Securities Act (17 CFR 230.425)

 - Soliciting material pursuant to Rule 14a-12 under the Exchange Act (17 CFR 240.14a-12)

 - Pre-commencement communications pursuant to Rule 14d-2(b) under the Exchange Act (17 CFR 240.14d-2(b))

 - Pre-commencement communications pursuant to Rule 13e-4(c) under the Exchange Act (17 CFR 240.13e-4(c))
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ITEM 1.01. ENTRY INTO A MATERIAL DEFINITIVE AGREEMENT

On May 6, 2010, ImmunoGen, Inc. (the Company) entered into an underwriting agreement (the Underwriting Agreement) with J.P. Morgan Securities Inc., as representative of the several underwriters (the Underwriters) named in Schedule 1 of the Underwriting Agreement, related to a public offering of 9,000,000 shares of the Company s common stock, par value \$0.01 per share (the Common Stock), at a price of \$8.00 per share less the underwriting discount (the Offering). Under the terms of the Underwriting Agreement, the Company has granted the Underwriters an option, exercisable for 30 days, to purchase up to an additional 1,350,000 shares of Common Stock to cover over-allotments, if any, at the same price. The Offering is expected to close on May 12, 2010, subject to the satisfaction of customary closing conditions. The net proceeds to the Company are expected to be approximately \$67.4 million after deducting estimated expenses associated with the Offering.

The Offering is being made pursuant to a prospectus supplement dated May 6, 2010 and an accompanying prospectus dated April 22, 2010, pursuant to the Company s existing effective shelf registration statement on Form S-3 (File No. 333-165981), which was filed with the Securities and Exchange Commission (the Commission) on April 9, 2010 and declared effective by the Commission on April 22, 2010.

The Underwriting Agreement contains customary representations, warranties, and agreements by the Company, and customary conditions to closing, indemnification obligations of the Company and the Underwriter, including for liabilities under the Securities Act of 1933, as amended, other obligations of the parties, and termination provisions.

A copy of the opinion of Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C. relating to the legality of the issuance and sale of the shares in the Offering is attached as Exhibit 5.1 hereto. A copy of the Underwriting Agreement is filed herewith as Exhibit 1.1 and is incorporated herein by reference. The foregoing description of the Offering by the Company and the documentation related thereto does not purport to be complete and is qualified in its entirety by reference to such Exhibits.

ITEM 8.01. OTHER EVENTS.

On May 6, 2010, the Company issued a press release announcing that it had priced the public offering described in Item 1.01 of this Current Report on Form 8-K. The Company s press release is filed as Exhibit 99.1 to this Report and is incorporated herein by reference.

In connection with the public offering described in Item 1.01 of this Current Report on Form 8-K, the Company included the following updated business overview in the prospectus supplement dated May 6, 2010.

Company overview

We develop novel, targeted 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 be stably attached to the antibodies while in the blood stream and 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 a particular 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. Our TAP technology is designed to enable the creation of highly effective, well-tolerated anticancer products.

We believe that our TAP technology and our expertise in the development and humanization of monoclonal antibodies will enable us to become a leader in the application of antibody-based anticancer compounds. We plan to achieve this goal through the development of our own anticancer products and through collaborations with other companies. There are now six TAP compounds in clinical trials through our own programs and those of several of our collaborators. Our collaborators currently include: Amgen, Bayer HealthCare, Biogen Idec, Biotest, Genentech (a wholly owned member of the Roche Group) and sanofi-aventis.

On April 29, 2010, we reported our financial results for the third quarter of fiscal year 2010, ended March 31, 2010, including a balance of cash and marketable securities of approximately \$42.2 million.

Our product candidates

T-DM1

The most advanced compound in our pipeline is trastuzumab-DM1, or T-DM1, which is in global development by Roche for the treatment of HER2+ metastatic breast cancer, or MBC. T-DM1 consists of our DM1 cell-killing agent attached to trastuzumab, which is the active component of the marketed anticancer compound, Herceptin®. Herceptin was developed by Genentech, a wholly owned member of the Roche Group.

In April 2010, Roche reported that, based on discussions with the U.S. Food and Drug Administration, or FDA, Roche plans to submit a marketing application to the FDA for T-DM1 for the treatment of third-line or later HER2+ MBC in the United States in 2010. Assuming

Roche submits its application in 2010, we believe Roche could receive marketing approval for T-DM1 in the United States in late 2010 or early 2011. Roche noted that the basis for this application is to be the Phase II clinical trial that was reported at the San Antonio Breast Cancer Symposium, or SABCS, in December 2009 that was designed to enroll 100 patients.

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This Phase II clinical trial enrolled 110 patients with advanced HER2+ MBC that had undergone prior treatment with regimens that included an anthracycline, a taxane, Herceptin, Tykerb® and Xeloda®. The T-DM1 objective response rate, or ORR, was

32.7%, as assessed by an independent review facility, or IRF. ORR is the proportion of patients in the trial who had a durable complete or partial response to treatment with T-DM1, and was the primary endpoint of the trial. The clinical benefit rate, or CBR, was 44.5%, as assessed by an IRF. CBR includes patients who had stable disease for six months or longer as well as patients who had an objective response to T-DM1. The percentage of patients treated with T-DM1 whose best response was assessed to be progressive disease, which we categorize as not having had clinical benefit, was 18.2%. Data from this clinical trial also suggested that T-DM1 could provide better tolerability than standard chemotherapy-containing treatment regimens. The toxicities of T-DM1 reported were considered to be acceptable, manageable and consistent with those reported in other T-DM1 trials.

Roche has discussed other clinical trials that are planned or underway with T-DM1, including:

- A Phase III clinical trial (EMILIA) that compares T-DM1 used alone to Tykerb used together with Xeloda as second-line therapy for HER2+ MBC. This trial is designed to enroll 580 patients, and its primary endpoint is progression-free survival. The trial commenced in February 2009, and Roche has disclosed that this trial could lead to a potential regulatory submission with the FDA and in the European Union during 2012 for T-DM1 for second-line use in HER2+ MBC. We believe that Roche will provide information related to this trial during 2010, such as an update on the status of patient enrollment.
- A Phase III clinical trial (MARIANNE) to assess T-DM1 as a first-line treatment for HER2+ MBC. The trial will assess T-DM1 used alone against T-DM1 used together with pertuzumab and against Herceptin used together with a taxane and is designed to enroll 1,092 patients. Roche has indicated that this trial is expected to commence in the second half of 2010 and will have as a primary endpoint progression-free survival. Roche has disclosed that this trial could lead to a potential regulatory submission for T-DM1 use as a first-line treatment for HER2+ MBC, and the timing would be after 2013, the latest period of its projections.
- A Phase II clinical trial assessing T-DM1 as a first-line therapy for HER2+ MBC that compares T-DM1 used alone against trastuzumab used together with docetaxel. This trial is designed to include 120 patients, and its primary endpoint is progression-free survival. Roche has indicated that it expects to report preliminary data from this trial at the European Society for Medical Oncology, or ESMO, annual meeting in October 2010.
- A Phase Ib/II clinical trial assessing the tolerability of T-DM1 used together with pertuzumab. This trial was designed to enroll 40 patients. Findings from this trial have been accepted to be reported at the American Society of Clinical Oncology, or ASCO, meeting in June 2010.

In addition to the trials discussed above, several studies are underway that assess T-DM1 used in combination with other anticancer agents. We believe that additional clinical data with T-DM1, used alone or in combination, will be reported at SABCS in December 2010.

Roche has indicated that it believes that peak T-DM1 sales, if it is approved, could be between 2 and 5 billion Swiss francs annually. Roche has reported that there are approximately 6,100 HER2+ MBC patients in the United States that are eligible for second-line treatments and approximately 8,300 such patients in five major markets in the European Union, and that there are approximately 7,600 HER2+ MBC patients in the United States that are eligible for third-line and later treatments and approximately 5,450 such patients in five major markets in the European Union. We believe that T-DM1 has the potential to be a valuable new pharmaceutical for the treatment of patients with HER2+ MBC.

