Amelioration of activated macrophage-induced adhesion molecules production in endothelial SEVC cells and endothelial permeability by *Chlorella*-11 peptide

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The inflammatory response in large vessels involves the up-regulation of vascular adhesion molecules such as vascular cell adhesion molecule (VCAM)-1, intercellular adhesion molecule (ICAM-1), and E-selectin. Inflammatory cytokines are thought to play important roles in the development of atherosclerosis. *Chlorella* has been shown to suppress high fat diet-induced atherosclerosis. In addition, *Chlorella*-11 peptide possesses strong anti-inflammatory effect. The aim of this study is to investigate the possible preventing role of *Chlorella*-11 peptide on pro-inflammatory cytokine-induced expression of vascular adhesion molecules and vascular permeability. Endothelial SEVC cells were treated with conditioned culture media (normal culture media contains 50% of LPS-activated macrophage culture media, in which contained TNF-α and IL-6) with and without high (0.038 mM) or low (0.009 mM) concentration of *Chlorella*-11 peptide. Indomethacin (0.25 M) was used as a positive control. Productions of VCAM-1, ICAM-1 and E-selectin, endothelin-1 gene expression and cell permeability were monitored. Productions of ICAM-1, VCAM and E-selectin were all increased by the conditioned culture media. The induction of E-selectin and ICAM-1 was significantly prevented by both concentrations of *Chlorella*-11 peptide. The induced VCAM production and endothelin-1 gene were only suppressed in high dose of *Chlorella*-11 peptide treated cells. Indomethacin was only effectively in preventing the conditioned culture media-induced ICAM production. Cell permeability was also increased in the presence of conditioned culture media. The increased permeability was inhibited by *Chlorella*-11 peptide in a dose-dependent manner. These data indicate that *Chlorella*-11 peptide can be a potential material in preventing chronic inflammatory-related vascular diseases.

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