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Protective effects skin keratinocyte of *Oenothera biennis* on hydrogen peroxide-induced oxidative stress and cell death via Nrf2/Ho1 pathway.

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Oenothera biennis, commonly known as evening primrose, a potential source of natural bioactive substances: flavonoids, steroids, tannins, fatty acids and terpenoids responsible for a diverse range of pharmacological functions. However, whether extract prepared from aerial part of *O. biennis* (APOB) protects skin against oxidative stress remains unknown. To investigate the protective effects of APOB against oxidative stress-induced cellular damage and elucidated the underlying mechanisms in the HaCaT human skin keratinocytes. Our results revealed that treatment with APOB prior to hydrogen peroxide (H_2O_2) exposure significantly increased viability, and the highest DPPH radical-scavenging activities and reducing power of HaCaT cells. APOB also effectively attenuated H2O2-induced comet tail formation and inhibited the H_2O_2-induced phosphorylation levels of the histone ɣH2AX, as well as the number of apoptotic bodies and Annexin V-positive cells. In addition, APOB exhibited scavenging activity against intracellular reactive oxygen species (ROS) accumulation and restored the mitochondrial membrane potential loss by H_2O_2. Moreover, H_2O_2 enhanced the cleavage of caspase-3 and degradation of poly (ADP-ribose)-polymerase (PARP), a typical substrate protein of activated caspase-3, as well as DNA fragmentation; however, these events were almost totally reversed by pretreatment with APOB. Furthermore, APOB increased the levels of heme oxygenase-1 (HO-1), which is a potent antioxidant enzyme, associated with the induction of nuclear factor-erythroid 2-related factor 2 (Nrf2). According to our data, APOB is able to protect HaCaT cells from H_2O_2-induced DNA damage and cell death through blocking cellular damage related to oxidative stress through a mechanism that would affect ROS elimination and activating the Nrf2/HO-1 signaling pathway.

Key words: *Oenothera biennis*, H_2O_2, oxidative stress, cell death, Nrf2/HO-1

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