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Research Published in *Nature Communications* Explores Harnessing Body’s Innate Ability to Prevent Heart Attacks and Stroke

ALBANY, N.Y., Sept. 23, 2016 — A multicenter team of researchers, led by scientists at Albany Medical College, Columbia University Medical Center and Brigham and Women’s Hospital has identified a promising new approach to the treatment of atherosclerosis (clogged arteries), a disease which can lead to heart attacks and strokes.

In their report, published online in *Nature Communications*, the investigators identify a crucial dysfunction in the way immune cells communicate in atherosclerotic plaques. Chemicals released by immune cells that are normally protective in keeping arteries healthy are defective in those with atherosclerosis.

Lead author Gabrielle Fredman, Ph.D., assistant professor in the Department of Molecular and Cellular Physiology at Albany Medical College proposes that these molecules could be used to develop drugs that could prevent complications from the disease.

“Despite the use of cholesterol-lowering drugs and other treatments, cardiovascular diseases remain the leading cause of death in the western world. So while current drugs can be effective, there is much more to understand about heart disease,” said Dr. Fredman.

Her work provides important new insight into the mechanisms behind heart disease and stroke. Specifically, some heart attacks and strokes are caused by rupture of lipid-laden and immune cell-rich plaques within the walls of the arteries. Dr. Fredman discovered that these dangerous plaques in both humans and mice are associated with an imbalance in chemical mediators that promote inflammation versus those that resolve inflammation, known as resolvins. Resolvins are generated by cells within the body to calm inflammation and promote tissue repair.

“The best part of this approach is that it takes advantage of nature’s own design for preventing inflammation-induced damage, which does not compromise host defense and promotes tissue repair,” said co-senior author Ira Tabas, M.D., Ph.D., the Richard J. Stock Professor, Department of Medicine and professor of pathology and cell biology at Columbia. The other co-senior author is Matthew Spite, Ph.D., assistant professor in the Department of Anesthesiology and Center for Experimental Therapeutics and Reperfusion Injury at Brigham and Women’s.
Dr. Fredman found that administration of resolvin to mice prevented the formation of these unstable rupture-prone plaques. This provides a rationale for developing resolvin-based drugs, which could prevent the clinical progression of plaques. In the future, she imagines that such treatments could be used in conjunction with current therapies such as statins.

“There is currently no immunomodulatory drug available to the plaque,” she said.

The paper is titled “An imbalance between specialized pro-resolving lipid mediators and pro-inflammatory leukotrienes promotes instability of atherosclerotic plaques.” The other contributors on the study were: from Albany Med, David M. Jones, M.D.; from Brigham and Women’s, Jason Hellmann, Ph.D., and Romain A. Colas Ph.D.; from Columbia, E. Sander Connolly, M.D., Robert Solomon, M.D., Jonathan D. Proto, Ph.D., George Kuriakose, and Eric J. Heyer, M.D.; and from the University Medical Center of the Johannes Gutenberg University, Bernhard Dorweiler, M.D., Ph.D.

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The study is available at [http://www.nature.com/ncomms/index.html](http://www.nature.com/ncomms/index.html)

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