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評估山苦瓜果實和葉片萃取物減緩 UVB 誘導角質細胞株傷害的潛力

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Evaluation of protective effects of wild bitter melon fruit and leaf extracts on UVB-irradiated human HaCaT keratinocytes

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Abstract Hualien No.1 wild bitter melon (WBM; *Momordica charantia* Linn. var. *abbreviata* Ser.) leaf extract has been shown to possess antioxidant activity and could be potentially beneficial for mitigating reactive oxygen species (ROS)-mediated skin disorders. In this study, two crude extracts were respectively prepared from leaf (HL-1L) and fruit (HL-1F) of Hualien No.1 WBM to evaluating their potential to protect against the effects of ultraviolet (UV) B irradiation on human keratinocyte HaCaT cells. Our data showed that HL-1L and HL-1F significantly lowered levels of UVB-induced ROS generation by up to 58%. Both extracts also significantly suppressed UVB-activated mRNA and proteins of cyclooxygenase-2 (COX-2) and matrix metalloproteinase-1 (MMP-1). Our findings suggested that both leaf and fruit extracts of WBM could have the potential to protect keratinocytes from UVB damage.

Key words: ultraviolet B (UVB); wild bitter melon; anti-inflammatory

INTRODUCTION

The human skin in the body's primary defense organ against various pathogens and environmental stimuli, including ultraviolet B (UVB) radiation. In addition to its role as a physiological barrier, skin also has various enzymatic and small-molecule non-enzymatic antioxidants to reduce oxidative damage caused by UVB irradiation [(1)]. However, chronic exposure to UVB radiation can cause injury and inflammation in cutaneous tissue, suppress immune system and ultimately lead to non-melanoma skin cancer [(2,3)]. UVB irradiation causes cellular oxidative stress in keratinocytes and skin fibroblasts through over-production of a variety of

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reactive oxygen species (ROS), resulting in lipid peroxidation, DNA mutation and organelle damage [4]. UVB irradiation up-regulates the expression of matrix metalloproteinase (MMP)-1 which can degrade collagen, thus contributing to pre-mature skin aging (photo-aging) and wrinkles. Cyclooxygenase-2 (COX-2) and its product, prostaglandin E_2 (PGE_2), have been associated with UVB-induced signaling to MMP expression. These changes represent a hallmark of aging [5]. Thus, based on the fact of phyto-compounds exerting potent antioxidant and anti-inflammatory properties, developing a strategy-based approach to suppress oxidative stress and inflammatory responses might be applicable to alleviate UVB-stimulated inflammatory skin diseases.

Wild bitter melon (*Momordica charantia* L. var. *abbreviata* Ser.; WBM) is a nutrient-dense vegetable that is also used to treat conditions such as hyperglycemia, inflammatory responses and oxidative stress [6-8]. Previously, we reported that methanolic extract of WBM leaf exerted the antioxidant and cell-protective activities in UVB-irradiated HaCaT keratinocytes, and also suppressed tyrosinase activity and melanin levels in B16-F10 melanocytes [9]. Since extracts of WBM fruit and leaf significantly relieved inflammatory responses stimulated by lipopolysaccharides (LPS), bacteria (*Cutibacterium acnes*) or chemicals (dextran sulphate sodium) [7,10,11], we were greatly interested in determining whether the fruit extract of WBM could also suppress UVB-irradiated inflammatory responses in keratinocytes in this study.

MATERIALS AND METHODS

Materials

2',7'-dichlorodihydrofluorescein diacetate (H2DCF-DA), 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT), dimethylsulfoxide (DMSO), and sodium dodecyl sulfate (SDS) were obtained from Sigma Chemical Co. (St. Louis, MO, USA). Fetal bovine serum (FBS), phosphate-buffered saline (PBS), Dulbecco's modified Eagle's medium (DMEM), and antibiotic-antimycotic were purchased from Gibco (Carlsbad, CA, USA). All reagent-grade organic solvents were from Burdick and Jackson (Muskegon, MI, USA).

