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Modulation of NF-κB, MMP-1, and HAase by *Coriandrum sativum* Seed Extract in UV-Induced Skin Aging Model

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Abstract

Background: *Coriandrum sativum* L. (coriander) contains abundant flavonoids and phenolic compounds that contribute substantially to its antioxidant capacity. These constituents exhibit strong free radical–scavenging activity, offering protection against cellular damage and premature skin aging, particularly under Ultraviolet (UV) exposure. This study evaluated the anti-aging effects of Coriander Seed Extract (CSE) in an *in vitro* UVB-induced photoaging model using human dermal fibroblasts (BJ cells).

Methods: CSE was prepared through ethanol maceration. Cytotoxicity was assessed using the WST-8 assay. CSE was tested at concentrations ranging from 1.56 to 100 *µg/ml*. Hyaluronidase (HAase) protein levels were quantified using a human HAase ELISA kit. Gene expression of *NF-κB* and *MMP-1* was analyzed using quantitative Real-Time PCR (qRT-PCR).

Results: CSE concentrations up to 25 *µg/ml* exhibited no cytotoxic effects. Treatment with 25 *µg/ml* CSE significantly downregulated *NF-κB* and *MMP-1* expression in UV-irradiated BJ cells compared with the untreated UV control. In addition, CSE reduced HAase protein levels, indicating improved preservation of the extracellular matrix and reduced hyaluronic acid degradation. Notably, the percentage of senescent cells decreased by nearly 50% compared with the UV-exposed group ($p<0.05$), restoring levels comparable to non-irradiated cells.

Conclusion: Overall, CSE may exert anti-aging activity by modulating inflammatory pathways, preventing extracellular matrix breakdown, and limiting UV-induced cellular senescence *in vitro*. These findings suggest its potential for development as a natural active ingredient for anti-aging skincare formulations; however, further *in vivo* and clinical studies are required to confirm its safety and efficacy.

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Introduction

Skin aging is a multifaceted biological process shaped by intrinsic factors related to natural chronological progression and extrinsic influences originating from environmental exposure. Skin aging is generally classified into intrinsic aging and extrinsic aging (photoaging), the latter being predominantly induced by Ultraviolet (UV) radiation and other environmental stressors [1,2]. Among these factors, prolonged UV exposure is widely recognized as the primary environmental contributor to premature skin aging because it induces oxidative stress, inflammation, and DNA damage [3]. These molecular alterations ultimately lead to structural and functional deterioration of the dermal extracellular matrix [4].

Growing interest in anti-aging strategies has led to the development of various approaches, ranging from synthetic compounds and retinoids to antioxidants and plant-derived phytochemicals. Botanical extracts enriched with bioactive constituents are considered promising due to their multifunctional biological effects, favorable safety profiles, and high biocompatibility [5]. Within this context, *Coriandrum sativum* L. (coriander) has gained attention for its antioxidant, anti-inflammatory, and enzyme-modulating activities.

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Despite increasing interest in plant-derived compounds for photoaging management, comprehensive evaluation of Coriander Seed Extract (CSE) in UV-induced dermal fibroblast models remains limited.

Coriandrum sativum, a species within the Apiaceae family, is widely cultivated and traditionally used as both a culinary and medicinal plant. Its seeds contain significant levels of flavonoids, phenolic acids, linalool, quercetin, and coumarins [6,7]. These phytoconstituents demonstrate strong free radical scavenging properties that may counteract oxidative stress, which is a fundamental driver of the skin aging process. Furthermore, coriander exhibits inhibitory effects on inflammatory cascades and matrix-degrading enzymes, making it a potential candidate for anti-aging applications [8]. However, although these bioactivities have been reported, the integrated effects of CSE on key molecular markers of UV-induced photoaging in human dermal fibroblasts have not been systematically characterized.

