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# Inhibitory Effect of CBPharm-001 on Inflammatory Response in UVB-Irradiated Human Fibroblast Cells and Lipopolysaccharide-Induced Raw 264.7 Cells.

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Exposure to ultraviolet not only causes photoaging but also leads to acute inflammatory reaction. Histological analysis of photodamaged skin shows slight epidermal changes but major modifications are observed in the dermis including alterations of the extracellular matrix (ECM), which is predominantly composed of type I and type III collagens. These observations indicate that UV radiation induces changes in ECM proteins and suggest that matrix - metalloproteinases participate in this process. Transforming growth factor-β(TGF-β)is multifunctional cytokines. TGF-βs play important roles in cellular differentiation and biosynthesis of extra cellular matrix. Also, exposure to LPS stimulates cellular inflammatory responses, and releases several inflammatory mediators. Activation of inducible nitric oxide synthase (iNOS) catalyzes the formation of large amount of nitric oxide (NO), which plays a key role in the pathogenesis. The purpose of this study was to investigate the effects of CBPharm-001, an antioxidant single compound isolated a sudden plant, on UV-induced cellular inflammatory response. CBPharm-001 prevented cell damage induced by UVB (40 mJ/㎠) irradiation in dose-dependent manner (0.5-2.5μM) and stimulated TGF-β, procollagen type I expression and inhibited matrix metalloproteinase 3 (MMP-3) in human fibroblast cells. CBPharm-001 also inhibited NO production and iNOS expression in dose-dependent manner but no changed cyclooxigenase (COX-2), IL-1a in LPS-induced murine Raw 264.7 macrophages. CBPharm-001 also inhibited in NF-κB DNA binding in LPS-induced Raw 264.7 cells. These results suggest that CBPharm-001 may contribute to prevention of UVB-induced inflammatory damages in fibroblast cells and inhibition of LPS-induced inflammation in Raw 264.7 cells.

**Keyword:** Inflammation, UV, LPS