WASHINGTON, DC, APRIL 8, 2013 --- Immunomedics, Inc. (Nasdaq: IMMU), a biopharmaceutical company primarily focused on the development of monoclonal antibody-based products for the targeted treatment of cancer, autoimmune and other serious diseases, today announced the creation of a new class of antibody-cytokine conjugates using the Company’s patented DOCK-AND-LOCK™ (DNL™) platform technology. These DNL™ complexes demonstrated potent anti-tumor activity in preclinical studies.

The Company has previously reported the development of antibody-directed interferon-α2b (IFNα2b) complexes. (For more information, please refer to the Company’s press release at www.immunomedics.com/pdfs/news/2009/PR04222009A.pdf). Veltuzumab-IFNα2b is the most advanced product from this group of IFNα-based DNL™ complexes, and is currently being developed for improved therapy of B-cell malignancies, partially supported by a grant from the Small Business Innovation Research program of the National Cancer Institute totaling $2.8 million.

While functioning similarly to IFN-α in eliciting anti-viral, anti-tumor, and immune-modulating activities, IFN-λ is being considered as a potential alternative to existing IFN-α therapeutic regimens due to its more restricted cellular targets. Antibody-targeted IFN-λ may further improve its safety, potency and pharmacokinetics.

In the current study, 3 DNL™ complexes of IFN-λ, designated (E1)-λ1, (15)-λ1, and (C2)-λ1, were generated by site-specifically conjugating IFN-λ1 to 3 of the Company’s proprietary humanized antibodies, hRS7 (anti-TROP-2), hMN-15 (anti-CEACAM6), and hL243 (anti-HLA-DR), respectively. These antibody-cytokine conjugates were evaluated in human malignant cell lines of cervix, colon, esophagus, lung, liver, and skin.

Targeting of these antibody-cytokine conjugates to antigen-expressing cells markedly increased the amount of IFN-λ1 localized at the cell surface. As a result, (E1)-λ1 inhibited the in vitro proliferation of the cervical, lung and esophageal cancer cell lines at less than 1 picomolar (pM) concentration, which was 1,000-fold more potent than commercial IFN-λ1. Likewise, the anti-proliferation activity of (15)-λ1 was enhanced ~100-fold in cervical and esophageal cancer cells, but not in CEACAM6-negative lung cancer cells.

“These promising results, attributable to increased localization and stronger binding to antibody-targeted cells, warrant further exploration as potential cancer therapeutics,” remarked Cynthia L. Sullivan, President and Chief Executive Officer.
Cancers are generally believed to be derived from single cells that are genetically mutated, resulting in many divisions resulting in an expanding tumor that eventually spreads to local and distant organs. Cancer deaths usually are due to this metastasis affecting the new organs invaded. The initiating cells are often referred to as cancer stem cells. How these turn into heterogeneous populations of cancer cells within the original tumor and even in the metastases is the subject of intense recent research, which has also focused on the tumor’s microenvironment and how cancer cells interact with their neighboring supportive cells.

In a separate poster presentation at the same AACR Annual Meeting, the Company reported that genes of human cancer jump into adjacent normal cells by fusion of both cells. This study was done in collaboration with researchers from the Garden State Cancer Center, Center for Molecular Medicine and Immunology in Morris Plains, NJ, and the Laboratory of Pathology, Center for Cancer Research, National Cancer Institute, NIH, in Bethesda, MD.

This team of scientists discovered a transfer of human genes to normal hamster cells when cells from tissues involved by Hodgkin’s lymphoma from two patients were implanted to hamsters. In all, 7 of 24 human genes tested were present in the malignant tumors growing in the hamsters for extended periods of 5 to 6 years, with evidence of metastasis in the hamsters within 21 days of the original grafting of the human tumors. By means of sensitive DNA coloring of the tumor cells, the investigators proved that human and hamster DNA were present in single cancer cells, representing fused cells of both species.

“These findings may explain how cancers develop and change with time, spreading by metastasis to other organs by overcoming immunity to the cancer cells and could lead to therapeutic strategies,” commented the lead investigator, Dr. David M. Goldenberg, Chief Scientific Officer and Chief Medical Officer of Immunomedics, and President of the Center for Molecular Medicine and Immunology.

About Immunomedics
Immunomedics is a New Jersey-based biopharmaceutical company primarily focused on the development of monoclonal antibody-based products for the targeted treatment of cancer, autoimmune and other serious diseases. We have developed a number of advanced proprietary technologies that allow us to create humanized antibodies that can be used either alone in unlabeled or “naked” form, or conjugated with radioactive isotopes, chemotherapeutics, cytokines or toxins, in each case to create highly targeted agents. Using these technologies, we have built a pipeline of therapeutic product candidates that utilize several different mechanisms of action. We also have a majority ownership in IBC Pharmaceuticals, Inc., which is developing a novel DOCK-AND-LOCK™ (DNL™) method with us for making fusion proteins and multifunctional antibodies, and a new method of delivering imaging and therapeutic agents selectively to disease, especially different solid cancers (colorectal, lung, pancreas, etc.), by proprietary, antibody-based, pretargeting methods. We believe that our portfolio of intellectual property, which includes approximately 220 active patents in the United States and more than 400 foreign patents, protects our product candidates and technologies. For additional information on us, please visit our website at www.immunomedics.com. The information on our website does not, however, form a part of this press release.
This release, in addition to historical information, may contain forward-looking statements made pursuant to the Private Securities Litigation Reform Act of 1995. Such statements, including statements regarding clinical trials, out-licensing arrangements (including the timing and amount of contingent payments), forecasts of future operating results, potential collaborations, and capital raising activities, involve significant risks and uncertainties and actual results could differ materially from those expressed or implied herein. Factors that could cause such differences include, but are not limited to, risks associated with any cash payment that the Company might receive in connection with a sublicense involving a third party and UCB, which is not within the Company’s control, new product development (including clinical trials outcome and regulatory requirements/actions), our dependence on our licensing partners for the further development of epratuzumab and veltuzumab for non-cancer indications, competitive risks to marketed products and availability of required financing and other sources of funds on acceptable terms, if at all, as well as the risks discussed in the Company’s filings with the Securities and Exchange Commission. The Company is not under any obligation, and the Company expressly disclaims any obligation, to update or alter any forward-looking statements, whether as a result of new information, future events or otherwise.

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