NF-κB, a key transcription factor involved in oxidative stress pathways and inflammatory regulation, becomes activated under UV exposure and stimulates the release of pro-inflammatory cytokines that contribute to accelerated skin aging [9]. MMP-1, an essential matrix metalloproteinase responsible for degrading type I and III collagen, plays a major role in wrinkle formation through its involvement in dermal extracellular matrix degradation [10]. HAase, an enzyme responsible for hyaluronic acid breakdown, contributes to reduced skin hydration and dermal thinning when present at elevated levels [11]. Additionally, cellular senescence represents a major hallmark of aging, characterized by irreversible growth arrest accompanied by increased secretion of pro-inflammatory mediators. UV radiation is known to induce premature senescence in dermal fibroblasts, thereby exacerbating aging-related changes [12]. Notably, many *in vitro* photoaging studies assess these pathways individually, without simultaneously evaluating inflammatory signaling, extracellular matrix degradation, and senescence-related alterations within a single experimental framework.

Although coriander has been reported to possess antioxidant and anti-inflammatory properties, its specific effects on NF-κB, MMP-1, HAase, and cellular senescence in UV-induced human dermal fibroblasts have not been comprehensively evaluated. Therefore, this study aims to investigate whether CSE can modulate key inflammatory, extracellular matrix–related, and senescence-associated markers in UV-induced BJ fibroblasts, thereby addressing this research gap and providing preliminary mechanistic insight into its potential anti-photoaging activity.

Materials and Methods

Coriander seed extraction

Fresh coriander seeds were collected from farmers in West Java, Indonesia, and washed thoroughly. CSE was prepared at PT Fathonah Amanah Shidiq Tabligh (FAST), Indonesia, following Good Manufacturing Practice (GMP) standards of the Indonesian Food and Drug Supervisory Agency (BPOM). The extraction process was conducted under standardized GMP procedures to ensure batch consistency; however, quantitative phytochemical profiling such as total phenolic or flavonoid content analysis was beyond the scope of the present study. CSE was prepared using maceration with 96% ethanol as the solvent. The seeds were soaked for 72 *hr* with periodic agitation, after which the mixture was filtered. The resulting filtrate was concentrated using a rotary evaporator to obtain a crude extract. The concentrated extract was subsequently blended with lactose as a carrier to obtain a dry powdered extract and stored at 25±2°*C* until further use [13].

BJ cell culture and UV induction

BJ cells (ATCC® CRL-2522™) obtained from Aretha Medika Utama, Indonesia, were maintained in complete MEM medium (Biowest, L0416-500) with 10% fetal bovine serum (Biowest, S1810-500). As a commercially available human cell line, this study did not require specific institutional ethical approval. Senescence induction was carried out on BJ cells that had reached 80% confluence using UVB light [14], prior to CSE treatment. UVB irradiation was performed using a Kernel UVB lamp (Model KN-4003BL, wavelength range 280-320 *nm*). BJ fibroblasts were exposed at a fixed distance of 15 *cm* from the light source for 75 *min*. The irradiation conditions were optimized in preliminary experiments to induce sublethal photoaging responses without causing extensive cell death [14,15]. Preliminary optimization experiments confirmed that these conditions resulted in sublethal cellular stress, as indicated by preserved cell viability above 80% in subsequent assays, supporting their suitability as a photoaging model rather than an acute cytotoxic model. Experimental treatments were applied under standardized laboratory conditions. Due to the *in vitro* design and objective outcome measurements, formal blinding and randomization procedures were not implemented.

Cytotoxicity test

For the cytotoxicity assessment, BJ cells were seeded into 96-well plates and allowed to incubate for 24 *hr* at 37°*C* in a 5% CO_2 atmosphere. The medium was then refreshed, followed by UVB irradiation and treatment with CSE at concentrations (100 to 1.56 *µg/ml*). The concentration range of CSE (100-1.56 *µg/ml*) was selected based on preliminary screening experiments and commonly applied concentration ranges in *in vitro* phytochemical studies to evaluate both cytotoxic and sub-cytotoxic effects. The extract was dissolved in DMSO prior to dilution in culture medium. The final DMSO concentration in treated groups did not exceed 1%. Preliminary cytotoxicity testing confirmed that this concentration did not significantly affect BJ

Table 1. Primers sequence

Genes	Primer sequence (5' to 3')	Size (*bp*)	Annealing (*°C*)	Cycle	NCBI references sequence
NF-κB (Human)	F: TCTCTATGACCTGGATGACTC R: GTTTCATGTCTCCTTGTGCT	173	57	40	NM_001382628
MMP-1 (Human)	F: CCAAGCTCATCACCTGGTCT R: TCGATGTCAATGGTCTGGAA	429	57	40	NM_001145938.2
GAPDH (Human)	F: GCCAAAAGGGTCATCATCTC R: TGAGTCCTTCCACGATACCA	217	58	40	NM_001289726.1

Table 2. Concentration and Purity of RNA

Sample	Concentration (*ng/µl*)	Purity (λ260/λ280 *nm*)
NC	24.40	1.9308
PC	12.72	1.7213
DMSO	12.24	1.6500
CSE 25 *µg/ml*	10.72	1.4545
CSE 6.25 *µg/ml*	12.72	1.6406
CSE 1.56 *µg/ml*	12.32	1.5625

fibroblast viability. Subsequently, 20 *µl* of WST-8 reagent (Water-Soluble Tetrazolium-8; CCK-8 buffer, E-CK-A362) was added to each well to evaluate cell viability [16]. Absorbance was recorded at 450 *nm* using a microplate spectrophotometer [15].

Quantification of HAase level

The ELISA procedure was carried out following the instructions from the manufacturer for the Human HAase Kit (Elabscience, E-EL-H2201). All steps were performed according to the kit manual, and absorbance values were subsequently recorded using a microplate reader (Multiskan™ Spectrophotometer).

Gene expression analysis: NF-kB and MMP-1

Gene expression analysis was performed using a quantitative Real-Time PCR (qRT-PCR) workflow. After treatment, total RNA was extracted from BJ cells and converted into complementary DNA (cDNA). The primer pairs specific for NF-κB and MMP-1 used for transcript quantification are presented in table 1. BJ cell pellets were processed with Tri Reagent (Zymo Research, R2050-1-200), and RNA purification was completed using the Direct-zol™ RNA Miniprep Plus kit (Zymo Research, R2073). The concentration and purity profiles of the isolated RNA are shown in table 2. cDNA synthesis was conducted using the Sensi-FAST cDNA synthesis kit (Bioline, BIO-65054). Quantitative assessment of target gene expression was subsequently carried out with the AriaMx Real-Time PCR System (Agilent, G8830A) [15].

Senescence-associated β-galactosidase (SA-β-gal) assay

Cells were washed with Phosphate-Buffered Saline (PBS) and fixed using 4% paraformaldehyde for 10-15 *min* at room temperature. BJ cells were then incubated with SA-β-gal staining solution (pH=6.0) at 37*°C* (without CO_2) overnight according to the manufacturer's protocol. Senescent cells were identified by the presence of blue staining under a light microscope. Quantification was performed by counting stained (blue) and unstained cells in five randomly selected fields per well at 100× magnification. A minimum of 200 cells per group were evaluated. The percentage of senescent cells was calculated as the ratio of SA-β-gal–positive cells to total counted cells [12].

Statistical analysis

Statistical analyses were performed using SPSS version 23.0 (SPSS Inc., USA). Normality was assessed using the Shapiro–Wilk test and homogeneity of variance using Levene's test. Data that met both normality and homogeneity assumptions were analyzed using one-way ANOVA followed by Tukey's HSD post hoc test. For datasets that did not meet normality and/or homogeneity assumptions, the Kruskal–Wallis test was applied, followed by pairwise comparisons using the Mann–Whitney test. A significance level of $p<0.05$ was considered statistically significant. All experiments were performed in triplicate and repeated in at least three independent experiments. Data are presented as mean±standard deviation (SD). Due to the exploratory nature of this *in vitro* study and the limited sample size, formal effect size calculations and post hoc power analyses were not performed. However, consistent trends were observed across independent biological replicates. Graphical representations were generated as histogram plots using GraphPad Prism software (version 8.0.244) [13].

Results

Effect of CSE on cell viability and inhibition in UV-induced BJ cells

Cell morphology is illustrated in figure 1A. BJ fibroblasts exhibited typical fibroblast-like morphology across all treatment groups. The cells demonstrated plastic adherence and spindle-shaped appearance, with elongated cytoplasmic extensions and centrally located nuclei. In the negative control group (normal BJ cells), the morphology was consistent with healthy fibroblasts—uniform, fusiform, and well spread across the culture surface. In contrast, UV-induced cells showed altered morphology characterized by increased granularity, cytoplasmic shrinkage, and a less organized monolayer, indicating stress and early signs of senescence. Treatment with CSE, particularly at 25 *µg/ml*, partially preserved fibroblast morphology, with cells maintaining a spindle-like shape and improved confluence

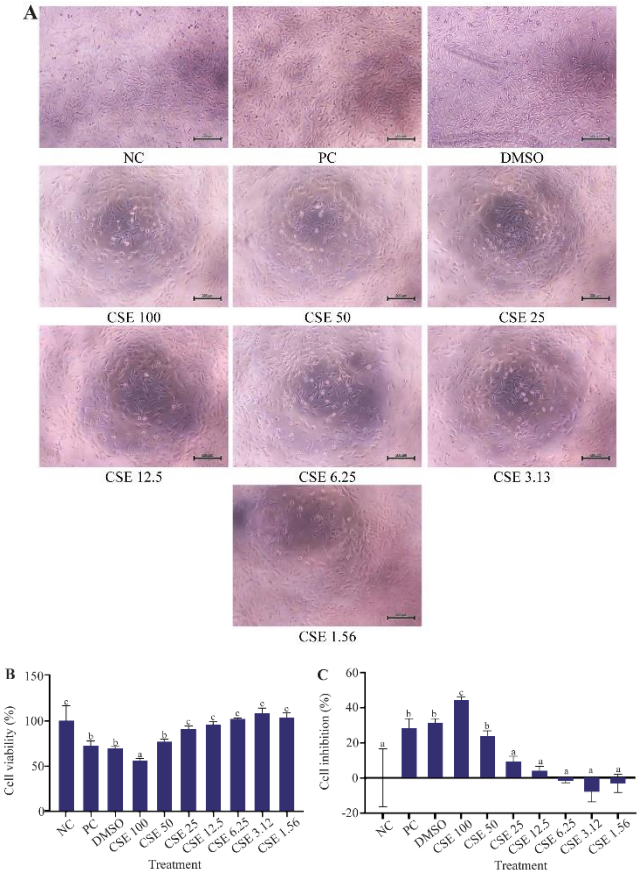

Figure 1. A) Morphology of BJ Cells, B) Effect of CSE concentrations toward viability percentage in BJ Cells, C) Effect of CSE Concentrations toward inhibition percentage in BJ Cells.
* Data are presented as mean average±SD from four replications. NC: negative control (normal BJ cell); PC: positive control (UV-induced BJ cell); DMSO: comparative control (positive control+DMSO 1%); CSE: treatment with different concentration (100, 50, 25, 12.5, 6.25, 3.12, 1.56 *μg/ml*). Different letters indicate significant differences based on Mann–Whitney pairwise comparison following Kruskal–Wallis analysis ($p<0.05$).

compared with the UV-exposed group. These morphological observations were qualitative and not supported by quantitative morphometric analysis (*e.g.*, cell density or nuclear morphology measurements).

In line with these morphological observations, cell viability analysis further supported the protective effect of CSE. UV exposure significantly reduced cell viability compared to the negative control ($p<0.05$), confirming cellular damage caused by photooxidative stress. Notably, CSE at high concentrations (100 and 50 *μg/ml*) further decreased cell viability, with CSE 100 *μg/ml* showing the lowest viability among all groups, indicating potential cytotoxicity at excessive doses. Conversely, CSE at concentrations of 25 *μg/ml* and below (12.5, 6.25, 3.12, and 1.56 *μg/ml*) significantly improved cell viability compared to the positive control ($p<0.05$), with values not significantly different from the negative control ($p>0.05$) at 25 *μg/ml*. These results indicate that lower doses of CSE are non-

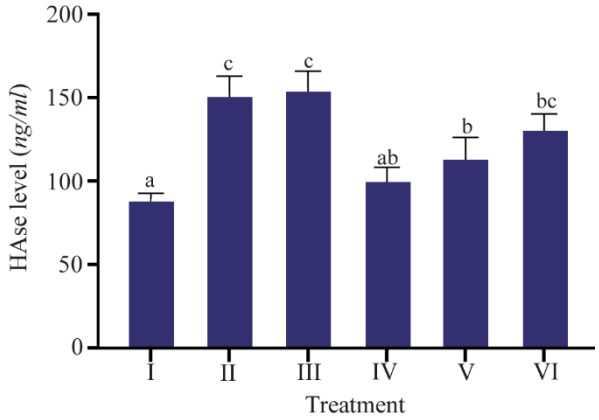

Figure 2. Effect of CSE on HAase level in UV-induced BJ cell.
*Data are presented as mean average±SD from four replications. I: negative control (normal BJ cell); II: positive control (UV-induced BJ cell); III: comparative control (positive control+DMSO 1%); IV: positive control+CSE 25 *μg/ml*; V: positive control+CSE 6.25 *μg/ml*; VI: positive control+CSE 1.56 *μg/ml*. Different letters indicate significant differences based on Mann–Whitney pairwise comparison following Kruskal–Wallis analysis ($p<0.05$).

toxic and may exert cytoprotective effects in UV-stressed cells. Some variability was observed at higher concentration ranges, which may reflect biological heterogeneity under UV-induced stress conditions. All experiments were performed in three independent replicates.

This pattern is further supported by the cell inhibition percentage (Figure 1C), where higher concentrations of CSE (50 and 100 *μg/ml*) resulted in the greatest inhibition of cell growth, consistent with their cytotoxicity. Meanwhile, treatments with CSE at 25 *μg/ml* and lower showed minimal or even negative inhibition percentages, suggesting either a neutral or proliferative effect on BJ cells. A concentration range yielding cell viability values above 80% was selected for subsequent experiments, specifically 25, 6.25, and 1.56 *μg/ml*.

Effect of CSE on HAase level in UV-induced BJ cell

The effect of CSE on hyaluronidase (HAase) levels in UV-induced BJ cells is shown in figure 2. UV irradiation significantly increased HAase levels compared with the negative control ($p<0.05$), indicating enhanced extracellular matrix degradation as a hallmark of photoaging. Treatment with CSE at various concentrations (Groups IV-VI) resulted in a dose-dependent reduction of HAase levels. The most notable reduction was observed at 25 *μg/ml* (IV), which significantly decreased HAase expression compared to the UV control ($p<0.05$). No statistically significant difference was observed between the 25 *μg/ml* group and the non-irradiated control ($p>0.05$), indicating partial restoration toward baseline levels. The 1.56 and 6.25 *μg/ml* concentrations (V and VI) also decreased HAase levels but to a lesser extent. These findings suggest that CSE, particularly at 25 *μg/ml*, can attenuate UV-induced

