Bernardo L Trigatti, Biol Syst Open Access 2015, 4:2
http://dx.doi.org/10.4172/2329-6577.S1.002

OMICS International
conferenceseries.com

3rd International Conference on
Integrative Biology

August 04-06, 2015 Valencia, Spain

High density lipoprotein signaling in macrophages and its role in atherosclerosis protection

Bernardo L Trigatti
McMaster University, Canada

Macrophages play key roles in the development of atherosclerosis, a leading cause of coronary heart disease, stroke and peripheral vascular disease. Macrophages participate in all stages of atherosclerotic plaque formation. Lipid laden macrophages accumulate in developing atherosclerotic plaques. Macrophage apoptosis contributes to the development of lipid rich, cell free necrotic cores within atherosclerotic plaques, a key feature of the so-called vulnerable plaque. Vulnerable plaques with large, unstable necrotic cores are prone to rupture resulting in occlusive and potentially devastating athero-thrombosis. High Density Lipoproteins (HDL) protect against the development contribute to the stabilization and also trigger the regression of pre-established atherosclerotic plaques. Recent studies have revealed that HDL, in addition to its well established role in mediating cholesterol removal from atherosclerotic plaques can also act as a signaling molecule in a variety of cell types. Our studies have explored the ability of HDL to trigger signaling events in macrophages activating pro-survival and migratory responses. These contribute to the protection of macrophages against apoptosis in atherosclerotic plaques limiting the formation of necrotic cores as well as the stimulation of macrophage egress from plaques promoting the regression of pre-established atherosclerotic plaques. Identification of the signaling pathways involved in these processes will allow us to identify new therapeutic targets for plaque stabilization and to induce atherosclerotic plaque regression.

Biography

Bernardo L Trigatti has completed his PhD in 1995 at McMaster University and Postdoctoral studies from the Massachusetts Institute of Technology before re-joining McMaster University's Department of Biochemistry and Biomedical Sciences in 2000. He is an Associate Professor at McMaster University and joined the newly formed Thrombosis and Atherosclerosis Research Institute in 2010. He is leading a lab group focusing on uncovering molecular pathways involved in the development of atherosclerosis and associated cardiovascular disease.

Bernardo.Trigatti@taari.ca

Notes: