Pearl extract protects HaCaT cells from UV radiation-induced apoptosis through mitochondrial pathway regulation

Poster ID

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Introduction:

Human keratinocytes (HaCaT cells) are the main cellular constituent of the epidermis (outermost layer of skin), accounting for more than 90% of epidermal cells [1]. The keratinocyte cells prevent external physical, chemical and microbial damage and maintain the stability of the body's internal environment [2]. As well, they can protect the skin by absorbing 95% of ultraviolet (UV) radiation that reaches the skin [3]. Human keratinocytes participates in various cellular and biological processes, such as apoptosis and inflammation [4]. Damage caused by UV radiation to human epidermal keratinocytes occurs mainly because of the production of reactive oxygen species (ROS) [5], which induce DNA damage, enzyme activity and mitochondrial dysfunction, resulting in damage to various cell functions [6]. To maintain the normal function of human epidermal keratinocytes, the skin can be covered by clothing, which reduces UV radiation exposure and oxidative damage [7]. Additionally, antioxidants are effective in reducing oxidative damage [8]. Jian-min et al. found that the 50% ethanol macroporous resin elution site of Eucommia ulmoides effectively protected against UVA and UVB-induced photoaging in HaCaT cells [9]. Zhiwu et al. reported that rose water inhibited UV-induced apoptosis of HaCaT cells by regulating the nuclear factor-kappa B (NF-κB) nuclear transcription factor pathway [10]. Min et al. demonstrated that hesperidin antagonized the decreased antioxidant enzyme activity in HaCaT cells caused by UVB and showed photoprotective effects [11].

Pearl powder is used as a traditional Chinese medicine to moisturize the heart, liver and muscle [12] and retard skin aging [13]. Anti-inflammation and anti-apoptosis properties have also been described [14,15]. We previously reported that pearl extract (PE) effectively reduced the melanin content in cells by inhibiting the activity of intracellular tyrosinase, suggesting that PE has a whitening effect [16].

The inhibitory effect of PE on UV photodamage-induced HaCaT has not been reported. In this study, an in vitro photoaging cell model was established to evaluate the effect of PE on UV-induced damage and apoptosis of UV-irradiated HaCaT cells and explored the molecular mechanisms involved.

Materials & Methods:

HaCaT cells were cultured in RPMI-1640 medium supplemented with 10% foetal bovine serum and 1% penicillin and streptomycin in 5% CO_2 at 37℃. The cells were irradiated with 10 J/cm² UV, while control cells were sham-irradiated by covering with tin foil. Cell viability was assessed by the CCK8 assay. The cell suspensions were collected and assayed for ROS and MDA levels, and GSH-Px and SOD activities using assay kits in accordance with the manufacturer's instructions. Total RNA was isolated from HaCaT cells after treatment using the RNAiso Plus kit according to the manufacturer's guidelines. After 48 h of the indicated treatment, the supernatant of each group of cells in the 6-well plate was collected. TNF-α and IL-10 were detected in accordance with the enzyme-linked immunosorbent assay kit instructions.

Results & Discussion:

PE concentrations of 0.1 and 1 μg/mL were considered as the most effective and safe concentrations. Compared to the control group, superoxide dismutase and glutathione peroxidase activities in the photoaging group were significantly reduced, while malondialdehyde and reactive oxygen species content, along with tumour necrosis factor-alpha (TNF-α) and interleukin (IL)-10 mRNA and protein levels were markedly increased. In contrast, Bcl-2 protein expression was significantly decreased, while caspase-3, caspase-9 and Bax protein expression levels were significantly increased. Compared to the photoaging group, HaCaT cell proliferation was significantly increased in the PE group. Both PE concentrations significantly increased superoxide dismutase and glutathione peroxidase activities in cells, reduced malondialdehyde and reactive oxygen species content, decreased TNF-α and IL-10 mRNA expression in cells, and reduced TNF-α and IL-10 protein levels in the supernatant. Additionally, Bcl-2 protein expression levels were significantly increased, while caspase-3, caspase-9, and Bax protein expression levels were significantly reduced by PE treatment.

Conclusions:

Our study shows for the first time that PE can reduce HaCaT cell damage caused by UV radiation, which is mainly regulated by reducing ROS and MDA content, increasing the activity of SOD and GSH-Px, inhibiting inflammatory response and mitochondrial mediated apoptosis pathway. The findings indicate that PE can effectively prevent cell damage caused by UV.

Acknowledgements:

Not applicable.

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