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Short Communication

Protective Effects of Standardized Peucedanum Japonicum Thunb. Extract Against UVB-Induced Corneal Epithelial Apoptosis via Mitochondrial Regulation

Hye-Sun Lim[1], Young-Sik Yoo[2,3], Gunhyuk Park[1,*,†], Sunoh Kim[4,*,†]

[1]Herbal Medicine Resources Research Center, Korea Institute of Oriental Medicine, 58245 Naju-si, Republic of Korea
[2]IU Eye Clinic, Ophthalmology Practice, 05606 Seoul, Republic of Korea
[3]Mediverse Co., Ltd., 06616 Seoul, Republic of Korea
[4]Central R&D Center, B&Tech Co., Ltd., 58205 Naju, Republic of Korea
*Correspondence: gpark@kiom.re.kr (Gunhyuk Park); sunoh@daum.net (Sunoh Kim)
[†]These authors contributed equally.
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Abstract

Background: Ultraviolet B (UVB) irradiation is a major environmental factor causing corneal epithelial cell apoptosis, leading to ocular surface damage and vision impairment. **Objective**: This study aimed to investigate whether the standardized extract of Peucedanum japonicum Thunb. (SBP) protects corneal cells from UVB-induced apoptosis and explore its mitochondrial regulatory mechanisms. **Methods**: Corneal epithelial cells were exposed to UVB irradiation, with or without treatment with SBP extract or its fractions. Nicotinamide adenine dinucleotide dehydrogenase activity, mitochondrial membrane potential, and mitochondrial morphology were assessed, and apoptosis-related proteins were analyzed using a cytokine antibody array kit. *In vivo* mouse models were also used to evaluate corneal damage following UVB exposure. **Results**: The SBP extract, particularly the n-butanol (n-BuOH) fraction, significantly attenuated UVB-induced mitochondrial dysfunction and reduced apoptosis. Treatment restored mitochondrial membrane potential and improved corneal morphology in UVB-exposed mice. Chlorogenic acid, a major active compound, exhibited similar protective effects. The n-BuOH fraction demonstrated protective effects comparable to those of chlorogenic acid. **Conclusions**: SBP protects corneal cells from UVB-induced apoptosis through mitochondrial stabilization, suggesting its potential as a therapeutic agent for ocular surface disorders.

Keywords: peucedanum japonicum; apoptosis; corneal diseases; ultraviolet rays; mitochondria

1. Introduction

Ultraviolet B (UVB) radiation is among the most harmful environmental eye stressors [1,2]. Cornea is the most anterior and directly exposed tissue and acts as the primary barrier against ultraviolet light; nonetheless, it is also highly vulnerable to phototoxic damage [1]. Repeated or acute UVB exposure results in excessive reactive oxygen species production, mitochondrial dysfunction, and apoptotic pathway activation in corneal epithelial cells, potentially leading to the loss of epithelial cells, corneal opacity, and impaired vision [1]. Clinically, UV-induced keratitis and progressive ocular surface disorders are significant public health concerns, especially in populations with high sunlight exposure or occupational risks [2]. Nevertheless, effective therapeutic strategies for preventing or attenuating UVB-induced corneal apoptosis remain limited.

Previous approaches have largely focused on antioxidant supplementation or topical formulations using well-known compounds, such as vitamin C, vitamin E, or plant-derived flavonoids [3]. Although these agents exhibit some efficacy, their clinical utility is often restricted by their instability, limited bioavailability, and insufficient potency to counteract severe oxidative stress [3]. Therefore, identifying natural compounds with potent antioxidant activity, reliable safety, and strong cellular protective effects is warranted in ophthalmic research.

Peucedanum japonicum Thunb. (SBP) is a medicinal herb widely used in East Asian herbal pharmacology [4]. Historically, it has been used for its antipyretic, analgesic, and anti-inflammatory properties [5]. Phytochemical analyses revealed that SBP contains coumarins, chromones, and phenolic acids that exhibit strong antioxidant capacities and free radical scavenging properties [4,6]. Recent pharmacological studies have suggested that SBP extract protects against inflammation-induced tissue injury, regulates immune responses, and modulates oxidative stress in different experimental models [5,6]. Our previous reports demonstrated that it protected ocular tissues from urban particulate matter (UPM)-mediated oxidative stress and mitigated delayed wound healing following blue light irradiation [7,8]. These studies establish SBP as a potential therapeutic agent for environmentally induced ocular surface damage.