Preparation of WBM extracts

Fresh leaves and fruits of the WBM cultivar Hualien No. 1 (HL-1) were was kindly provided by Mr. Jong-Ho Chyuan, the Hualien District Agricultural Research and Extension Station (Hualien, Taiwan). A voucher specimen has been deposited in the Program of Nutrition Science, National Taiwan Normal University. The leaves were washed, air-dried, ground to a fine powder, and then extracted with methanol (1:20; w/v) at room temperature for 24 h, twice. The fruits were freeze-dried, finely ground and extracted with ethyl acetate (EA) (1:20; w/v) at room temperature for 24 h, twice. Both filtrates were collected and evaporated to dryness, respectively. The yields of HL-1 leaf methnaolic extracts (HL-1L), and HL-1 fruit EA extracts (HL-1F) were 22.3%, and 2.5%. Both extracts were reconstituted in DMSO to a concentration of 200 mg/mL for the subsequent experiments, respectively.

Cell culture and UV-B irradiation

Spontaneously immortalized human keratinocyte cell line HaCaT was grown in 100-mm dishes Dulbecco's modified Eagle's medium (DMEM; Gibco Co., NY, USA) supplemented with 10% fetal bovine serum (Gibco) and 1% Antibiotic-Antimycotic (Gibco). Cells were cultured at 37℃ in a humidified atmosphere of 5% CO_2 incubator (Thermo Co., Waltham, MA, USA).

HaCaT cells were maintained until 40-60% confluence and then pretreated with indicated concentrations of WBM extracts or DMSO alone (as vehicle control) for 24 h in culture medium prior to UVB irradiation. Medium was removed and the cells were rinsed twice with phosphate-buffered saline (PBS) and the cells were irradiated with UVB (Bio-Link Crosslinker BLX-E312, Vilber Lourmat, France)) at 30 mJ/ cm^2 with 2 mL of PBS. Immediately after UVB treatment, PBS was then removed and the cells were further incubated in fresh culture medium the cells were maintained with FBS-containing DMEM in the incubator for 30

min (ROS generation), 16 h (mRNA analysis) or 24 h (protein analysis), respectively.

Detection of intracellular ROS generation

The probe 2', 7'-dichlorofluorescein diacetate (H_2 DCF-DA; Sigma) was used to monitor the intracellular ROS generation of HaCaT cells by UVB irradiation. After UVB irradiation, cells were harvested and washed twice with PBS, and then re-suspended in 10 μM H_2 DCFDA at 37℃ for 30 min incubation. Stained cells were washed, re-suspended in 1mL PBS. ROS generation of these cells was determined by flow cytometry (FACscan, Hercules, CA, USA) using 488 nm for excitation and 525 nm for emission. Mean fluorescence intensity (MFI) detected by FLl channel was analyzed using WinMDI2.8 software.

Real-time reverse transcription polymerase chain reaction (PCR) analysis

Total RNA was extracted and isolated from HaCaT cells using TRIzol reagent (Invitrogen; Carlsbad, CA, USA), following the manufacturer's instructions. The cDNA was then reverse transcribed from the isolated RNA with a reaction mixture of oligo dT PCR primers and reverse transcriptase (Promega, Madison, WI, USA), and amplified by real-time qPCR using an iCycler iQ Real-Time detection system (Bio-Rad, Hercules, CA, USA). To amplify respective cDNA, thermal cycling conditions applied for all PCR assays as described elsewhere [10]. Data analysis of the PCR products was performed using iQ™ 5 optical system software (ver. 2.1; Bio-Rad). The gene primers used were as follows: MMP-1 (forward 5'-AGTTTGTGGCTTATGGATTC-3'; reverse 5'-CCTGTCTCTTTCTGTC-

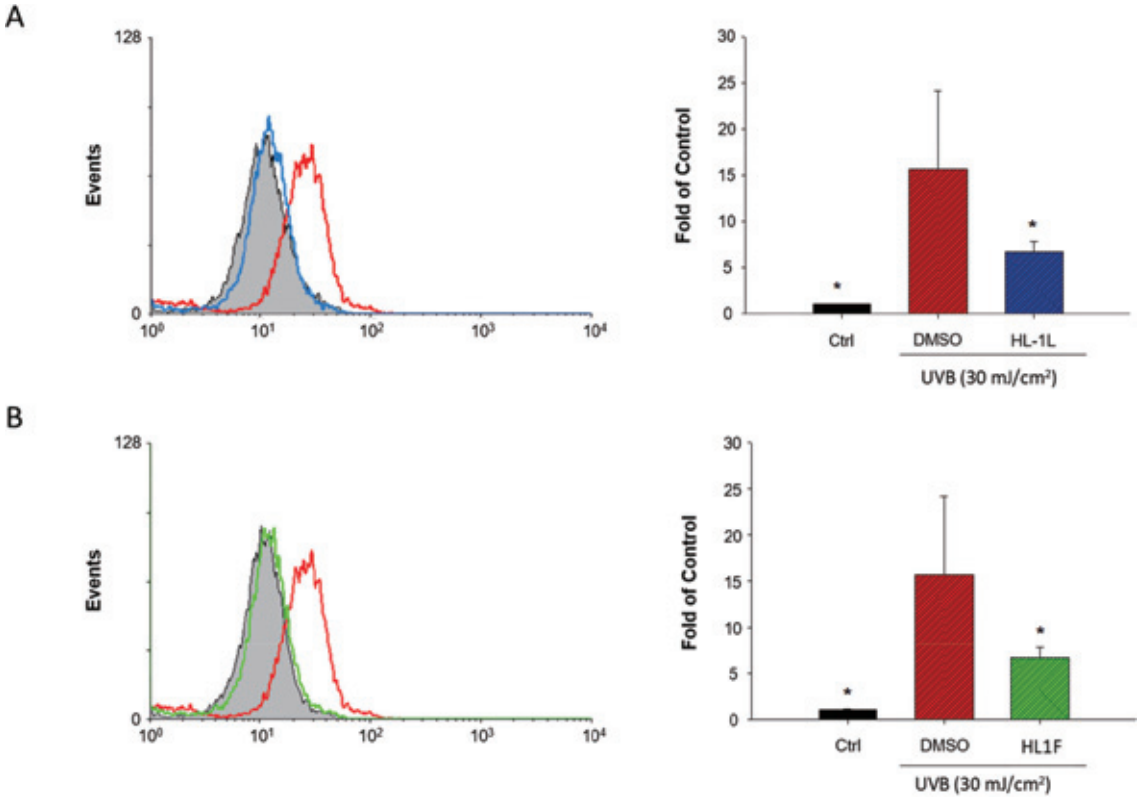

Figure 1. Effects of leaf (HL-1L) and fruit (HL-1F) extracts from wild bitter melons (100 μg/mL) on UVB-induced ROS production in human keratinocytes (HaCaT cells). HaCaT cells (1×10^6 cell/we11) were stimulated with or without UVB-irradiation, and the cells were harvested after 30 min for ROS measurement. The ROS generation was assessed by flow cytometry, using H_2DCF-DA as a probe. Values are means ± SD. * Significant difference compared with the UVB-treated group ($p < 0.05$).

TTGA-3'), COX-2 (forward 5'-TGGTGCCTGGTC-TGATGATGTATGC-3'; reverse 5'-ATCTGCCTGCT-CTGGTCAATGGAAG-3'), and glyceraldehydes 3-phosphate dehydrogenase (GAPDH) (forward 5'-GT-GAAGGTCGGAGTCAACG-3'; reverse 5'-TGAGG-TCAATGAAGGGGTC-3').

Western Blot Analysis

Total protein from HaCaT keratinocytes was extracted, quantified and separated by 10% (w/v) sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE), and subsequently transferred onto a polyvinylidene difluoride (PVDF) membrane (Millipore, MA, USA). The membranes were incubated with a primary antibody specific for MMP-1 (Merck, Darmstadt, Germany), COX-2 (Abcam, Cambridge, MA, USA) and or β-actin (Sigma), followed by reaction with a secondary antibody, immunoglobulin conjugated with horseradish peroxidase (Sigma). The signals of interest proteins were analyzed using the enhanced chemiluminescence (ECL) Western blotting detection system (ChemiDoc XRS, Bio-Rad). Signal strengths were detected and quantified using densitometric program (Image Lab, Bio-Rad).

Statistical analysis

All data were collected and expressed as means ± SD, and performed using the SPSS Statistics for Windows, version 19.0 (SPSS Inc., Chicago, IL, USA). Statistical significance was analyzed using one-way ANOVA followed by LSD multiple range test. Mean differences among groups were considered as statistically significant at the $p < 0.05$ levels.

RESULTS AND DISCUSSION

To determine whether WBM extracts suppressed UVB-activated responses, human HaCaT keratinocytes were incubated with a variety of concentrations (50, 100 or 200 μg/mL) of HL-1L or HL-1F for 24 h, followed by UVB irradiation (30 mJ/cm^2). In our preliminary study, HL-1L (up to 0.31 mg/mL) and HL-1F (up to 0.63 mg/mL) had no adverse effect on cell proliferation in human HaCaT keratinocytes [12].

Using HaCaT cells and human skin models, Dong and colleagues [13] reported that DNA damage by UVB directly or indirectly by UVB-induced ROS generation might be the major cause resulting in photoaging and wrinkling. We investigated whether WBM extracts inhibited ROS generation in keratinocytes, since stimulating production of ROS by UVB irradiation is one of major factors to induce inflammatory responses [14]. The cell viability of HaCaT cells was not significantly affected by 30-min exposure to UVB irradiation, but it significantly decreased after a 16-hour exposure to UVB irradiation. WBM leaf extract showed a protective effect on the viability of irradiated HaCaT cells [9]. Figure 1 shows that levels of ROS were significantly increased in response to UVB irradiation, however, pre-incubation of HaCaT cells with HL-1L or HL-1F suppressed the generation of ROS by up to 58%.

Ample evidences demonstrated that excessive amounts of ROS generated from UVB-irradiated keratinocytes initiated cellular inflammatory responses by activating downstream receptor-mediated signaling cascades [14, 15]. Increasing levels of ROS induced by UVB trigger phosphorylation and activation of signaling intermediates leading to activation of transcription factor activator protein 1 (AP-1) which then up-regulates MMP-1 gene expression [13-15]. Our results showed that the mRNA levels of MMP-1 and COX-2 were increased in response to UVB irradiation, respectively, as compared to the untreated control (Fig. 2). Incubation of cells with HL-1L or HL-1F prior to UVB exposure significantly suppressed expression of MMP-1 up to 85% or 73% at transcription level (Fig. 2). Similarly, levels of COX-2 mRNA were reduced up to 7% or 12% by HL-1L or HL-1F, respectively (Fig. 2). Result of Figure 3 also demonstrated that two inflammatory proteins, MMP-1 and COX-2, were over-expressed at translational level when cells were exposed to UVB radiation. However, HL-1L significantly suppressed MMP-1 and COX-2 expression by up to 56% and 60%, respectively. When cells were pre-treated with HL-1F, the levels of these two proteins were reduced by up to 56% and 31% (Fig. 3).

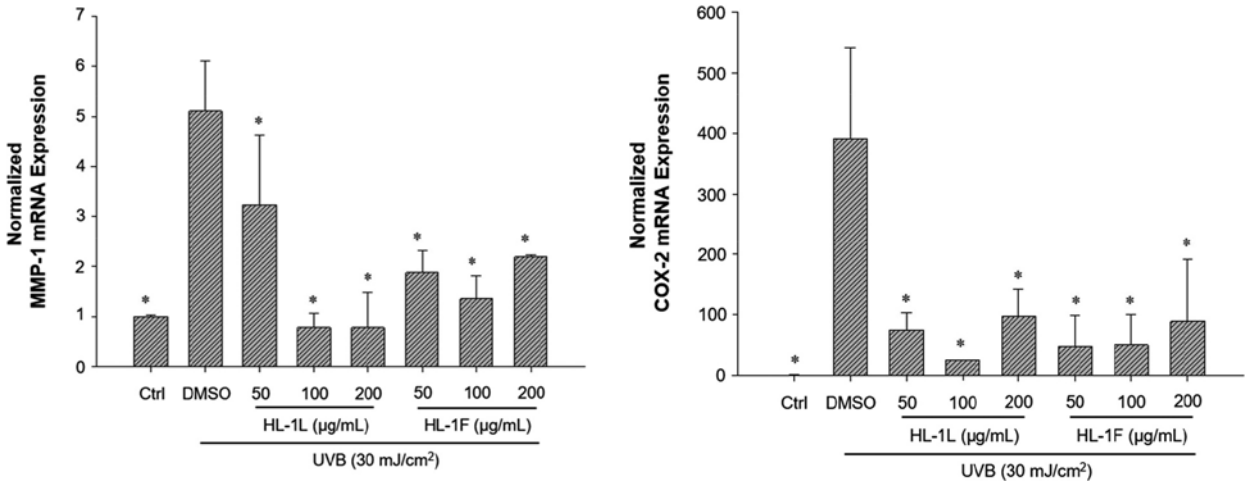

Figure 2. Effects of leaf (HL-1L) and fruit (HL-1F) extracts from wild bitter melons on UVB-induced MMP-1 and COX-2 mRNA expressions in human HaCaT karatinocytes. After treatment with extracts for 16 h, cells were mock treated or irradiated with UVB (30 mJ/cm^2). The cells were further incubated for 16 h. Amounts of MMP-1 and COX-2 mRNA were quantified using GADPH as an internal control and are expressed relative to those control. Values are means ± SD. * Significant difference compared with the UVB-treated group ($p < 0.05$).

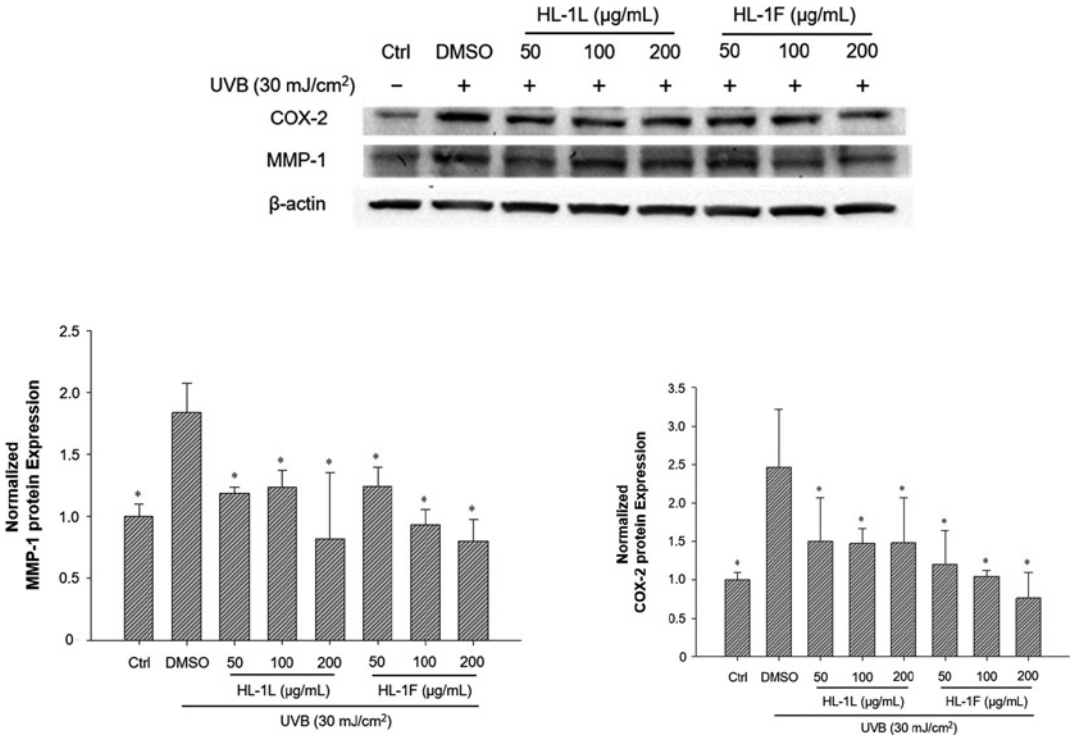

Figure 3. Effects of leaf (HL-1L) and fruit (HL-1F) extracts from wild bitter melons on UVB-induced MMP-1 and COX-2 protein levels in human HaCaT karatinocytes. After treatment with extracts for 16 h, cells were mock treated or irradiated with UVB (30 mJ/cm^2). The cells were further incubated for 24 h. Protein amounts of MMP-1 and COX-2 were quantified using beta-actin as an internal control and are expressed relative to those control. Values are means ± SD. * Significant difference compared with the UVB-treated group ($p < 0.05$).

MMP-1, known as interstitial collagenase, is a neutral metalloproteinase secreted by epidermal keratinocytes and dermal fibroblasts to preferentially degrade fibrillar-type collagens, including type I and III collagen. This specific collagenase play a key role in initiating collagen fragmentation and reorganization during the process of skin aging [16]. It is well-known that over-expression of MMP-1 was transiently induced by UVB irradiation [17], and levels of over-expressed MMP-1 were also augmented by inflammatory mediators, such as tumor necrosis factor-α (TNF-α), interleukin-1 (IL-1), prostaglandin E_2 (PGE_2), and others in the pathogenesis of disorders [18, 19]. Previously, various WBM extracts were reported to attenuate inflammatory responses stimulated by endotoxins or pathogens by suppressing production of cytokines (TNF-α, IL-1β, IL-6 and IL-8) and over-expressions of COX-2 and inducible nitric oxide synthase (iNOS) [11,20,21]. Thus, the suppressive effect of both HL-1L and HL-1F on gene and protein expression of MMP-1 and COX-2 (Figs. 2 and 3) might be attributed, in part, to their potent anti-inflammatory properties.

In this study, we demonstrated that both WBM extracts significantly inhibited gene expression of MMP-1 and COX-2, and postulated whether certain bioactive compounds in the extracts might play critical roles in suppressing UVB-induced inflammation. Our previous reports showed that numerous bioactive phytochemicals, such as apigenin, gallic acid, lutein, phytol and quercetin were identified from WBM extracts [10, 20]. These compounds, along with other phytochemicals, have been reported to protect against UVB-induced damages through direct absorption, scavenging of reactive oxygen and nitrogen intermediates, and induction of cytoprotective responses to detoxify various oxidants and electrophiles [22]. Our previous study showed that the WBM leaf extract exerted potent free radical-scavenging activities by scavenging 2,2-diphenyl-1-picrylhydrazyl (DPPH), nitric oxide (NO), superoxide and hydroxyl radical [9]. Furthermore, phenolic acids, flavonoids, triterpenoids and other components from WBM extracts exerted potent *in vivo* and *in vitro* anti-inflammatory effect to reduce inflammatory responses stimulated by pathogens [10,20,23,24]. Taken together, bioactive phytochemicals-rich WBM extract suppressed UVB-induced inflammation might be attributed to its anti-oxidative property, which lower ROS generation and its anti-inflammatory potential in modulating cell signaling cascades and downstream inflammatory mediator production.

A limitation of this study and its potential implication is that remains to be shown whether topical application of WBM extracts on human skin is safe and effective. Based on results of previous and current studies, products containing WBM extract may be effective in relieving pathogen- and UVB-stimulated inflammatory responses. However, there are certain safety concerns regarding the numerous unfamiliar components and pigments in WBM extracts. In our preliminary clinical study, no skin allergies or adverse effects, such as scaling, erythema, dryness, burning and itching, were observed on facial skin of 29 adults (23 female and 6 male), after they applied the test cream containing both WBM leaf and fruit extracts (0.02%) for 12 weeks [12]. Thus, these findings suggested that WBM extracts is a safe ingredient for developing convincing topical products. However, future investigations regarding topical administration of WBM extracts with clinical studies to verify photoprotective effects and toxicological evaluation are needed.

In conclusion, our findings showed that fruit and leaf extracts of WBM exerted suppressive effects on UVB-induced inflammation, and that these two extracts can be considered as photo-protective agents for modulating *in vitro* immune responses involved in UVB-irradiated skin inflammation.

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Authors' contributions

Designed and performed experiments, Y.-T.H. and P.-J. T.; statistical analysis, Y.-T.H. and L.-T.C.; validation and data curation, Y.-T.H. and P.-J. T.; writing—original draft preparation, L.-T.C.; writing—review and editing, L.-T.C., Y.-T.H. and P.-J. T.; supervision, P.-J. T.; funding acquisition, P.-J.T. All authors have read and agreed to the published version of the manuscript.

Competing Interests

All authors declare no conflict of interest.

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評估山苦瓜果實和葉片萃取物減緩 UVB 誘導角質細胞株傷害的潛力

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摘要　照射 UVB 會促進皮膚角質細胞之活性氧物質的生成。已知花蓮一號山苦瓜葉片萃取物具有良好的抗氧化活性、降低活性氧物質造成角質細胞傷害的潛力。本研究以 UVB 照射人類角質細胞株 HaCaT 的細胞模式，同時評估花蓮一號山苦瓜果實（HL-1F）和葉片（HL-1L）萃取物減緩 UVB 傷害的潛力。結果發現 HL-1F 和 HL-1L 能有效的抑制 UVB 所誘發之活性氧物質的生成量，也顯著降低 UVB 誘導角質細胞的促發炎性的環氧化合酶-2（cyclooxygenase-2）與基質金屬蛋白酶-1（matrix metalloproteases）之 mRNA 和蛋白質表現量，因此推測山苦瓜果實和葉片萃取物均具有保護角質細胞抵抗 UVB 傷害的潛力。

關鍵字：UVB、山苦瓜、抗發炎

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