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ORIGINAL ARTICLE

Protective Effect of *Viburnum grandiflorum* against Ultraviolet-B Radiation-induced Cellular and Molecular Changes in Human Epidermal Keratinocytes

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ABSTRACT

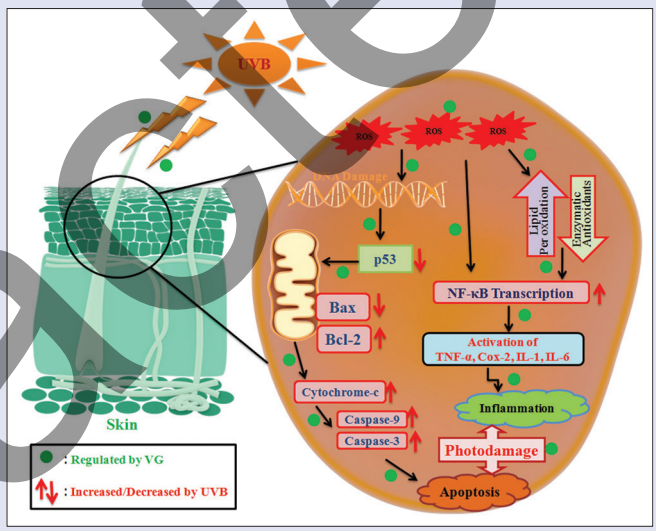
The aim of the present study was to evaluate the photoprotective potential of *Viburnum grandiflorum* (VG) against ultraviolet-B radiation-induced responses in HaCaT cells. The HaCaT cells were pretreated with VG prior ultraviolet-B (UVB)-radiation exposure and were further examined for lipid peroxidation, enzymatic antioxidant activity, % reactive oxygen species, DNA damage, mitochondrial membrane potential and for inflammatory, and apoptotic signaling markers such as tumor necrosis factor-alpha, nuclear factor kappa B, interleukin-1 (IL-1), IL-6, cyclooxygenase-2, p53, caspase-3/9, cytochrome-c, Bax, and Bcl-2. The VG pretreatment in UVB exposed cells shows significantly regulated both inflammatory as well as apoptotic signaling cascades. Our findings suggest that VG may be functional against UVB-induced photo-damages.

Key words: Apoptosis, inflammation, mitochondrial membrane potential, ultraviolet-B radiation, *Viburnum grandiflorum*

SUMMARY

- *Viburnum grandiflorum* (VG) is used as a diuretic, antispasmodic, and anti-sedative; it protects the liver and acts as anti-inflammatory medicine in traditional medicine. Nevertheless, the effect of VG on radiation-induced cellular damages in HaCaT has been explored against ultraviolet-B (UVB) encouraged photo-damages. The observation illustrated that VG offers protection against UVB-induced photo-damages by reducing the oxidative stress, modulation of lipid peroxidation, restoring the mitochondrial membrane potential, and regulating the inflammatory and apoptotic signaling cascades in skin epidermal cells. The findings suggest that VG might be the promising functional agent against UVB-induced photo-damages.

Abbreviations used: VG: *Viburnum grandiflorum*; UVB: Ultraviolet-B radiation; TBST: Tris-buffered saline (TBS) and Tween-20; EDTA: Ethylenediaminetetra acetic acid; Rh-123: Rodamine123; FBS: Fetal bovine serum; PBS: Phosphate-buffered saline; DMSO: Dimethyl sulfoxide; DMEM: Dulbecco's Modified Eagle Medium; ROS: Reactive oxygen species; AO: Acridine Orange; EtBr: Ethidium bromide; MTT: 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide, IC_{50}: The half-maximal inhibitory concentration; MED: Minimal erythema dose; MAPK: Mitogen-activated protein kinases; COX-2: Cyclooxygenase-2; DCFHDH: 2-7-diacetyl dichlorofluorescein diacetate; PMS: Phenazine methosulfate; DTNB: 5, 5-dithiobis 2-nitrobenzoic acid; TBARS: Thiobarbituric acid.



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INTRODUCTION

Ultraviolet radiation comes from the sunlight contains of three main components such as UVA (400-320 nm), ultraviolet-B (UVB) (320-280 nm), and UVC (280-100 nm). Among these three components, UVB is the most destructive module of sunlight, reaching the earth's surface. UVB has both, direct as well as indirect biological effects, including reactive oxygen species (ROS) production, DNA damage, oxidative imbalance, resulting in photo-aging, erythema, and inflammation.[1] UVB radiation leads to the induction of transitions (C to T) at dipyrimidine sites, resulting in the formation of typical photoproducts which are associated with DNA damage.[2] In a day's time, a person can receive 15 minimal erythema doses (MEDs) of UVB. Epidemiological studies on fair-skinned population have stated the incidence of erythema merely following 20 min of sunlight

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