However, its role in preventing UVB-induced corneal epithelial apoptosis, which is mechanistically distinct from

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UPM- or blue light-mediated oxidative injury, has not yet been systematically addressed. Because mitochondrial dysfunction is a central driver of UVB-induced apoptosis, we assessed whether SBP fractions, particularly the n-butanol (n-BuOH) fraction, could stabilize mitochondrial membrane potential, preserve mitochondrial morphology, and suppress apoptotic signaling in corneal epithelial cells. Additionally, chlorogenic acid, previously identified as a major bioactive component of SBP, was tested *in vivo* as a reference compound to validate its protective effects.

2. Materials and Methods

2.1 Preparation of Standardized SBP Extract and Fractions

Dried roots of SBP were provided by B&Tech Co., Ltd. (Naju, Republic of Korea), and a voucher specimen (No. KIOM-SBP or B&T-PJE) was deposited in the herbarium of Korea Institute of Oriental Medicine (Daejeon, Republic of Korea). The phytochemical profile of this SBP extract, including all fractions, was previously analyzed and standardized using HPLC in our earlier publication [7,8]. The same source material was used here.

2.2 Cell Culture and Treatments

Human corneal epithelial cells were obtained from Dr. Sunoh Kim, Central R&D Center, B&Tech Co., Ltd., 58205 Naju, Republic of Korea and kept in Dulbecco's modified Eagle medium supplemented with 10% heat-inactivated fetal bovine serum and 1% penicillin/streptomycin in an atmosphere of 95% air and 5% CO_2 at 37 °C. The cells were seeded in 96-well plates and treated with SBP or its fraction for 24 h; otherwise, they were pretreated with SBP or its fraction for 1 h, subsequently stimulated with 150 mJ/cm^2 UVB for an additional 23 h. All the human corneal epithelial cells used in this study were validated by short tandem repeat (STR) profiling and tested negative for mycoplasma contamination to ensure authenticity and quality.

2.3 Measurement of Nicotinamide Adenine Dinucleotide Dehydrogenase Activity and Apoptosis Signaling Profiling

Nicotinamide-adenine dinucleotide (NADH) dehydrogenase activity was assessed using cell counting kit-8 (Dojindo, Kumamoto, Japan) following the instructions of the manufacturer. For cytokine profiling, apoptosis-related proteins were analyzed using a multiplex cytokine antibody array kit according to the protocol of the supplier. Quantitative analyses were completed within 1 month of treatment.

2.4 Measurement of Mitochondrial Morphology and Membrane Potential ($\Delta\psi m$)

Corneal epithelial cells were cultured on coverslips in 24-well plates and treated with the indicated concentrations of SBP extract under UVB exposure. For mitochondrial morphology analysis, cells were fixed with 4% paraformaldehyde, rinsed with PBS, and stained with MitoTracker (1:1000, Thermo Fisher Scientific, Waltham, MA, USA) for 15 min at room temperature. For the $\Delta\psi m$ assessment, cells were incubated with JC-1 dye at 37 °C for 30 min. After washing with PBS, samples were mounted and visualized under a fluorescence microscope (Olympus BX51, Olympus Corporation, Tokyo, Japan). The fluorescence intensity was quantified using a microplate reader (SpectraMax Gemini EM, Molecular Devices, San Jose, CA, USA). All experiments were independently performed at least three times.

