عنوان مقاله:

Gut Microbiota Derived Trimethylamine-N-Oxide (TMAO) Prompts Atherosclerosis Via the Expression of TLR2 and NADPH Oxidase

محل انتشار:

سومین کنگره بین المللی پزشکی شخصی ایران (سال: 1397)

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نویسندگان:

Abbas Ahmadi - *Cellular and Molecular Research Center, Research Institute for Health Development, Department of Molecular Medicine and Genetics, Faculty of Medicine, Kurdistan University of Medical Sciences, Sanandaj, Iran*

Zakaria Vahabzadeh - *Liver and Digestive Research Center, Department of Clinical Biochemistry, Faculty of Medicine, Kurdistan University of Medical Sciences, Sanandaj, Iran*

خلاصه مقاله:

Background: Gut microbiota-dependent metabolites, especially trimethylamine N-oxide (TMAO), have lately been proposed to induce atherosclerosis. However, the exact mechanism is not well understood. Commitment of macrophages toll-like receptors (TLRs) and consequently their downstream NADPH oxidases (NOX) activate the signaling pathways that initiate pro-inflammatory cytokines as well as ROS production. The present study was designed to evaluate the expression of TLR2, TLR6 and NOX2 in macrophages.Methods: Macrophages cell lines (U937 and THP-1) were treated with different concentration of TMAO (37.5, 75, 150 and 300 μM) for 24 h. The cells were also treated with Tunicamycin (TN), as a positive control. RT-qPCR was used to evaluate the expression of TLR2, TLR6, and NOX2 at mRNA levels.Results: Unlike TN, high dose of TMAO significantly increased TLR2 and NOX2 mRNA levels compared to the control cells ($p<0.05$). TN alone significantly increased the mRNA levels of TLR6 ($p=0.010$). Conclusion: Our findings provide positive evidence to support the influence of TLR2 and NOX2 in .proatherogenic mechanism of TMAO during the foam cell formation and abnormal activation of macrophages

کلمات کلیدی:

Trimethylamine-N-Oxide, Atherosclerosis, Toll-like receptors, NADPH oxidase