Lorvotuzumab mertansine

Our most advanced wholly owned compound is lorvotuzumab mertansine, which we previously called IMGN901. The target for this TAP compound, CD56, is found on a number of tumor types, including small-cell lung cancer, ovarian cancer, Merkel cell carcinoma and the liquid tumor, multiple myeloma. We believe lorvotuzumab mertansine has the potential to be the first effective antibody-based therapy for the treatment of these targeted cancers. Based on scientific literature and/or our own studies, we believe that CD56 is expressed on approximately 100% of small-cell lung cancer and Merkel cell carcinoma cases, 58% of ovarian cancer cases, and 70% of multiple myeloma cases. Based on American Cancer Society estimates, we believe that approximately 43,900 new cases of small-cell lung cancer, 21,550 new cases of ovarian cancer and 20,580 new cases of multiple myeloma will be diagnosed in the United States in 2010. Based on other published data, we believe approximately 1,900 new cases of Merkel cell carcinoma will be diagnosed in the United States in 2010. In the case of small-cell lung cancer newly diagnosed patients generally respond to their first treatment regimen, but typically their disease then recurs. While many patients with recurrent small-cell lung cancer could be eligible for additional treatment, survival at this stage is usually less than 6 months. Metastatic Merkel cell carcinoma is also associated with a poor outcome, with a median survival time of 6.8 months. Therefore, there is an unmet medical need to treat these patient populations.

We are evaluating lorvotuzumab mertansine for the treatment of CD56+ cancers, focusing on small-cell lung cancer, Merkel cell carcinoma and ovarian cancer in a two-phase Phase I clinical trial that we call Study 002. This trial was designed to determine the maximum tolerated dose of lorvotuzumab mertansine when dosed daily for three consecutive days in a 21-day cycle and then expand into the second phase, or expansion phase, designed to gain additional experience with lorvotuzumab mertansine when dosed at the previously determined maximum tolerable dose. We are encouraged by the findings to date. We plan to use data from the Phase I clinical trial, together with input gained from regulatory agencies, to make a decision in late 2010 as to whether to commence a pivotal Phase II clinical trial of lorvotuzumab mertansine for the treatment of Merkel cell carcinoma in 2011. We also expect that findings from the ongoing clinical trial will help inform our future evaluation of the compound for ovarian cancer, a more prevalent cancer than Merkel cell carcinoma.

In November 2009, we reported interim results from Study 002 with respect to the six patients with Merkel cell carcinoma that had received lorvotuzumab mertansine at that time. All of these patients had received prior chemotherapy regimens for their cancer and entered the trial with metastatic disease. Two of these six patients had a marked, objective response to treatment with lorvotuzumab mertansine, while a third patient had clinically relevant stable disease for this patient population. One of these three patients had a partial response, or PR, after the first lorvotuzumab mertansine treatment cycle and reached a complete response, or CR, by the end of the third treatment cycle. This patient has been in remission for more than four years. The second patient had marked tumor reduction after the first lorvotuzumab mertansine treatment cycle, but declined further therapy due to the occurrence of an adverse event. This patient had a confirmed PR and based on clinical exam has shown continued improvement in her tumors for over eight months. The third patient entered this Phase I trial with bone metastases and had previously been treated with three different combination regimens of chemotherapy. On treatment with lorvotuzumab mertansine, this patient had stable disease that lasted for 79 days. Lorvotuzumab mertansine was found to be generally well tolerated. In the dose-escalation phase of this trial, the maximum tolerated dose was established at 75 mg/m²/day. We are now dosing patients at 60 mg/m²/day in the expansion phase of Study 002 to gain additional experience with the compound when administered at that dose. We are submitting an abstract with updated findings from Study 002 for presentation at the ESMO annual meeting in October 2010.

In July 2009, we reported findings for the 68 small-cell lung cancer patients that had been treated to date with lorvotuzumab mertansine in either Study 002 or in another of our Phase I trials, called Study 001. All of these patients had received prior chemotherapy, and most had received at least two previous regimens. The estimated clinical benefit rate was 25%, consisting of patients with an objective response and/or sustained stable disease, defined as non-progression for at least 77 days. An objective response was reported in a patient whose small-cell lung cancer had recurred within four months of treatment with cisplatin, etoposide, and topotecan plus radiation therapy. This patient had a PR after his first lorvotuzumab mertansine treatment cycle and reached a 91% reduction in tumor size by the end of his third cycle. His disease progressed after his fourth cycle, which was 24 weeks after he first received lorvotuzumab mertansine. Another patient had an objective response (an unconfirmed PR) and no evidence of disease progression for more than 8 weeks. This patient had previously undergone two other treatment regimens for the cancer. Fifteen patients had sustained stable disease, with an estimated time-to-progression, or TTP, ranging from 77 to 168 days, or 11 to 24 weeks. Lorvotuzumab mertansine was found to be generally well tolerated.

In December 2009, we reported at the annual meeting of the American Society of Hematology, or ASH, interim results from our Phase I clinical trial, called Study 003, that assesses lorvotuzumab mertansine when used alone to treat multiple myeloma that has progressed on approved therapies. The findings reported were for the 26 patients enrolled in this trial at that time. One patient had a PR while receiving lorvotuzumab mertansine. This patient has continued on treatment for more than a year. Three patients had a minimal response, or MR, while receiving lorvotuzumab mertansine and two of these patients remained on treatment for at least 45 weeks. The third patient withdrew from the trial due to a broken leg while continuing to show disease improvement. Eleven patients had stable

disease, or SD, with eight of these patients remaining on treatment for at least 12 weeks at the time of data cut-off for presentation of the data. These include four patients who have received lorvotuzumab mertansine for at least 24 weeks and two other patients still undergoing treatment. Ten patients remained on lorvotuzumab mertansine longer than on regimens received earlier in the course of their disease, and eight of these patients were on lorvotuzumab mertansine longer than on their last regimen with approved therapies. Lorvotuzumab mertansine was found to be generally well tolerated and was not associated with significant myelosuppression or other side effects that would limit its ability to be administered in combination with other active agents. Lorvotuzumab mertansine was granted orphan drug designation in the United States and similar designation in the European Union for Merkel cell carcinoma in early 2010. If a product that has orphan drug designation subsequently receives the first FDA approval for the disease for which it has such designation, the product is entitled to orphan product exclusivity, which means that the FDA may not approve any other applications to market the same drug for the same indication, except in very limited circumstances, for seven years.

In addition to the trial information discussed above, we are actively engaged in several other planned and ongoing clinical trials with lorvotuzumab mertansine, including:

- A Phase I/II clinical trial, called Study 007, to assess the safety and provide information on the efficacy of lorvotuzumab mertansine when used in combination with etoposide/carboplatin as a first-line treatment of small-cell lung cancer. We plan to commence this trial by late 2010. Assuming satisfactory safety data are obtained in the first phase of this trial, we plan to randomize patients during the second phase of this trial to compare lorvotuzumab mertansine used with etoposide/carboplatin against etoposide/carboplatin used alone, which is the current standard of care for first-line treatment of small-cell lung cancer.
- Two Phase I clinical trials evaluating lorvotuzumab mertansine for the treatment of multiple myeloma are underway. Study 003, as has been discussed, evaluates lorvotuzumab mertansine when used as a single agent and is currently in the expansion phase. Study 005 is designed to assess the tolerability of lorvotuzumab and gain information on its efficacy when used in combination with the standard treatment for this cancer, lenalidomide plus dexamethasone. We expect to report interim data from one or both of these trials at the ASH annual meeting in December 2010.
- We plan to make a decision in late 2010 on whether to commence a pivotal Phase II clinical trial of lorvotuzumab mertansine for the treatment of Merkel cell carcinoma. This decision will be informed by a number of considerations, including additional findings in Study 002 and the input obtained from regulatory agencies on trial design.

SAR3419

We created SAR3419 for the treatment of non-Hodgkin's lymphoma and licensed it to sanofi-aventis as part of a broader collaboration. SAR3419 consists of our DM4 cell-killing agent attached using one of our engineered linkers to a CD19-binding antibody that was created and humanized by us.

Sanofi-aventis is evaluating SAR3419 for the treatment of non-Hodgkin's lymphoma in two Phase I clinical trials that have different dosing schedules. The first study evaluated the compound when dosed once every three weeks and initial findings from it have been reported. We expect data from the second Phase I trial, which evaluates the compound when dosed weekly, to be reported at the ASH annual meeting in December 2010. We expect SAR3419 to advance into Phase II clinical testing in the second half of 2010.

The findings from the first Phase I clinical trial were reported at the ASH annual meeting in December 2009. The trial found that 17 of 27 of patients, or 63%, who were response-evaluable at the time of data cut-off for presentation experienced a reduction in tumor size (7% to 86% reduction). These included 7 of 14 patients, or 50%, who had disease that was refractory to treatment with rituximab. Five patients had an objective response, all of whom received SAR3419 at its maximum tolerated dose or the next highest or lowest dose. Among these responders was a patient with rituximab-refractory disease. All but one of these five patients reached the best response either during the last treatment cycle allowed under the trial protocol, which was cycle 6, or after their last dose of SAR3419. This is consistent with the observation that the best response to treatment typically occurred after a patient had received several doses of SAR3419. A primary endpoint of the trial was to establish the maximum tolerable dose of SAR3419 when administered once every three weeks. This was determined to be 160 mg/m². Additional patients will receive SAR3419 at this dose to gain more information on the tolerability and activity of the compound when administered at its maximum tolerable dose.

Other product candidates under development

In addition to T-DM1, lorvotuzumab mertansine and SAR3419, several other TAP compounds are in development through our own programs and those of our partners, including:

Proprietary ImmunoGen product candidates

- IMGN388 is a TAP compound consisting of our DM4 cell-killing agent attached to an integrin-targeting antibody that was developed by Centocor. IMGN388's target occurs on many types of solid tumors and also on vascular endothelial cells in the process of forming new blood vessels, or angiogenesis. Angiogenesis is needed for a tumor to grow. IMGN388 is in Phase I testing and clinical data from this trial have been accepted for poster presentation at ASCO in June 2010.
- We have three TAP compounds currently in or positioned to begin preclinical toxicology studies. One of these compounds, IMGN529, is being developed for the treatment of certain liquid tumors and we expect to submit an investigational new drug, or IND, application to the FDA for this product candidate in 2011. One of the other compounds is a potential treatment for certain liquid tumors and the other is a potential treatment for certain solid tumors.

Partnered product candidates

- BT-062 was created by Biotest under a 2006 license that grants Biotest the exclusive right to use our maytansinoid TAP technology with antibodies that target CD138, an antigen found on multiple myeloma and certain other cancers. BT-062 consists of Biotest's anti-CD138 antibody with our DM4 cell-killing agent attached using one of our engineered linkers. Biotest advanced BT-062 into Phase I evaluation in September 2008 and initial results from this trial were reported at the ASH annual meeting in 2009. We have opt-in rights on BT-062 for the United States.
- BIIB015 was created by Biogen Idec under a 2004 license that grants Biogen Idec the exclusive right to use our maytansinoid TAP technology with antibodies that target Cripto, an antigen found on a number of solid tumors. BIIB015 consists of Biogen Idec's Cripto-binding antibody with our DM4 cell-killing agent attached using one of our engineered linkers. BIIB015 advanced into Phase I testing in the summer of 2008.
- We expect two compounds to advance into clinical testing in 2010 through our collaboration with sanofi-aventis.
- In October 2008, we entered into a development and license agreement with Bayer HealthCare AG. The agreement grants Bayer HealthCare exclusive rights to use our maytansinoid TAP technology to develop and commercialize therapeutic compounds to a specific target. We recently achieved a milestone payment for their achievement of a preclinical event under this collaboration.
- Amgen has taken two licenses to use our TAP technology with antibodies to undisclosed targets, and Genentech, now a wholly owned member of the Roche Group, has taken four licenses in addition to the HER2 license that enabled development of T-DM1.