2.5 Animal Study Design and Outcome Measurements

Male C57BL/6 mice (8-week-old, 23–25 g; Doo Yeol Biotech, Seoul, Korea) were housed under controlled temperature (20–23 °C) and light cycles (12-h light–dark) with free access to food and water. All animal procedures were approved by the Institutional Animal Care and Use Committee of the Korea Institute of Oriental Medicine (KIOM: #25-010) and performed in accordance with established guidelines. The mice were randomly divided into six groups (n = 5/group): control, UVB, UVB + SBP 100 mg/kg, UVB + SBP 200 mg/kg, UVB + n-BuOH fraction 50 mg/kg, and UVB + chlorogenic acid 10 mg/kg. The SBP, n-BuOH fractions, and chlorogenic acid were administered orally once daily for 14 consecutive days. On day 15, mice were subjected to a single UVB exposure (375–400 mJ/cm^2 for 15 min) using TL20W/12RS UVB lamps (Philips, Eindhoven, Netherlands) equipped with ultraviolet C cut filters. The difference in UVB irradiation doses between the *in vitro* (150 mJ/cm^2) and *in vivo* (375–400 mJ/cm^2) settings reflects the distinct physical properties of each model. The *in vivo* cornea is a multi-layered and curved tissue covered by the tear film, which scatters and partially reflects UVB light; therefore, a higher dose is required to achieve comparable physiological stress. UVB intensity was calibrated before each experiment using a UV radiometer (E202850, Analytik Jena, Upland, CA, USA) to ensure uniform exposure. The irradiation distance was maintained at 20 cm, and radiation uniformity across the exposure field was verified prior to each experiment. To minimize animal suffering, all procedures involving UVB irradiation and tissue collection were performed under anesthesia. Mice were anesthetized with a mixture of Alfaxan (alfaxalone, Dechra Veterinary Products, North Kansas City, MO, USA) and Rompun (xylazine, Bayer Animal Health, Shawnee Mission, KS, USA) at a 3:1 ratio (alfaxalone 30 mg/kg + xylazine 10 mg/kg, intraperitoneal injection) prior to UVB exposure and before tissue collection. For euthanasia, mice received a terminal overdose (alfaxalone 60 mg/kg + xylazine 20 mg/kg, intraperitoneal injection) of the same anesthetic mixture followed by cervical dislocation to ensure death, as approved in the institutional protocol. Corneal damage was assessed by fluorescein staining on day 2 after UVB exposure. Following euthanasia, ocular tissues were collected for histological analyses, including hematoxylin and eosin (H&E) staining and terminal deoxynucleotidyl transferase dUTP

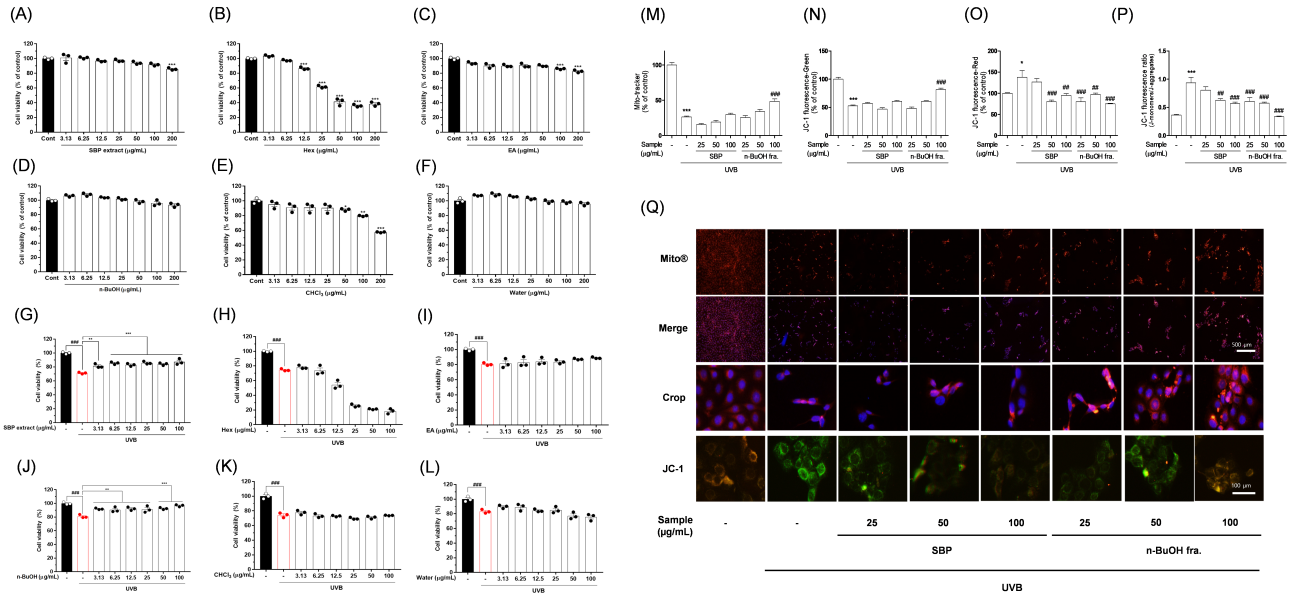

Fig. 1. Protective effects of SBP extract and its solvent fractions against UVB-induced mito-toxicity in corneal epithelial cells. Cell viability after treatment with (A,G) SBP extract at the indicated concentrations, with or without UVB irradiation; (B,H) hexane fraction; (C,I) ethyl acetate fraction; (D,J) n-BuOH fraction; (E,K) chloroform fraction; (F,L) water fraction. (M) Quantitative analysis of mitochondrial morphology and fluorescence intensity. (N–P) Ratio of red to green fluorescence indicating mitochondrial membrane potential ($\Delta\Psi$m). (Q) Representative fluorescence images of corneal epithelial cells stained with JC-1 (red: aggregated, intact mitochondria; green: monomeric, depolarized mitochondria). SBP extract and its n-BuOH fraction significantly restored mitochondrial membrane potential and preserved elongated mitochondrial morphology compared with the UVB-only group. Scale bars: top two rows = 500 μm; bottom two rows = 100 μm. Data are expressed as mean ± SD from at least three independent experiments. $*p < 0.05$, $**p < 0.01$, $***p < 0.001$ versus control; $\#\#p < 0.01$, $\#\#\#p < 0.001$ versus UVB group. UVB, ultraviolet B; SBP, Peucedanum japonicum Thunb; n-BuOH, n-butanol.

nick-end labeling (TUNEL) assays, as previously described (**Supplementary Method**).

2.6 Statistical Analysis

All statistical parameters were calculated using the GraphPad Prism software (version 5.0; GraphPad Software, San Diego, CA, USA). Data are expressed as the mean ± standard error of the mean (SEM) from at least three independent experiments unless otherwise stated. All graphical data are presented as mean ± SEM, which is clearly indicated in each figure legend. Statistical comparisons between different treatments were performed using one-way analysis of variance with Tukey's multiple comparison post hoc test. Statistical significance was set at $p < 0.05$.

3. Results and Discussions

This study demonstrated the anti-apoptotic effect of SBP extract on corneal epithelial cells, thereby identifying a novel natural substance capable of protecting the eye from UV-induced damage [1]. These findings provide an important basis for the development of new therapeutic agents to maintain ocular health. By elucidating apoptosis through mitochondrial damage as the major mechanism, this research reinforces the scientific validity of using SBP as a protective agent. When corneal epithelial cells were exposed to UVB irradiation, cell viability markedly decreased, accompanied by the loss of mitochondrial membrane potential and morphological abnormalities characteristic of apoptosis [1,2]. Moreover, JC-1 staining revealed a pronounced shift from red to green fluorescence in the UVB group, indicating mitochondrial depolarization [9]. To exclude cytotoxicity and identify the bioactive fractions, we screened SBP fractions using an NADH dehydrogenase activity assay. The n-BuOH fraction demonstrated no mitotoxicity, displaying clear protective effects against UVB-induced damage (Fig. 1A–L). Given that the MTT assay primarily reflects mitochondrial activity, SBP may act through mitochondrial regulation. Subsequent analyses with JC-1 and MitoTracker confirmed this hypothesis [10]. SBP treatment restored the red/green fluorescence ratio in a dose-dependent manner, with the n-BuOH fraction exhibiting the most potent protective effect (Fig. 1M–Q). Furthermore, morphological imaging with MitoTracker revealed that the mitochondria in UVB-exposed cells were fragmented and punctate, reflecting oxidative stress and apoptotic signaling initiation. Conversely, SBP-treated cells maintained elongated tubular mitochondria, indicating mitochondrial integrity preservation (Fig. 1M–Q). Overall, SBP may pre-

