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Caprion Highlights Recently Published Data on Strengthening Immune Response in HIV Utilizing CD160 and PD1-Targeted Antibodies

*The new findings also have positive implications in the
treatment of cancer with checkpoint blockade therapies*

Montreal, Canada – October 9, 2014— Caprion announced today that its immune monitoring business unit, ImmuneCarta™, published data regarding the enhancement of HIV (Human Immunodeficiency Virus)-specific T cell responses using antibodies directed against CD160 and PD-1 checkpoint molecules. Data was jointly published with a sponsor in the recent issue of *The Journal of Translational Medicine*, entitled “CD160 isoforms and regulation of CD4 and CD8 T-cell responses”. The full article is presently available on Open Access at <http://www.translational-medicine.com/content/pdf/s12967-014-0217-y.pdf>.

One of the challenges in fighting chronic HIV infection is the need to overcome a state known as T cell exhaustion. T cell exhaustion is the consequence of sustained immune activation and results in the accumulation of dysfunctional HIV-specific T cells and disease progression. Previous work done by ImmuneCarta has shown that CD160 and PD-1 expressing T cell subsets identify activated and exhausted HIV-specific CD8⁺ T cells.¹

Capitalizing on these findings, this study assessed whether antibodies specifically targeted to block the interaction between CD160 and HVEM (Herpes Virus Entry Mediator) protein would enhance and rescue

HIV-specific T-cell functionality. Through in vitro and ex vivo cellular assays, the study demonstrated that antibodies used in combination against CD160 and PD-1, significantly increased HIV-specific CD8⁺ T-cell proliferation. The enhanced immune response observed from this co-targeting strategy represents an interesting approach to complement current antiretroviral treatments and extends to several chronic inflammatory conditions which lead to T-cell dysfunction.

Yoav Peretz, Ph.D., Scientific Director at ImmuneCarta said, “These exciting findings have positive implications for HIV infected patients and also in the treatment of cancer where checkpoint blockade therapy has shown promising clinical response.” Dr. Peretz further commented, “HIV infection as utilized in this study appears to be an ideal model to monitor immune restoration and biological activity of novel oncology compounds targeting checkpoint molecules such as PD-1, LAG-3, Tim-3, CD160 and HVEM.”

Martin LeBlanc, CEO of Caprion, stated, “We are pleased to provide pharmaceutical and biotech companies with broad applicability of our immune monitoring expertise in multiple therapeutic areas, particularly in oncology and infectious disease. Checkpoint blockade therapeutics is the fastest growing sector of immunotherapy, and Caprion is delighted to be at the forefront.”

1. Peretz Y, He Z, Shi Y, et al. *CD160 and PD-1 co-expression on HIV-specific CD8 T cells defines a subset with advanced dysfunction*. PLoS Pathog Aug 2012; 8(8): e1002840.

About Caprion

Caprion is the leading provider of proteomics and immune monitoring services to the pharmaceutical and biotechnology industry. Its immune monitoring business unit, ImmuneCarta™, offers proprietary multiparametric flow cytometry for functional analyses of innate and adaptive immune responses. Caprion’s proteomics business unit, ProteoCarta™, offers proprietary gel-free, label-free mass spectrometry (MS) for comprehensive, quantitative and robust comparative measurement of proteins across large sets of biological samples for the discovery and validation of protein biomarkers. Caprion also leverages ProteoCarta to develop its own in-vitro diagnostic products targeting cancer, metabolic and infectious diseases. With research sites in Montreal, Canada and in Menlo Park, CA, Caprion has been providing large-scale proteomics and immune monitoring services to over 50 major pharmaceutical and biotech clients for more than 10 years. Caprion, a privately-held company, is majority owned by Chicago Growth Partners. For more information, please visit www.caprion.com.