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ACCUMULATION OF THE IMPACT OF TNF-α TO HUMAN UMBILICAL VEIN ENDOTHELIAL CELLS IN THE ACCUMULATION OF CELLULAR OXIDATION PRODUCTS AND THE EXPRESSION OF MCP-1

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Objectives To study the tumour necrosis factor-α (TNF-α), as one of inflammatory factors, how to promote the accumulation of intracellular oxidation products and endothelial dysfunction charactered by overexpression of monocyte chemoattractant protein-1 (MCP-1), especially in senescent endothelial cells.

Methods Senescent human umbilical vein endothelial cells were prepared by continuous sub-culturing in vitro as described. TNF-α was added to the substratum for cell culture of vein endothelial cells (VECs) or not. Reactive oxygen species (ROS) and cell autofluorescence between young and senescent endothelial cells were compared by flow cytometry. The level of MCP-1 messenger RNA (MCP-1 mRNA) was detected by RT-PCR.

Results The generation of ROS, cell autofluorescence and the expression of MCP-1 in VECs were obviously increased after extended serial passage, even though this trend was not obvious in the 8th cell passage. In the 2th cell passage, TNF-α could increase ROS and the intensity of autofluorescence significantly (324.11 ±96.46 vs 676.32±133.73 and 1.35±0.62 vs 3.69±0.95, $p<0.05$) and relative optical density that represents for MCP-1 mRNA increased from 0.81±0.05 in TNF-α group to 0.95±0.11 in control group ($p<0.05$). Similarly, TNF-α displayed the same effects in VECs over two passages.

Conclusions HUVECs will produce more ROS and other metabolic products as it is stimulated by TNF-α, which links the damage and dysfunction of VECs to inflammation and cytokines around intimae. Reinforced expression of MCP-1 is the important mechanism which leads to the damage and dysfunction of VECs.

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