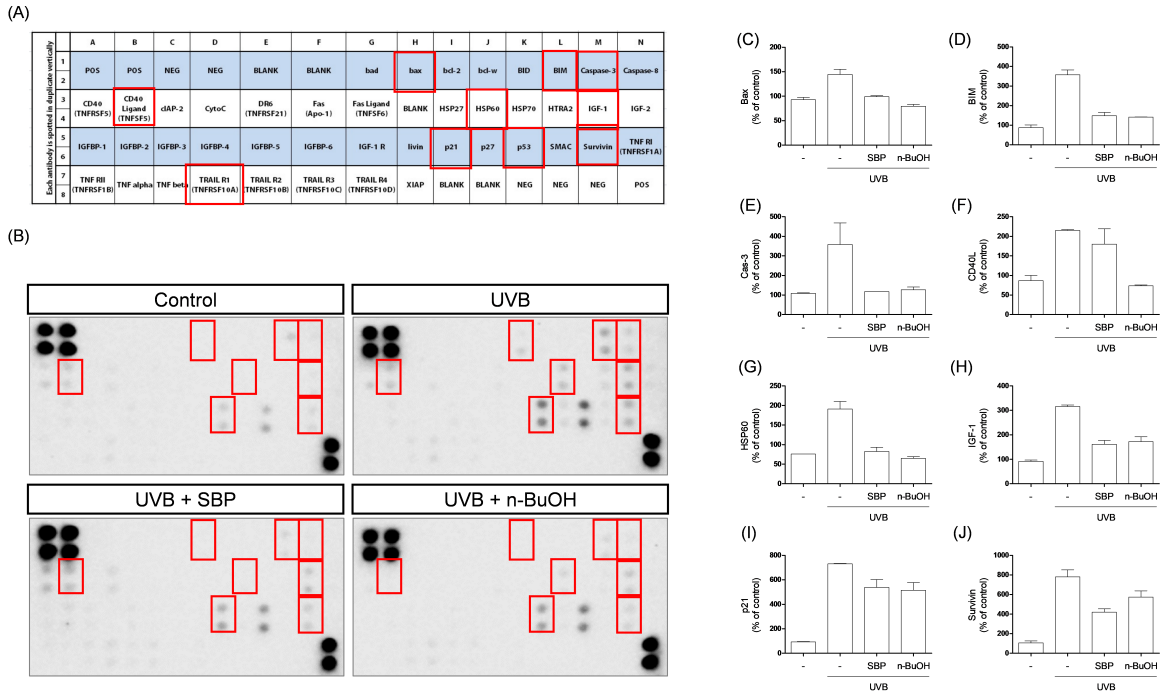

Fig. 2. Effects of SBP extract and its n-BuOH fraction on UVB-induced apoptosis-related protein expression in corneal epithelial cells. (A) Layout of the apoptosis antibody array showing the positions of 43 apoptosis-related proteins, with red boxes marking protein spots that exhibited notable expression changes among the groups. (B) Representative protein array blots from each group: control, UVB, UVB + SBP, and UVB + n-BuOH fraction. Red boxes indicate protein spots that showed detectable and significant expression changes between groups. Quantification of (C) Bax expression levels. (D) Bim expression levels. (E) Caspase-3 expression levels. (F) CD40L expression levels. (G) HSP60 expression levels. (H) IGF-1 expression levels. (I) p21 expression levels. (J) Survivin expression levels. UVB exposure markedly increased pro-apoptotic protein expression, which was attenuated by SBP extract and its n-BuOH fraction. SBP and n-BuOH treatments reduced UVB-induced upregulation of Bax, Bim, Caspase-3, CD40L, HSP60, IGF-1, and p21, while partially restoring Survivin levels. Data are presented as mean ± SD from three independent experiments. UVB, ultraviolet B; SBP, Peucedanum japonicum Thunb; Bax, Bcl-2–associated X protein; Bim, Bcl-2–interacting mediator of cell death; Caspase-3, Cysteine-aspartic protease-3; CD40L, CD40 ligand; IGF-1, Insulin-like growth factor-1; p21, Cyclin-dependent kinase inhibitor 1A (CDKN1A).