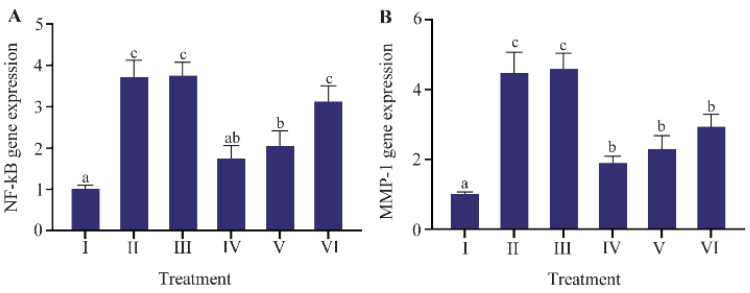

Figure 3. *NF-κB* and *MMP-1* gene expression after treatment with CSE. A) *NF-κB* gene expression, B) *MMP-1* gene expression.
*Data are presented as mean average±SD from four replications. I: negative control (normal BJ cell); II: positive control (UV-induced BJ cell); III: comparative control (positive control+DMSO 1%); IV: positive control+CSE 25 *μg/ml*; V: positive control+CSE 6.25 *μg/ml*; VI: positive control+CSE 1.56 *μg/ml*. Different letters indicate significant differences based on Mann–Whitney pairwise comparison following Kruskal–Wallis analysis ($p<0.05$).

increases in HAase levels, indicating its potential to preserve the extracellular matrix and mitigate skin aging.

Effect of CSE on NF-kB and MMP-1 gene expression in UV-induced BJ cell

Figure 3 illustrates the effects of CSE on the expression of two key photoaging-related genes: NF-κB (Figure 3A) and MMP-1 (Figure 3B) in UV-induced BJ cells. UV irradiation significantly upregulated both *NF-κB* and *MMP-1* gene expression compared with the negative control ($p<0.05$), confirming that UV exposure induces inflammation and extracellular matrix degradation, two hallmarks of photoaging. Treatment with CSE at 25 *μg/ml* significantly reduced *NF-κB* expression compared with the UV group ($p<0.05$). Although expression levels were markedly reduced, complete normalization to baseline levels was not assumed. Lower concentrations (1.56 and 6.25 *μg/ml*) also showed downregulation of *NF-κB*, though the 1.56 *μg/ml* dose did not differ significantly from the UV-exposed groups, suggesting reduced efficacy at minimal doses.

A similar trend was observed for *MMP-1* gene expression, where all CSE-treated groups showed a significant decrease compared to the UV-induced group ($p<0.05$). The 25 *μg/ml* concentration resulted in the lowest *MMP-1* levels, indicating the strongest inhibitory effect on collagen degradation; however, no significant difference was observed among the treated groups. These findings indicate modulation of inflammatory and matrix-related gene expression under *in vitro* conditions.

Effect of CSE on senescence cell in UV-induced BJ cell

Treatment with CSE reduced the proportion of senescent cells in a dose-dependent manner (Figure 4). Cellular senescence was assessed using senescence-associated β-galactosidase (SA-β-gal) staining, and positively stained (blue) cells were quantified under light microscopy as a percentage of total cells. The 25

