

IMMUNOMEDICS DEVELOPS NOVEL ANTIBODY-BASED THERAPEUTICS FOR MANTLE CELL AND OTHER LYMPHOMAS**-- Three Preclinical Studies were Presented at the 2011 ASH Annual Meeting --**

San Diego, CA, December 12, 2011 -- 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 that two new bispecific antibodies that target both the CD20 and CD74 receptors on B cells exhibited potent anti-tumor activities against mantle cell (MCL) and other lymphomas in preclinical studies.

MCL is an aggressive form of B-cell non-Hodgkin lymphoma (NHL) for which there is no standard of care. About 3,500 new cases are detected every year in the United States with the disease being more prominent among adults 60 years and older. Chemotherapy along with the chimeric anti-CD20 antibody, rituximab, increases overall survival but the disease relapses in virtually all patients. Recently, the potential advantage of targeting both CD20 and CD74 was reported in a preclinical study in which milatuzumab, the Company's humanized anti-CD74 antibody, combined with rituximab and a crosslinking antibody resulted in anti-tumor activity in MCL cell lines and patient samples *in vitro*.

An attractive alternative to combination therapy is the use of antibodies that can target both antigens simultaneously. For this purpose, two new bispecific antibodies were generated using the Company's proprietary protein conjugation technology, Dock-and-Lock (DNL). 74-(20)-(20) was created by linking four fragments of veltuzumab, the Company's humanized anti-CD20 antibody, to milatuzumab. Likewise, 20-(74)-(74) contains four fragments of milatuzumab fused to veltuzumab.

The two anti-CD20/CD74 antibodies potently inhibited the growth of MCL cell lines, as well as other NHL cell lines, at a low concentration of 10 nanomolar without the need of a secondary, crosslinking antibody. In contrast, neither parental antibody, alone or in combination, was active under the same conditions, suggesting the requirement of placing CD74 and CD20 in close proximity for the observed cytotoxicity.

Both of the hexavalent DNL constructs induced 25-30% cell death in MCL cell lines and in clinical samples from MCL and chronic lymphocytic leukemia (CLL) patients, compared to 10-15% seen with the parental antibodies, alone or in combination. In a mouse model of human MCL, 20-(74)-(74) and 74-(20)-(20) were effective at the 370- μ g dose level, resulting in 60% and 30% increases in median survival time, respectively, compared to saline controls.

The effects of adding FTY720 to the two bispecific antibodies were reported in the second study presented at the same meeting. FTY720, also known as fingolimod, is an immunosuppressive agent approved by the FDA as a frontline treatment for relapsing multiple sclerosis. It has

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shown promising pre-clinical activity in CLL and MCL. More recently, FTY720 was also found to increase CD74 expression and sensitize MCL cells to milatuzumab-mediated cell death.

In the second preclinical study, FTY720 induced an almost 6-fold increase in the levels of CD74 on various MCL cell lines tested, a modest decrease of CD20, and no apparent change in CD22 and HLA-DR expression, when compared to untreated controls. The decrease in CD20 expression was later confirmed as due to non-specific toxicity by the drug. Combined treatments of FTY720 and 20-(74)-(74) at 4 μ M and 33 nM, respectively, resulted in about 85% cell death, which is statistically significant when compared to 20-(74)-(74) and FTY720 alone (30% and 55% cell deaths, respectively). Similar treatments with FTY720 and 74-(20)-(20) resulted in about 75% cell death, also with statistically significant *P* values compared to each agent alone.

These results indicate that anti-MCL activity of the two novel bispecific anti-CD20-/CD74 antibodies can be significantly enhanced by FTY720. Studies are underway to evaluate similar cytotoxic effects in mouse models of MCL and to define the mechanisms of action of the combination effects.

The mechanism by which FTY720 increases CD74 expression was examined by the Company's collaborators at The Ohio State University, Columbus, OH, in the third study. The researchers found that the phosphorylation of FTY720 and its interaction with receptors known as sphingosine-1-phosphate are not required for FTY720-mediated cell death. Instead, the movement of the enzyme, lysosomal hydrolase, from the cell membrane to the interior of the cell was responsible for cell death. This disruption of the autophagic-lysosomal pathway, a cellular process for the degradation and disposal of cell waste, by FTY720 led to increased levels of CD74, thereby sensitizing cells such as MCL to milatuzumab-mediated cell death.

The Ohio State University scientists tested their hypothesis that FTY720 sensitizes MCL cells to milatuzumab in a mouse model of human MCL. Mice were divided into 5 groups with each group treated with placebo (saline), trastuzumab (a non-MCL specific antibody), FTY720, milatuzumab or the combination of FTY720 and milatuzumab. The primary end-point of this study was survival.

The median survival time for the combination-treated group was 36 days, 27 days for the trastuzumab-treated mice, 31 days for the FTY720-treated mice, and 33.5 days for the milatuzumab-treated mice, compared to 28 days for the saline-treated mice. Thus, the combination treatment significantly prolonged survival compared to the two control groups ($P < 0.0001$), FTY720 ($P = 0.0001$) and milatuzumab ($P = 0.0048$). These findings support clinical evaluation of this combination in patients with MCL.

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) methodology 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 185 patents issued 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 new product development (including clinical trials outcome and regulatory requirements/actions), our dependence on our licensing partners for the further development of epratuzumab for autoimmune indications 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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