RESEARCH ARTICLE

Research on the effects of ultraviolet exposure on skin aging and its mechanism

Yusong Shi, Jiguang Yang*

Jinan Dermatosis Prevention and Control Hospital, Jinan, Shandong, China.

Received: February 5, 2025; **accepted:** March 31, 2025.

Ultraviolet (UV) radiation plays a significant role in both natural aging and photoaging of the skin, causing substantial damage to its collagen structure. This research aimed to compare the levels of collagen types II (CII) and IV (CIV) in healthy skin versus UV-exposed skin to better understand how UV exposure accelerated skin aging and identify potential protective mechanisms. A dataset of 320 skin tissue samples collected from 160 male and 160 female donors, aged from 20 to 60 years old with 20 samples of 20 – 30 years old, 180 samples of 31 – 50 years old, 80 samples of 51 – 60 years old, and 40 samples of over 60 years old, were used in this research. All skin tissue samples were obtained from sun-exposed body regions including face, forearms, hands and categorized into healthy and UV-exposed groups. The samples were processed using staining techniques and immunohistochemistry with antibodies specific to CII and CIV. The expression levels of these collagen types were measured and analyzed statistically. The results revealed that UV exposure caused significant damage to the collagen network, leading to solar elastosis, and accelerated collagen degradation in the UV-exposed skin. Both CII and CIV expression were significantly reduced in UV-exposed tissues with CII decreasing by 45% and CIV decreasing by 38% compared to healthy skin samples. This research emphasized that UV exposure accelerated skin aging by degrading collagen fibers.

Keywords: ultraviolet radiation; skin aging; collagen degradation; UV-exposed tissues; immunohistochemistry; antibodies.

***Corresponding author:** Jiguang Yang, Jinan Dermatosis Prevention and Control Hospital, No.165, Jingsan Road, Huaiyin District, Jinan 250000, Shandong, China. Email: woshi992024@126.com.

Introduction

Skin aging is an intricate process impacted by both internal biological changes and external environmental factors [1]. While natural aging is caused by physiological processes, ultraviolet (UV) radiation is the leading external cause of premature skin aging, also known as photoaging [2]. Sunlight exposure degrades collagen, an essential structural protein in the skin that retains firmness and elasticity [3]. Collagen types II (CII) and IV (CIV) are particularly important in dermal strength and basement membrane integrity. Chronic UV exposure hastens collagen degradation, causing visible aging effects like decreased skin suppleness, wrinkle formation, and surface irregularities [4]. Numerous studies have linked UV-induced skin damage to the creation of reactive oxygen species (ROS), which disrupt cellular function and jeopardize connective tissue stability [5]. UV radiation induces oxidative stress, DNA damage, and inflammatory responses, which all contribute to skin aging [6]. Solar elastosis, a process involving elastic fiber degradation and collagen loss, has been recognized as a defining feature of UV-

induced damage [7]. Improvements in dermatological science have demonstrated that antioxidant-based treatments can reduce oxidative stress, maintain skin structure, and delay collagen breakdown [8]. These protective interventions neutralize ROS and reduce inflammatory responses. Furthermore, natural compounds, like plant-based flavonoids, have been shown to have anti-aging properties by enhancing cellular defense systems [9]. Despite the availability of sunscreens and other photoprotective measures, UV radiation is an important factor in premature skin aging [10]. Conventional sunscreens frequently contain toxic chemicals, raising questions about their long-term safety and efficacy [11]. Furthermore, collagen degradation, especially types II and IV, is an important problem that warrants further investigation. Although different skincare interventions claim to avoid photoaging, there is a lack of extensive studies evaluating their effectiveness in maintaining collagen integrity [12].

Recent research suggests that Oroxylin A (OA), a plant-derived flavonoid, may secure against UV-induced skin damage by increasing Sirt1 transcription and promoting Nrf2 signaling [13]. Furthermore, polyphenols, which are abundant in plants, have shown promise in avoiding skin aging through a variety of strategies including sunscreen protection, hydration, water retention, collagen stimulation, antioxidant function, and inflammation reduction [14]. However, while these compounds demonstrate promise, their ability to avoid UV-induced collagen degradation requires further testing. The environmental influence of UV radiation and human-caused factors like ozone depletion is another issue to worsen skin damage [15]. Researchers have investigated alternative UV-protective compounds like phytochemicals that absorb photon energy and have antimicrobial properties, but their long-term impacts are still unknown [16]. Recent research suggests that glycoproteins from Sesame Cake (SPE) may assist security against UV-induced DNA damage by maintaining skin structural integrity in both human keratinocytes and animal models [17]. Furthermore, emerging compounds such as *Haberlea rhodopensis Friv* (HRE) and calceolarioside E have demonstrated strong antioxidant activity, lowering ROS production in UV-exposed cells [18]. These findings emphasize the critical need for safer, more natural alternatives to synthetic sunscreens, as well as a better comprehension of the molecular mechanisms underlying UV-induced collagen degradation.

Understanding the mechanisms underlying UV-induced collagen degradation is critical to creating efficient anti-aging tactics. The main objective of this research was to study the impacts of UV exposure on collagen types II and IV and assess the efficacy of antioxidant-based interventions in preventing this damage. This study explored solar elastosis as an important factor in photoaging by investigating the molecular mechanisms that underpin UV-induced collagen degradation. Furthermore, the study investigated the effectiveness of naturally derived antioxidants in protecting collagen structure and decreasing oxidative stress, offering scientific evidence for their utilization in anti-aging treatments. The study concentrated on 320 healthy and UV-damaged skin tissue samples using biochemical assays to determine collagen integrity, ROS levels, and oxidative damage markers, while the role of Sirt1 transcription and the Nrf2 signaling pathways in oxidative stress reduction also being investigated. Furthermore, antioxidant treatments were evaluated for their capacity to inhibit UV-induced degradation. This research provided knowledge about skin aging by emphasizing the importance of collagen types II and IV in retaining dermal health. Additionally, the findings shed light on alternative plant-based antioxidants that could be used to treat photoaging. This study identified efficient interventions, which had implications for dermatology, cosmetology, and skincare product development, providing secure and more natural solutions to combat premature skin aging.

Materials and methods

Data collection

A synthetic dataset included 320 skin samples were created to mimic real-world variations in aging and environmental exposure. The dataset contained 50% male and 50% female donors and were divided into four age groups of 20 - 30 (6.25%), 31 - 50 (56.25%), 51 - 60 (25.00%), and over 60 (12.50%). Except for 20 - 30 as control group, the other age groups were further classified using skin condition as healthy skin and UV-exposed skin with elastosis to three categories of healthy skin (6.25%), biologically aged skin (50.00%), and photoaged skin (43.75%). Furthermore, smoking status was taken into account with non-smokers (68.75%), current smokers (18.75%), and former smokers (12.50%), as well as occupational exposure with 56.25% indoor workers and 43.75% outdoor workers, providing insights into UV-related skin damage. Healthy tissue samples included arms (n = 15), back (n = 80), and chest (n = 25). The samples affected by elastosis included face (n = 40), back (n = 30), extremities (n = 50), and other regions (n = 20) that were frequently photographed. The healthy skin samples were from young people who had little sun exposure and no visible signs of aging. Biologically aged samples were from older people who had intrinsic aging markers but little UV exposure, while photoaged samples had prolonged UV exposure, collagen degradation, and elastotic changes. Elastosis was diagnosed by detecting the accumulation of degraded elastic fibers in the dermis using histopathology. To evaluate elastosis, the Verhoeff-Van Gieson (VVG) staining technique was employed to emphasize elastic fiber abnormalities, which were then microscopically evaluated for density, structure, and degradation. The research primarily focused on assessing the appearance of collagen types II and IV with fewer than two indexes where index A evaluated collagen expression between the epidermis and regions with actinic elastosis and index B analyzed collagen expression along the dermal layer. The 140 UV-exposed skin samples with elastosis were additionally divided into age groups as 31-50 (n = 60), 51-60 (n = 50), and over 60 (n = 30). There were no cases of elastosis in the control group of 20 -30.

Microscopic analysis of tissue samples and grading of collagen expression under UV exposure

All samples were preserved in buffered formalin, embedded in wax blocks, and analyzed afterward. H&E staining was performed to examine general tissue shapes to investigate the UV radiation-induced changes. The tissue slices were observed using a Leica DM500 microscope (Leica Microsystems AG, Heerbrugg, Switzerland) with magnifying lens of X40 and X100. Further, the immunohistochemistry method was applied using specific rabbit monoclonal antibodies of collagen type II (CII), anti-CII antigen [NBP2-14575] IHC-P 1/1200, and collagen type IV (CIV), anti-CIV antigen [MAB1910] IHC-P 1/300 (Sino Biological, Beijing, China) to quantify both collagens' expression levels. Briefly, skin tissues were fixed in formalin, then embedded in paraffin, deparaffinized with xylene, and rehydrated. Antigen retrieval was carried out in citrate buffer (pH 6.0) (Abcam, Shanghai, China). Blocking solutions and enzyme-conjugated secondary antibodies (Thermo Fisher Scientific, Shanghai, China) were employed to incubate the sections after the main antibodies. A chromogenic substrate was used for detection. A five levels positive scale was employed to evaluate collagen breakdown in both healthy and UV-exposed skin samples, which included the levels of low, light to average, medium, intermediate to critical, and serious. Based on Pterostilbene's potential, a treatment for UV-induced elastin breakdown in structures, the quantitative data for the severity of collagen loss was obtained from the visual grading scale, giving basic information on the damaged ocular tissues due to UV that led to structural collagen damage

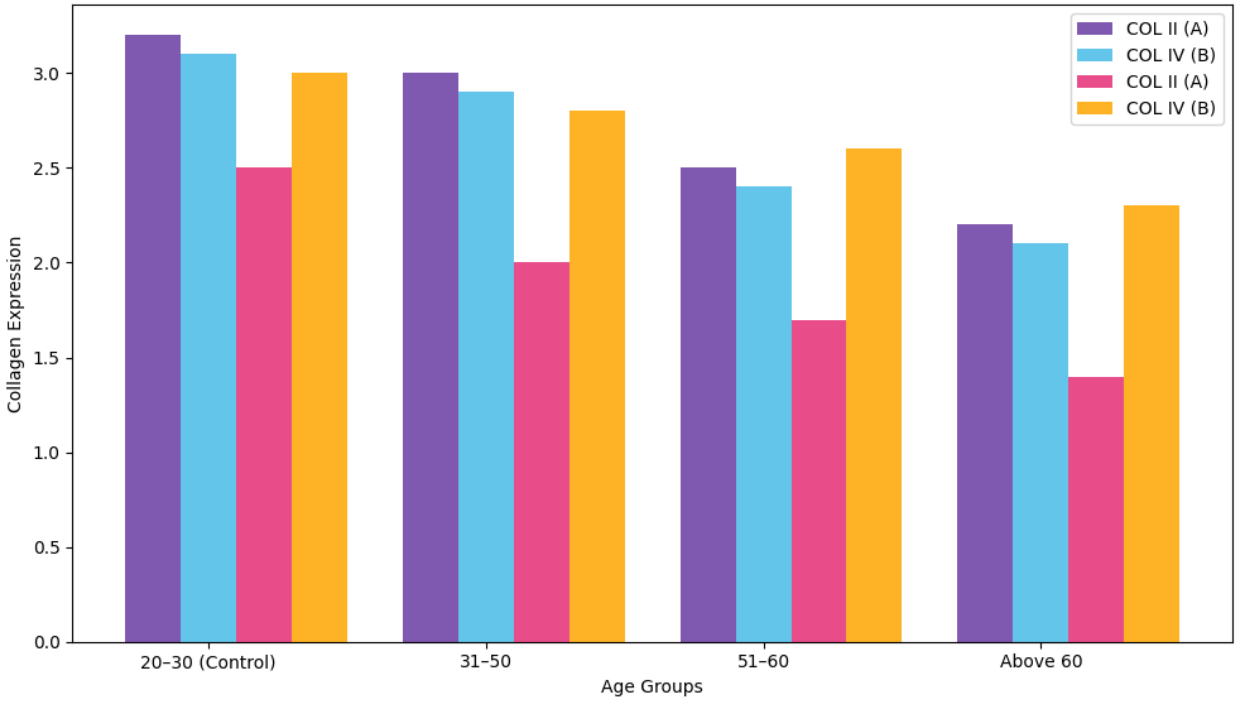

Figure 1. Collagen expression across biological aging groups (purple and blue colors) and photoaging groups (pink and orange colors).

and integrity.

Statistical analysis

SPSS 26.0 (IBM, Armonk, NY, USA) was employed to analyze gender, age group, body part, and kind of wound on the skin. Chi-square (X^2) test was used to examine relationships between categorical variables including the type of lesion, body part, and collagen expression levels of CII and CIV. t-test was applied to compare the expression levels of CII and CIV in biologically aged and photoaged skin tissues. Descriptive analysis was performed to report the mean values and standard deviations of collagen expression. *P* value less than 0.05 was defined as a statistically significant difference between compared groups.

Results and discussion

The comprehensive analysis of collagen degradation in skin tissues exposed to UV radiation.

By examining 320 tissue samples categorized into healthy, biologically aged, and UV-exposed groups, the research focused on the expression levels of collagen types II and IV across different age groups. The results showed that UV exposure caused significant damage to the collagen network, leading to accelerated collagen degradation in UV-exposed skin. Both CII and CIV expression were significantly reduced with CII decreasing by 45% and CIV decreasing by 38% compared to healthy skin samples. Previous reports claimed that protective measures such as antioxidants demonstrated the efficacy in mitigating oxidative stress and preventing collagen breakdown with a 22% improvement in collagen preservation in UV-exposed skin, emphasizing their protective role. The data highlighted significant changes in collagen integrity, emphasizing the impact of UV exposure and Elastosis on skin aging with potential implications for skin health and protective strategies. The results demonstrated the CII and CIV collagen expression patterns across specified age groups with the collagen amount decreased

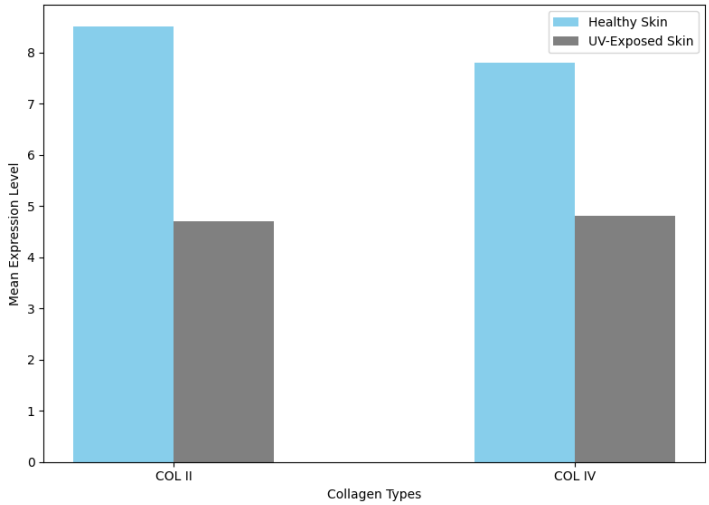

Figure 2. Collagen expression levels in healthy and UV-exposed skins.

Table 1. Collagen expression in UV-exposed skin with fibroelastic dysfunction.

Body Part	C II		CIV		Fibrosis and collagen expression	Skin elasticity and structural integrity	Collagen synthesis and remodeling	*P* value
	(A)	(B)	(A)	(B)				
Arms	3.20	2.10	1.40	3.60	2.80	3.60	2.60	0.01
Back	3.50	2.30	1.30	3.80	2.90	3.90	2.80	0.02
Chest	3.10	2.40	1.35	3.50	2.85	3.70	2.80	0.04
Shoulders	2.80	2.20	1.25	3.30	2.60	3.50	2.70	0.03
Neck	3.00	2.00	1.50	3.70	2.80	3.60	2.80	0.01
Feet	2.70	1.90	1.10	3.20	2.45	3.40	2.60	0.05

Note: (A): healthy skin. **(B)**: UV-exposed skin.

while the individual's age increased. Further, CII expression levels reduced significantly in photoaging group compared to that in the biological aging group, while CIV showed mixed results (Figure 1). The results suggested that various intrinsic and extrinsic factors altered collagen levels that maintained skin structure alongside skin elasticity.

Comparison of collagen types of CII and CIV expression levels in normal and UV-treated skin

The average expression levels of CII and CIV in healthy skin and UV-exposed skin showed that the collagen expression level was significantly reduced in UV-exposed skin, which highlighted the detrimental effects of UV radiation on collagen integrity (Figure 2). The result emphasized the role of UV exposure in accelerating collagen degradation and contributing to photoaging, which supported the investigations into skin aging mechanisms and potential therapeutic strategies for preserving collagen levels.

Comparative analysis of CII and CIV alongside fibrosis, skin elasticity, and collagen remodeling across different body parts

The expression levels of CII and CIV in healthy and UV-exposed skins were shown in Table 1. The results showed that UV-exposed skin tissues across the body were all with decreased CII expression levels, while CIV expression levels all increased compared to that in healthy skin tissues. The results quantified the effects of UV radiation on collagen structure and skin integrity across various body regions and highlighted a

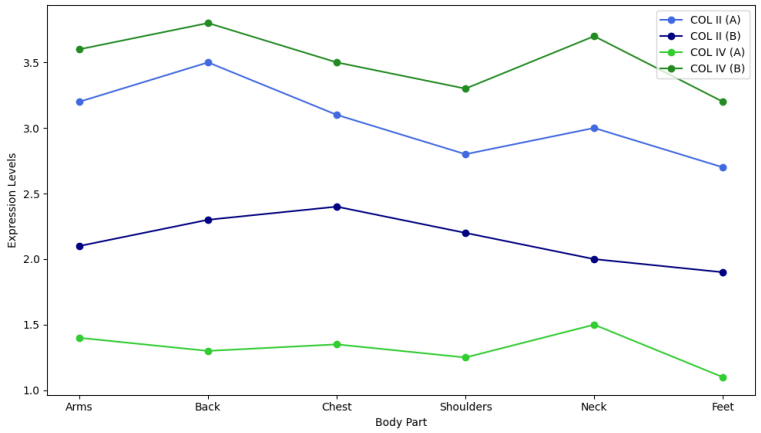

Figure 3. Collagen expression across different body parts.

Table 2. Regression analysis for UV exposure and collagen expression.

Collagen type	UV exposure (hours/day)	β	Standard Error (SE)	Confidence interval (95% CI)	R^2	*P* value
CII (A)	2.5	-0.30	0.09	(-0.48, -0.12)	0.58	0.0031
CIV (A)	2.5	-0.41	0.08	(-0.57, -0.25)	0.65	0.0027
CII (B)	2.5	-0.25	0.10	(-0.45, -0.05)	0.55	0.0055
CIV (B)	2.5	-0.38	0.09	(-0.56, -0.20)	0.62	0.0018

Note: (A): healthy skin. **(B)**: UV-exposed skin.

significant reduction of CII levels in UV-exposed areas, correlating with increased fibrosis, decreased elasticity, and impaired collagen remodeling. The findings aligned with the previous report, emphasizing the detrimental impact of UV experience on collagen degradation and skin aging and provided insight into targeted protective interventions for different body areas. The collagen expression levels of CII and CIV across six different body sections in healthy and UV-exposed skins were shown in Figure 3. The research explored tissue structure together with age-associated changes by identifying regions of dense or sparse expression. The results had specific benefits for research that analyzed variations in biological or environmental elements that influenced collagen distribution, therefore created targeted therapeutic approaches.

The relationship between UV exposure and collagen expression

The regression analysis of CII and CIV under UV exposure (hours/day) demonstrated that the rate of CIV degradation under UV exposure was -0.56 ($P = 0.0001$) compared to -0.42 of CII ($P = 0.0002$). Confidence intervals and R^2 values highlighted the strength and reliability of these relationships. Quantifying UV-induced collagen degradation demonstrated statistically significant differences, which supported the conclusion that UV exposure accelerated collagen loss, emphasizing its role in skin aging and photoaging mechanisms (Table 2).

UV radiation engaged in collagen degradation processes that accelerated the aging of skin tissue. Researchers discovered that abnormal concentrations of collagen types II and IV occurred after UV exposure with reductions of 45% in type II and 38% in type IV collagens when comparing affected skin to its unexposed state. UV radiation led to decreased levels of skin structural components and elasticity markers, which determined premature aging. A particular

evaluation of distinct body areas demonstrated different amounts of collagen degradation, in which the back along with the neck presented higher levels of damage because of elevated UV irradiation risk. This study emphasized how external sources of UV radiation accelerated collagen deterioration faster than that of natural biological degeneration. Based on the previous report, protective antioxidants helped to reduce oxidative stress by a 22% improvement in collagen protection, preventative skin intervention techniques that combined sunscreen protection and antioxidants might help in minimizing skin aging from sun exposure. This research added new knowledge about skin preservation strategies while advancing the understanding of collagen breakdown processes. This research focused solely on collagen types II and IV, which might limit the insights into other extracellular matrix elements. Future research should explore additional collagen types and molecular markers of photoaging. Advanced strategies like gene therapy and novel antioxidants could provide improved methods to prevent and reverse UV-induced skin aging effectively.

References

1. Kontaxi NI, Panoutsopoulou E, Ofrydopolou A, Tsoupras A. 2024. Anti-inflammatory benefits of grape pomace and tomato bioactive as ingredients in sun oils against UV radiation for skin protection. Appl Sci. 14(14):6236.
2. Zhang X, Zhuang H, Wu S, Mao C, Dai Y, Yan H. 2024. Marine bioactive peptides: anti-photoaging mechanisms and potential skin protective effects. Curr Issues Mol Biol. 46(2):990-1009.
3. Zhu X, Zhang X, Wang Z, Ren F, Zhu X, Chen B, *et al*. 2025. Screening and preparation of highly active antioxidant peptides of apricot and their inhibitory effect on ultraviolet radiation. Food Chem. 463:141336.
4. Hussein RS, Bin Dayel S, Abahussein O, El-Sherbiny AA. 2025. Influences on skin and intrinsic aging: Biological, environmental, and therapeutic insights. J Cosmet Dermatol. 24(2):e16688.
5. Yuan M, Fu H, Mo Q, Wang S, Wang C, Wang D, *et al*. 2024. Protective mechanism of *Rosa roxburghii* Tratt fermentation broth against ultraviolet-A-induced photoaging of human embryonic skin fibroblasts. Antioxidants. 13(3):382.
6. Tai M, Chen J, Chen J, Shen X, Ni J. 2024. Endoplasmic reticulum stress in skin aging induced by UVB. Exp Dermatol. 33(1):e14956.
7. Slominski RM, Chen JY, Raman C, Slominski AT. 2024. Photo-neuro-immuno-endocrinology: How the ultraviolet radiation regulates the body, brain, and immune system. Proc Natl Acad Sci USA. 121(14):e2308374121.
8. Ahn SY, Lee CD, Ku JJ, Lee S, Lee S. 2024. The anti-aging potential of *Cephalotaxus harringtonia* extracts: The role of harringtonine and homoharringtonine in skin protection. Appl Biol Chem. 67(1):96.
9. Ko HJ, Sim SA, Park MH, Ryu HS, Choi WY, Park SM, *et al*. 2024. Anti-photoaging effects of upcycled *Citrus junos* seed anionic peptides on ultraviolet-radiation-induced skin aging in a reconstructed skin model. Int J Mol Sci. 25(3):1711.
10. Song S, Li F, Zhao B, Zhou M, Wang X. 2025. Ultraviolet light causes skin cell senescence: From mechanism to prevention principle. Adv Biol (Weinh). 9(2):e2400090.
11. Tang X, Yang T, Yu D, Xiong H, Zhang S. 2024. Current insights and future perspectives of ultraviolet radiation (UV) exposure: Friends and foes to the skin and beyond the skin. Environ Int. 185:108535.
12. Draelos Z, Bogdanowicz P, Saurat JH. 2024. Top weapons in skin aging and actives to target the consequences of skin cell senescence. J Eur Acad Dermatol Venereol. 38:15-22.
13. Ferrara F, Pecorelli A, Pambianchi E, White S, Choudhary H, Casoni A, *et al*. 2024. Vitamin C compound mixture prevents skin barrier alterations and inflammatory responses upon real-life multi-pollutant exposure. Exp Dermatol. 33(1):e15000.
14. Liu T, Zhu S, Yang Y, Qin W, Wang Z, Zhao Z, *et al*. 2024. Oroxylin A ameliorates ultraviolet radiation-induced premature skin aging by regulating oxidative stress *via* the Sirt1 pathway. Biomed Pharmacother. 171:116110.
15. Dwevedi D, Srivastava A. 2024. Molecular mechanisms of polyphenols in the management of skin aging. Curr Aging Sci. 17(3):180-188.
16. He X, Gao X, Guo Y, Xie W. 2024. Research progress on bioactive factors against skin aging. Int J Mol Sci. 25(7):3797.
17. Mmbando GS, Ngongolo K. 2024. Environmental & health impacts of ultraviolet radiation: Current trends and mitigation strategies. Discov Sustain. 5(1):436.
18. Baik KK, Song WY, Song DK, Yun J, Jang JH, Oh JY, *et al*. 2024. Protective effects of sesame glycoproteins on ultraviolet-induced skin aging: *In vitro* and *in vivo* studies. Pharmaceuticals. 17(10):1306.