BENEFICIAL EFFECTS OF *CHLORELLA*-11 PEPTIDE ON BLOCKING LPS-INDUCED MACROPHAGE ACTIVATION AND ALLEVIATING THERMAL INJURY-INDUCED INFLAMMATION IN RATS

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***Chlorella* possesses various remarkable biological activities. One component, Val-Glu-Cys-Tyr-Gly-Pro-Asn-Arg-Pro-Gln-Phe (*Chlorella*-11 peptide) was found to be able to suppress LPS-induced NO production and inflammation. However, the molecular mechanism behind these findings and the consistency between *in vitro* and *in vivo* data have not been investigated. LPS-activated RAW 264.7 macrophages were used to study *in vitro* molecular anti-inflammatory effects of *Chlorella*-11 peptide. After activation, NO production and the expression of iNOS and NF-κB proteins as well as iNOS mRNA were measured using Griess colorimetric assay, Western blotting and RT-PCR, respectively. Alterations in PGE2 and TNF-α contents were also monitored by ELISA. For *in vivo* studies, thermal injury Wistar rats were used and inflammatory indications e.g. serum malondialdehyde (MDA), TNF-α levels and skin erythema were evaluated 48 h after injury implementation. *In vitro* results showed that *Chlorella*-11 peptide produced a dose- and time-dependent inhibition on NO production. The effective inhibition could remain for at least 6 h after LPS activation. It was also found that the expression of LPS-induced iNOS mRNA, iNOS and NF-κB proteins were diminished by the peptide treatment. Concurrently, the levels on TNF-α and PGE2 production after LPS activation were also inhibited. These findings are in agreement with the *in vivo* data that animal serum MDA and TNF-α levels and skin erythema in rats were considerably reduced compared to the control group (saline-treated). The significance of this study sheds light on the effectiveness of *Chlorella*-11 peptide in preventing inflammation progression *in vitro* and *in vivo* and its potential for clinical applications.**

Inflammatory effects of prostaglandins (PGs) are derived from arachidonic acid through the action of cyclooxygenase 2 (COX2). Nitric oxide (NO), derived from L-arginine via inducible NOS (iNOS), is also one of the important mediators of acute and chronic inflammation (1). Excessive NO production can cause detrimental effects to individuals and mediate chronic inflammation (2). Expressions of iNOS and COX2 proteins can be induced by activation of transcription factor of NF-κB (3). NF-

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