vent mitochondrial dysfunction, which is a central trigger of apoptosis. To further strengthen our mechanistic insights, an apoptotic protein array was performed. UVB irradiation increased the expression of pro-apoptotic proteins, including Bcl-2–associated X protein (Bax), Bcl-2–interacting mediator of cell death (Bim), Cysteine-aspartic protease-3 (Caspase-3), CD40 ligand (CD40L), Heat shock protein 60 (HSP60), Insulin-like growth factor-1 (IGF-1), and Cyclin-dependent kinase inhibitor 1A (p21), while the anti-apoptotic protein Survivin was dysregulated (Fig. 2). In particular, the upregulation of Bax and Bim promotes mitochondrial outer membrane permeabilization, leading to cytochrome c release and subsequent activation of Caspase-9 and Caspase-3, a hallmark of the intrinsic (mitochondria-mediated) apoptotic pathway [11]. Additional factors such as CD40L, HSP60, IGF-1, and p21 further reflect stress-induced apoptotic signaling and cross-talk with mitochondrial processes [12]. Dysregulation of Survivin, an inhibitor of apoptosis, further amplifies this pro-apoptotic cascade [12]. Importantly, SBP treatment reversed or attenuated these molecular alterations, restoring the balance between pro- and anti-apoptotic signaling and thereby counteracting UVB-induced mitochondrial apoptosis (Fig. 2). Thus, SBP may protect against UVB-induced corneal apoptosis by maintaining mitochondrial potential and influencing the expression of key regulators of the intrinsic apoptotic cascade. These protective effects were validated *in vivo*. Fluorescein staining of the corneal surfaces revealed that UVB exposure induced severe epithelial damage, characterized by diffuse punctate staining, whereas SBP-treated mice demonstrated markedly reduced fluorescein uptake, indicating preservation of corneal barrier integrity (Fig. 3A). This reduction in staining suggests that SBP protected epithelial tight junctions and maintained barrier function against UVB-induced disruption (Fig. 3A). Given that fluorescein staining is a clinical indicator of ocular surface health, these findings further highlight the translational potential of SBP in preventing UVB-related ocular surface disorders [13]. His-

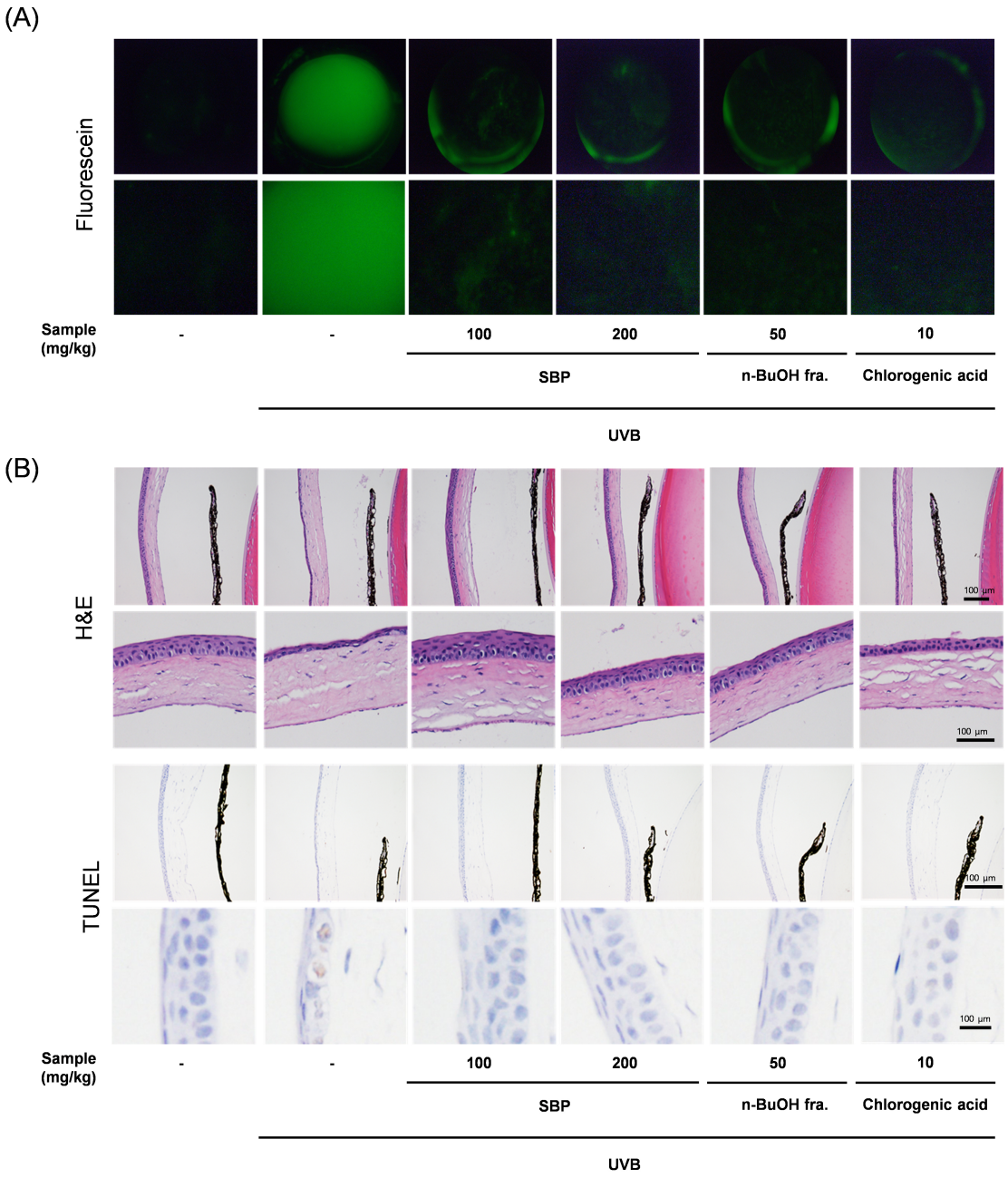

Fig. 3. Protective effects of SBP extract and its n-BuOH fraction against UVB-induced corneal damage in mice. (A) Representative fluorescein staining images of corneal surfaces. UVB exposure induced severe epithelial damage and punctate keratopathy, which were markedly reduced by SBP extract and n-BuOH fraction treatment. Due to the curved nature of the corneal surface and the imaging method used (fluorescence photography), exact spatial calibration was not feasible. Therefore, scale bars were not included. (B) Hematoxylin and eosin staining of corneal tissues. UVB irradiation caused epithelial thinning, disorganization, and stromal edema, whereas SBP-treated groups exhibited preserved epithelial structure and reduced tissue injury. Scale bar = 100 μm. TUNEL assay of corneal sections. Numerous TUNEL-positive nuclei were detected in the UVB group, indicating extensive apoptotic cell death. SBP extract and the n-BuOH fraction significantly decreased the number of TUNEL-positive cells, demonstrating their anti-apoptotic effects *in vivo*. UVB, ultraviolet B; SBP, Peucedanum japonicum Thunb; TUNEL, terminal deoxynucleotidyl transferase dUTP nick-end labeling.

tological examination of the corneal tissues from UVB-irradiated mice revealed epithelium thinning, disorganization of the cell layers, and stromal swelling. Contrastingly, the SBP-treated corneas retained their epithelial structures and displayed reduced tissue damage, particularly in the n-BuOH group. Although the present study did not include a drug-only (without UVB) control group, previous *in vitro* and *in vivo* data have confirmed that SBP and its n-BuOH

fraction are non-toxic to normal cells and tissues [7]. Future studies must include such drug-only groups to further evaluate the ocular safety and baseline effects of SBP under physiological conditions. The TUNEL assay provided additional evidence that numerous TUNEL-positive nuclei were observed in the UVB-exposed corneal epithelium; nonetheless, these apoptotic signals were significantly reduced in the SBP-treated groups (Fig. 3B). Quantitative HPLC analysis from our previous study [7] revealed that it contained high levels of chlorogenic acid (2.30 ± 0.03 mg/g), along with its related isomers, neochlorogenic acid (2.27 ± 0.03 mg/g) and cryptochlorogenic acid (2.57 ± 0.04 mg/g). These data confirm that the n-BuOH fraction is enriched in chlorogenic acid derivatives, which are the principal phenolic components responsible for its antioxidant and anti-apoptotic activities. In line with our previous phytochemical studies, three chlorogenic acid isomers (chlorogenic acid, cryptochlorogenic acid, and neochlorogenic acid) were tested for cytotoxicity *in vitro* (**Supplementary Fig. 1**). Among these, chlorogenic acid was the only compound that showed no detectable mito-toxicity at the tested concentrations, whereas cryptochlorogenic acid and neochlorogenic acid caused dose-dependent reductions in NADH dehydrogenase activity, consistent with their negative effects on cell viability shown in **Supplementary Fig. 2**. Accordingly, chlorogenic acid was selected as the reference compound for *in vivo* validation. Notably, previous studies have also identified chlorogenic acid as a major bioactive constituent of SBP, demonstrating its efficacy in protecting against oxidative stress and cellular injury [7,8]. Consistent with these findings, SBP treatment in the present study exerted protective effects comparable to those of chlorogenic acid, further supporting its potential as a natural therapeutic candidate. Notably, this comparison was based on nominal dose (mg/kg) rather than molar equivalence, as the n-BuOH fraction represents a complex phytochemical mixture enriched in chlorogenic acid and related phenolic compounds. Therefore, the comparable efficacy observed *in vivo* likely reflects a bioactivity-based equivalence rather than an exact molar match.

