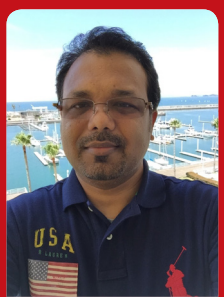
PULSUS
WWW.PULSUSCONFERENCE.COM

Shaik O Rahaman, Curr Res Integr Med 2018, Volume 3
DOI: 10.4172/2529-797X-C1-001

2nd International Conference on
CHRONIC DISEASES
July 16-17, 2018 Berlin, Germany



Shaik O Rahaman

University of Maryland, USA

Atherogenic role of TRPV4 in *P. gingivalis* induced exacerbation of oxidized LDL-mediated macrophage foam cell formation

Epidemiologic studies suggest an association between periodontitis and increased risk of atherosclerosis, a chronic arterial disease responsible for the majority of mortality associated with cardiovascular disease. Epidemiologic studies suggest an association between periodontitis and increased risk of atherosclerosis. *P. gingivalis* (P.g), a predominant causative agent of periodontitis, has been linked to development of atherosclerosis. Emerging data support a role for both a biochemical factor, e.g., Lipopolysaccharides (LPS) and a mechanical factor, e.g., matrix stiffness, in regulation of macrophage function, vascular elasticity, and atherogenesis. In recent, exciting preliminary data, we obtained evidence that TRPV4, an ion channel in the transient receptor potential vanilloid family and a known mechanosensor, may be the mediator of periodontitis-dependent accelerated atherosclerosis. Specifically, we found that: (1) Macrophage TRPV4 activity (Ca^{2+} influx) was increased in response to both *P. gingivalis* LPS (pgLPS) and pathophysiological range matrix stiffness and (2) genetic ablation of TRPV4 blocked pgLPS-induced and matrix stiffness-induced exacerbation of oxidized Low-Density Lipoprotein (oxLDL)-derived macrophage foam cell formation, a critically important process in atherogenesis. Mechanistically, we show that TRPV4 regulates oxLDL uptake but not its cell surface binding in macrophages and plasma membrane co-localization of TRPV4 and CD36 (a receptor for oxLDL) was sensitized to the increasing level of matrix stiffness under pgLPS-treated condition. Altogether, our results suggest that TRPV4 channels play an essential role in P.g-induced exacerbation of macrophage foam cell formation by modulating uptake of oxLDL.

Biography

Shaik O Rahaman is an Assistant Professor at the University of Maryland, USA. His laboratory is interested in elucidating the signaling events underlying the pathogenesis of atherosclerosis and fibrosis. He has received his PhD in Molecular Biology at Jadavpur University and a BS in Human Physiology (Honors) and an MS in Biophysics and Molecular Biology from University of Calcutta. From 2000-2014, he has worked at Cleveland Clinic, Cleveland, USA, as a Postdoctoral Fellow, eventually as a Project Scientist and Assistant Professor. He was the recipient of the American Heart Association Scientist Development Grant, NIH-R01 grant and NSF grant. He is the author or co-author of 23 research papers in high impact international peer-reviewed journals of repute. He has given numerous invited talks nationally and internationally and is a Reviewer/Editorial Board Member in numerous scientific journals. He also served as a Reviewer for National Institute of Health, USA.

srahaman@umd.edu

Notes: