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Short Communication

Changes in the level of cholesterol in the culture of human monocytes under the influence of various cytokines

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Abstract

Aim: Inflammation is a key factor at all stages of atherosclerosis progression. Macrophages involved in the pathogenesis of atherosclerosis were shown to be activated by pro-inflammatory cytokines that strongly influence atherosclerosis development. Accumulation of modified LDLs by macrophages activates cytokine production by these cells followed by further accumulation of inflammatory cells in the plaque. We have demonstrated how pro-inflammatory cytokines affect macrophage cholesterol and total lipid accumulation.

Methods: THP-1-derived macrophages were co-incubated with modified LDL and interleukins (Il6, Il8, Il12, Il15, Il17A, Il18). Lipid accumulation was evaluated by measuring cholesterol and total protein content in the cells.

Results: Data analysis showed an increase in the accumulation of cholesterol by THP-1 cells during co-incubation with LDL and IL6 ($p < 0.01$), as well as in medium with IL6, but without LDL ($p < 0.01$) compared to the control. Incubation of THP-1 cells with IL8 and LDL revealed an increase in cholesterol. However, upon incubation of cells with IL8 without the addition of LDL, no increase in cholesterol accumulation compared to the control was found ($p > 0.01$). The cultivation of THP-1 cells with IL12 and LDL ($p < 0.01$) led to an increase in cholesterol accumulation compared to the control, a similar result was observed for IL15 with LDL ($p < 0.01$). However, in experiments with IL12 and IL15 in the absence of LDL, no statistical significance was found during lipid accumulation. In a series of experiments with IL17 and IL18, both with the addition of LDL and in its absence, there was no statistically significant difference in cholesterol content compared with the control.

Conclusion: Thus, lipid accumulation by THP-1 cells may be increased by IL6, IL8, IL12 and IL15. In all appearances, pro-inflammatory cytokines can contribute to the accumulation of cholesterol by macrophages during atherosclerosis. This work was supported by the Russian Science Foundation («Institute of General Pathology and Pathophysiology»Grant#19-15-00010).

Biography

Victoria Khotina has completed her Master's degree last year. She is a PhD student and Junior Researcher of the laboratory of angiopathology at the Institute of General Pathology and Pathophysiology, Moscow, Russia. She has 11 publications in Journals, 5 of which are cited in Web of Science and Scopus, and 7 abstracts in conference proceedings, and her publication H-index is 1. She is a participant in 2 research projects supported by Russian Science Foundation Grants.

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