In summary, this study demonstrated that the SBP extract exerts significant protective effects against UVB-induced corneal epithelial apoptosis, with the n-BuOH fraction showing the strongest efficacy both *in vitro* and *in vivo*. This observed protection appeared to be closely linked to mitochondrial function preservation, suggesting that the active constituents within the n-BuOH fraction may enhance mitochondrial activity and prevent depolarization-induced apoptotic signaling. Based on previous phytochemical analyses and supporting evidence from earlier studies, chlorogenic acid has emerged as a plausible candidate that contributes to this effect. Our previous HPLC profiling confirmed that the n-BuOH fraction contained the highest concentrations of chlorogenic acid and its related derivatives, including neochlorogenic and cryptochlorogenic acids [7]. However, this fraction also contains minor coumarin- and flavonoid-type compounds, which possess antioxidant and anti-apoptotic activities. Therefore, synergistic interactions among multiple constituents may collectively contribute to the overall protective effects of SBP against UVB-induced corneal injury. Nevertheless, further investigations are warranted to confirm whether chlorogenic acid is the major bioactive compound or whether synergistic interactions among multiple n-BuOH fraction components are responsible. Future studies should focus on isolating the active constituents, clarifying mitochondria-related molecular pathways in greater detail, and assessing the long-term safety and clinical applicability in preclinical models. Furthermore, evaluating the ocular bioavailability and tissue distribution of SBP-derived compounds particularly chlorogenic acid and its derivatives will be essential for future translational development. Pharmacokinetic and corneal penetration studies are planned to determine the absorption, distribution, and retention of these compounds within ocular tissues. One limitation of this study is that only male mice were used in the *in vivo* experiments to minimize hormonal variability. However, sex-related physiological differences may affect oxidative stress responses and drug efficacy. Therefore, future studies should include both male and female mice of matching age and weight to comprehensively evaluate potential sex-dependent variations and enhance the translational relevance of our findings.

4. Conclusions

SBP extract, particularly its n-BuOH fraction, effectively protected corneal epithelial cells against UVB-induced apoptosis by preserving mitochondrial function and modulating apoptosis-related proteins. Both *in vitro* and *in vivo* evidence support its potential as a safe natural candidate for preventing or treating UV-related ocular surface disorders. Therefore, SBP is a promising therapeutic target for ophthalmic development. Further studies are warranted to isolate the active compounds and evaluate their long-term efficacy.

Abbreviations

NADH, nicotinamide adenine dinucleotide; n-BuOH, n-butanol; PBS, phosphate-buffered saline; SBP, Peucedanum japonicum Thunb.; SD, standard deviation; TUNEL, terminal deoxynucleotidyl transferase dUTP nick-end labeling; UVB, ultraviolet B; $\Delta\Psi m$, mitochondrial membrane potential.

Availability of Data and Materials

The datasets generated and analyzed during the current study are available from the corresponding author upon reasonable request.

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Author Contributions

Conceptualization, HL, YY, GP, SK; methodology, GP, HL; formal analysis, GP, HL; investigation, GP, SK; data curation, GP, HL; writing—original draft preparation, HL, GP; writing—review and editing, HL, YY, GP, SK; visualization, HL, GP, SK; supervision, GP, SK. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The Institutional Animal Care Committee of the Korea Institute of Oriental Medicine (KIOM) approved the experimental protocols KIOM: #25-010, which were performed according to the guidelines of the Animal Care and Use Committee at KIOM.

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Conflict of Interest

The authors declare no conflict of interest. Young-Sik Yoo is an employee of Mediverse Co., Ltd. Sunoh Kim is an employee of the Central R&D Center of B&Tech Co., Ltd. The authors declare that these affiliations did not influence the study design, data interpretation, or manuscript preparation.

Declaration of AI and AI-Assisted Technologies in the Writing Process

During preparation of this manuscript, the authors used ChatGPT [5.0 version, OpenAI] for language editing and grammar checking. After using this tool, the authors carefully reviewed and revised the text and take full responsibility for the content of the publication.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/FBL46498.

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