We continue to conduct research to develop additional cell-killing agents and linkers to further strengthen our position in the field, and expect over the next several years to be involved in numerous clinical trials for existing and new product candidates focused on various stages of development ranging from early stage to registration trials. We believe our continued focus on development of additional applications of our TAP technology could provide additional opportunities for partnerships and collaborations.

Our TAP technology

We developed our TAP technology to achieve highly effective, well tolerated anticancer drugs. Terms used to refer to our field include armed antibodies, empowered antibodies and antibody-drug conjugates, or ADCs. Our TAP technology and/or antibody expertise has generated over \$230 million in payments to us from our partners since 2000. Our existing collaboration and license agreements with partners have the potential to generate approximately \$565 million in additional payments to us in connection with potential development, clinical and regulatory milestones.

Traditional chemotherapy agents typically kill any rapidly dividing cell, including healthy cells, which can result in significant adverse side effects and limit their ability to be dosed

to full efficacy. Monoclonal antibodies can be created that bind specifically to targets found on cancer cells and, therefore, offer the potential to selectively target cancer cells. The invention of such antibodies has led to the creation of some successful anticancer therapeutics such as Rituxan® and Herceptin®. For many of the antigens found on cancer cells, however, the binding of a manufactured antibody to that antigen in and of itself has little, if any, anticancer effect.

Our TAP technology makes use of the targeting ability of monoclonal antibodies without needing the antibody to have meaningful anticancer activity on its own. A TAP compound consists of a tumor-targeting antibody with one of our highly potent cell-killing agents attached using one of our engineered linkers. The antibody serves to deliver our potent cell-killing agent specifically to cancer cells, to help minimize damage to healthy tissue. The cell-killing agent serves to kill the cancer cell. Our agents are far more potent than traditional chemotherapies. Our engineered linkers serve to keep the cell-killing agent attached to the antibody while the TAP compound is circulating in the bloodstream and then control its release once the TAP compound has bound to and entered a cancer cell.

We develop our own monoclonal antibodies for use in our proprietary products and also license to other companies the right to use our TAP technology with their antibodies to develop products for specific targets.

Herceptin® is a registered trademark of Genentech, a wholly owned member of the Roche Group. Rituxan® is a registered trademark of Biogen Idec Inc. Tykerb® is a registered trademark of GlaxoSmithKline plc. Xeloda® is a registered trademark of Roche. Other brands, names and trademarks contained herein are the property of their respective owners.

Special note regarding forward-looking statements

This Current Report on Form 8-K contains forward-looking statements within the meaning of Section 27A of the Securities Act of 1933, as amended, and Section 21E of the Securities Exchange Act of 1934, as amended. These statements relate to future events and our future financial performance.

These forward-looking statements are identified by their use of terms and phrases, such as anticipate, believe, could, estimate, expect, intend, may, plan, predict, project, will and other similar terms and phrases, including references to assumptions. These statements are contained in Risk Factors section, as well as other sections of this prospectus supplement.

Forward-looking statements in this Current Report on Form 8-K include, but are not limited to:

- our and our collaborators' expectations regarding clinical trials, development timelines and regulatory filings for T-DM1, lorvotuzumab mertansine, SAR3419, IMGN388 and other drug candidates under development by us and our collaborators;
- Roche's plan to submit a marketing application to the FDA for T-DM1 for the treatment of third-line and later HER2+ MBC in the United States in 2010 on the

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basis of the Phase II study of T-DM1 as a third-line treatment in HER2+ MBC that was presented at the San Antonio Breast Cancer Symposium in December 2009;

- our belief that Roche could receive marketing approval of T-DM1 in the United States in late 2010 or early 2011 assuming Roche submits its application in 2010;
- Roche's expectation that it expects interim data from a Phase Ib/II clinical trial assessing T-DM1 plus pertuzumab to be reported at ASCO in June 2010 and that it expects preliminary data from a Phase II clinical trial comparing T-DM1, as a single agent, against trastuzumab plus docetaxel for first-line treatment of HER2+ MBC to be reported at the ESMO meeting in October 2010;
- Roche's expectation to start a Phase III clinical trial to assess T-DM1 as a first-line treatment for HER2+ MBC in the second half of 2010;
- the expectation that Roche could file a marketing application for T-DM1 as second-line treatment in HER2+ MBC with the FDA and in the European Union during 2012 and as a first-line treatment with the FDA after 2013, that Roche will provide information related to the EMILIA trial during 2010, that additional clinical data with T-DM1, used alone or in combination, will be reported at SABCS in December 2010, and that peak T-DM1 sales could be between 2 and 5 billion Swiss francs annually;
- our belief that T-DM1 has the potential to be a valuable new pharmaceutical for the treatment of patients with HER2+ MBC and that lorvotuzumab mertansine has the potential to be the first effective antibody-based therapy for certain targeted cancers;
- our expectation as to the number of cases of small-cell lung cancer, ovarian cancer, multiple myeloma and Merkel cell carcinoma that will be diagnosed in the United States in 2010;
- our plan to use data from the ongoing clinical trial of lorvotuzumab mertansine, together with input gained from regulatory agencies, to make a decision in late 2010 as to whether to commence a pivotal Phase II clinical trial of lorvotuzumab mertansine for the treatment of Merkel cell carcinoma in 2011 and our expectations as to the design of this trial;
- our plan to start a Phase I/II clinical trial by late 2010 to evaluate lorvotuzumab mertansine in combination with etoposide/carboplatin, the standard care, for first-line treatment of small-cell lung cancer;
- our expectation to report interim data from one or more of our clinical trials of lorvotuzumab mertansine at the ESMO annual meeting in October 2010 and/or the ASH annual meeting in December 2010;

- sanofi-aventis expectation to report certain Phase I data for SAR3419 at the ASH annual meeting in December 2010 and to advance SAR3419 into Phase II clinical testing in the second half of 2010;

- our expectation that IMG388 Phase I data will be reported at ASCO in June 2010 and to submit an IND application to the FDA for IMG529 in 2011;
- our expectation that two compounds will advance into clinical testing in 2010 through our collaboration with sanofi-aventis;
- our expectation that our TAP technology potentially may be used with antibodies with limited or no anticancer activity of their own, enabling effective antibody-based therapies to be developed for many more types of cancers and that over the next several years we will be involved in numerous clinical trials for existing and new product candidates focused on various stages of development ranging from early stage to registration trials;
- our expectation of the amount and timing of future revenues, potential development, clinical and regulatory milestones, expenses, investments and other items affecting the results of our operations; and
- our expected uses of the net proceeds of this offering.

These forward-looking statements involve known and unknown risks, uncertainties and other factors that may cause actual results to be materially different from those contemplated by our forward-looking statements. These known and unknown risks, uncertainties and other factors are described in detail in our Annual Report on Form 10-K for the fiscal year ended June 30, 2009 and our subsequent Quarterly Reports on Form 10-Q. We disclaim any intention or obligation to update or revise any forward-looking statements, whether as a result of new information, future events or otherwise.

ITEM 9.01. FINANCIAL STATEMENTS AND EXHIBITS

(d) The following exhibits are being filed herewith:

Exhibit No.	Exhibit
1.1	Underwriting Agreement dated May 6, 2010 by and between ImmunoGen, Inc. and J.P. Morgan Securities Inc., as representative of the several underwriters named in Schedule 1 thereto
5.1	Opinion of Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C.
23.1	Consent of Mintz, Levin, Cohn, Ferris, Glovsky and Popeo, P.C. (included in the opinion filed as Exhibit 5.1)
99.1	Press release of ImmunoGen, Inc. dated May 6, 2010

SIGNATURES

Pursuant to the requirements of the Securities Exchange Act of 1934, the Registrant has duly caused this report to be signed on its behalf by the undersigned hereunto duly authorized.

ImmunoGen, Inc.
(Registrant)

Date: May 7, 2010

/s/ Daniel M. Junius

Daniel M. Junius
President and Chief Executive Officer

EXHIBIT INDEX

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