P025

Protective Effects and Mechanisms of Baicalin for Keratinocyte HaCaT Cells against UVC-induced Apoptosis

陳思方¹,賴韻仔¹,王守正²,包大靝³⁴,林松水¹

Sue-Fung Chen¹, Yun-Yu Lai¹, Shou-Cheng Wang², Da-Tian Bau^{3,4}, Song-Shei Lin¹

¹Department of Medical Imaging and Radiological Sciences, Central-Taiwan University of Science and Technology,

²Department of Internal Medicine, Division of Pulmonary and Critical Care Medicine, Taichung Armed Forced General Hospital,

³Terry Fox Cancer Research Laboratory, China Medical University Hospital,

⁴Graduate Institute of Clinical Medicine Science, China Medical University

Backgrounds: Long-term exposure to solar ultraviolet (UV) radiation can cause multiple skin disorders including skin cancer. Protection against UV-induced damage is therefore a worldwide concern. Baicalin, a major component of traditional Chinese medicine Scutellaria baicalensis, has been reported to possess antioxidant and cytostatic capacities to normal epithelial and normal peripheral blood and myeloid cells. In this study, we examined whether baicalin could also effectively protect human keratinocytes from the most damaging shortwave UVC irradiation. Materials and Methods: Baicalin scavenged the reactive oxygen species (ROS) elevated within 2 h after UV radiation, and reversed the UV-induced apoptosis. In addition, we identified the major products, cyclobutne pyrimidine dimers, after UV radiation with T4 UV endonuclease, finding that baicalin prevented the cyclobutne pyrimidine dimer formation induced by UV. Furthermore, baicalin could also prevent the oxidative adducts induced by UV. Results: Our results demonstrated the utility of baicalin to assess the contributions of traditional Chinese medicine in UV-induced genomic damage to skin and suggested their potential application as pharmaceutical agents in long-term sun-shining injury prevention. Conclusion: our findings indicated that baicalin had promising UVC-protective capacity via reducing UVC-induced HaCaT cell cytotoxicity, apoptosis, ROS production, CPD and oxidative DNA adduct formation.