Lowering high glucose-induced adhesion molecules by lipophilic extract of Chlorella in SEVC cells

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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 and inflammatory cytokines such as TNF-α, IL-1, or IL-6. Chronic hyperglycemia is now recognized as a major etiological factor causing both micro- and macrovascular lesions associated with diabetes mellitus. One of the possible cellular mechanisms by which hyperglycemia damages the vasculature is via an up-regulation of cell-surface adhesion molecule expression by endothelial cells. In addition, endothelin-1 (Et-1) has been implicated in regulation of vascular tonus and progression of atherosclerosis. We have previously shown that Lipophilic Chlorella Extract (LCE) possess strong anti-inflammatory effect. The aim of this study is to investigate the possible role of LCE in preventing high glucose-induced expression of vascular adhesion molecules.

Endothelial cells (SEVC cell line) were pretreated with 25 mM glucose for 24 h prior to exposing to a conditioned culture media (normal culture media contains 50% of LPS-activated macrophage culture media), in which there contained various pro-inflammatory cytokines. VCAM-1, ICAM-1 and E-selectin were measured by ELISA assay kits. Et-1 gene expression was also evaluated by PCR.

High glucose alone did not induce production of ICAM, VCAM or E-selectin, whereas the expression of endothelin-1 was induced by glucose. However, all of these parameters were much higher in conditioned culture media-treated endothelium cells and all were prevented when cells were treated with 0.5 mg/ml of LCE.

We have shown that high glucose alone cannot induce expression of adhesion molecules or indothelin-1. However, co-existence of pro-inflammatory cytokines their expressions are then increased. Our crude compound (LCE) shows a possible potential candidate for preventing development of atherosclerosis by lowering expression of adhesion molecules and endothelin-1 in normal and in high glucose condition.