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MICROGRAVITY AND INFLAMMATION: THE EFFECTS ON HUMAN ENDOTHELIAL CELLS

Abstract

Endothelial dysfunction (increased permeability, adhesive properties, secretory activity) occurs not only under the influence of pro-inflammatory agents, but also under the influence of space flight factors. As mechanosensitive cells, endothelium changes the functional state in response of various stimuli (tension, pressure etc.) including microgravity. At the present time the peculiarities of initial response and subsequent adaptation of endothelial cells (ECs) to microgravity, especially the rearrangement of cytoskeleton elements, changes in secretom, adhesive properties and cell-cell interactions, were shown. However, the question what will be the EC response on the joined action of a pro-inflammatory stimulus and microgravity is still opened. We have shown that the simultaneous effect of low doses of TNFa and simulated microgravity modulates the effects of the latter. 3D-clinorotation for 24 hrs impaired the integrity of endothelial monolayer, altered cell morphology, induced cytoskeleton reorganization, and reduced the expression of VE-cadherin. The additional proinflammatory activation revealed the same effects on microfilament and actin cytoskeleton of endothelial cells. The expression of cell adhesion molecules was more significant under pro-inflammatory activation compared with the simulated microgravity. However, as the changes of CAMs (ICAM, VCAM, E-selectin, PECAM-1) on EC surface or its transcription profile were dependent on basal expression level. A similar relationship was observed for the secretion of interleukins after the modification of the EC microenvironment. Thus, the endothelial response to simulated microgravity depends on the initial state of the cells (for example, the preactivation). Strong proinflammatory stimuli (TNFa, ILs) exceeds the simulated microgravity effects but is not additive for many indicators, while the joint action. This study was support by RNF grant N 16-15-10407.