



XBiotech Announces Presentation of Novel Findings for MABp1 in the Prevention of Coronary Thrombosis at the American Heart Association Scientific Sessions

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IL-1a Neutralizing Antibody MABp1 Found to Block NETs' Ability to Increase Endothelial Activation and Thrombogenicity

AUSTIN, Texas, Nov. 14, 2017 (GLOBE NEWSWIRE) -- XBiotech Inc. (NASDAQ:XBITE) announced today a presentation of findings on the role of IL-1 alpha (IL-1 α) associated with Neutrophil Extracellular Traps (NETs) in promoting endothelial activation and thrombogenicity. The oral presentation, entitled, "Neutrophil Extracellular Traps Increase Endothelial Activation and Thrombogenicity via IL-1 α and NF- κ B: Implications for Superficial Erosion", was given by researcher Thomas L. Mawson, Icahn School of Medicine at Mount Sinai, Sarnoff Fellow, Sarnoff Cardiovascular Research Foundation, at the American Heart Association Scientific Sessions on November 13 at 5:45 PM, PT. Dr. Eduardo Folco, scientist at the Cardiovascular Division of Brigham and Women's Hospital and Harvard Medical School, contributed equally to this study.

The findings resulted from an agreement between XBiotech, Brigham and Women's Hospital (BWH), and Massachusetts General Hospital to provide XBiotech's anti-IL-1 α antibody, MABp1, to Dr. Peter Libby. Dr. Libby, a cardiovascular medicine specialist at BWH and the Mallinckrodt Professor of Medicine at Harvard Medical School, was principal investigator of the research.

"I believe these data represent an exciting step forward in cardiovascular medicine," stated Dr. Libby. He further added, "They elucidate a novel mechanism that we believe can contribute to coronary thrombosis, a major cause of sudden cardiac death and acute myocardial infarction. We have demonstrated that MABp1, an anti-IL-1 α antibody, can block induction of key molecules in vascular endothelial cells that can promote erosion-associated thrombosis."

Coronary artery thrombosis can be caused by either plaque rupture or plaque erosion¹. Superficial plaque erosion causes up to one-third of all acute coronary syndromes^{2,3}. Erosion-prone atheromatous plaques, rather than lesions with stable or rupture-prone characteristics, associate with neutrophil extracellular traps (NETs)⁴. The study performed at Dr. Libby's laboratory probed the influence of NETs on the endothelial cell (EC) functions related to erosion-associated thrombosis. These data show that exposure of human saphenous veins ECs (HSVECs) to NETs increase expression of adhesion molecules on the surface of endothelial cells such as VCAM-1 and ICAM-1, which may participate in atherogenesis. In addition, pre-treatment of NETs with MABp1, an anti-IL-1 α -neutralizing antibody, or IL-1R antagonist, but not with an anti-IL-1 β -neutralizing antibody, blocked the initiation of VCAM-1, ICAM-1, and TF expression, each of which link to coronary thrombosis⁵. In conclusion, NETs increase thrombogenicity in vitro through a response mediated by IL-1 α . These data point to an important role for MABp1 therapy in heart disease. It also may expand treatment opportunities for other inflammatory diseases in which NETs play a deleterious role, such as cancer as well as pulmonary, autoimmune, and gastrointestinal diseases.

About True Human™ Therapeutic Antibodies

XBiotech's True Human™ antibodies are derived without modification from individuals who possess natural immunity to certain diseases. With discovery and clinical programs across multiple disease areas, XBiotech's True Human antibodies have the potential to harness the body's natural immunity to fight disease with increased safety, efficacy and tolerability.

About XBiotech

XBiotech is a fully integrated global biosciences company dedicated to pioneering the discovery, development and commercialization of therapeutic antibodies based on its True Human™ proprietary technology. XBiotech currently is advancing a robust pipeline of antibody therapies to redefine the standards of care in oncology, inflammatory conditions and infectious diseases. Headquartered in Austin, Texas, XBiotech also is leading the development of innovative biotech manufacturing technologies designed to more rapidly, cost-effectively and flexibly produce new therapies urgently needed by patients worldwide. For more information, visit www.xbiotech.com.

Cautionary Note on Forward-Looking Statements

This press release contains forward-looking statements, including declarations regarding management's beliefs and expectations that involve substantial risks and uncertainties. In some cases, you can identify forward-looking statements by terminology such as "may," "will," "should," "would," "could," "expects," "plans," "contemplate," "anticipates," "believes," "estimates," "predicts," "projects," "intend" or "continue" or the negative of such terms or other comparable terminology, although not all forward-looking statements contain these identifying words. Forward-looking statements are subject to inherent risks and uncertainties in predicting future results and conditions that could cause the actual results to differ materially from those projected in these forward-looking statements. These risks and uncertainties are subject to the disclosures set forth in the "Risk Factors" section of certain of our SEC filings. Forward-looking statements are not guarantees of future performance, and our actual results of operations, financial condition and liquidity, and the development of the industry in which we operate, may differ materially from the forward-looking statements contained in this press release. Any forward-looking statements that we make in this press release speak only as of the date of this press release. We assume no obligation to update our forward-looking statements whether as a result of new information, future events or otherwise, after the date of this press release.

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¹ Crea F, Libby P. Acute Coronary Syndromes: The Way Forward From Mechanisms to Precision Treatment. *Circulation* 2017;136:1155–1166.

² Franck G, et al. Flow Perturbation Mediates Neutrophil Recruitment and Potentiates Endothelial Injury via TLR2 in Mice – Implications for Superficial Erosion. *Circ Res* 2017;121:31-42.

³ Libby P. Superficial erosion and the precision management of acute coronary syndromes: not one-size-fits-all. [Eur Heart J](#). 2017;38(11):801-803. doi: 10.1093/eurheartj/ehw599.

⁴ Quillard T et al. TLR2 and neutrophils potentiate endothelial stress, apoptosis and detachment: implications for superficial erosion. [Eur Heart J](#). 2015 Jun 7;36(22):1394-404. doi: 10.1093/eurheartj/ehv044. Epub 2015 Mar 8.

⁵ [Tousoulis D](#), et al. Inflammatory and thrombotic mechanisms in coronary atherosclerosis. [Heart](#). 2003;89(9):993-7.

XBiotech Inc.