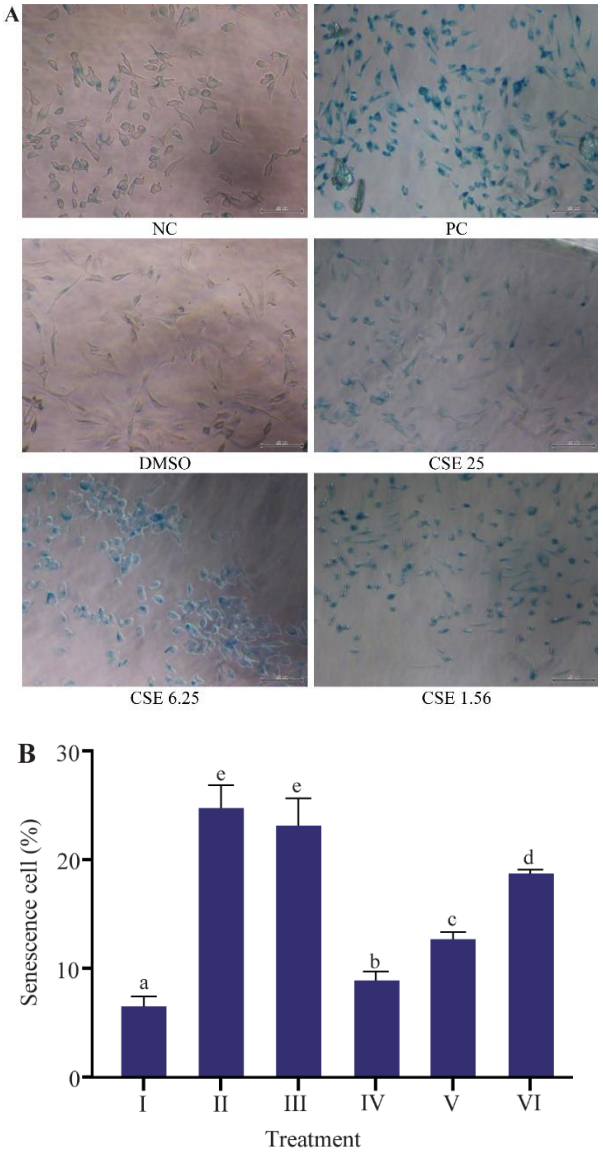

Figure 4. Effect of CSE on senescence cell in UV-induced BJ cell.
* Data are presented as mean average±SD from four replications. I: negative control (normal BJ cell); II: positive control (UV-induced BJ cell); III: comparative control (positive control+DMSO 1%); IV: positive control+CSE 25 *μg/ml*; V: positive control+CSE 6.25 *μg/ml*; VI: positive control+CSE 1.56 *μg/ml*. Different letters indicate significant differences based on Mann–Whitney pairwise comparison following Kruskal–Wallis analysis ($p<0.05$).

μg/ml concentration exhibited the most pronounced decrease in senescence, significantly lower than both the UV and DMSO groups ($p<0.05$), and not significantly different from the negative control ($p>0.05$). The 6.25 *μg/ml* dose also reduced senescence but was less effective than the 25 *μg/ml* dose, while the 1.56 *μg/ml* dose showed only a modest reduction and remained significantly higher than the lower concentrations. These results suggest a potential anti-senescent effect of CSE in UV-induced fibroblasts; however, the findings should be interpreted within the limitations of an *in vitro* model.

Discussion

The cytotoxic potential of *Coriandrum sativum* seed extract (CSE) was evaluated in UV-induced BJ fibroblasts using the WST-8 assay, which measures cellular metabolic activity as an indicator of viable cells. Importantly, the selected UVB exposure was designed to induce sublethal stress conditions characteristic of photoaging rather than apoptosis or necrosis. This is supported by the observed increase in senescence markers alongside maintained cell viability in the positive control group, suggesting that the model reflects features of premature aging rather than acute phototoxicity.

As shown in figure 1, CSE exhibited a concentration-dependent effect on cell viability and inhibition percentage. At higher concentrations (100 and 50 *µg/ml*), CSE significantly reduced cell viability compared to both the negative and positive controls, with the 100 *µg/ml* dose showing the most pronounced cytotoxic effect. This was reflected by a marked decrease in viability (below 60%) and a corresponding increase in cell inhibition percentage (>40%). These findings indicate that concentrations ≥50 *µg/ml* may induce cytotoxic stress under *in vitro* conditions. The present study did not investigate the exact mechanism underlying this cytotoxicity; therefore, further studies are required to determine whether this effect is related to oxidative imbalance, mitochondrial dysfunction, or other cellular pathways.

The DMSO control did not demonstrate significant cytotoxicity compared with the negative control, indicating that the reduced viability at ≥50 *µg/ml* was unlikely due to solvent effects. Additionally, the tested concentrations were not expected to meaningfully alter culture osmolarity. Therefore, the cytotoxicity observed at higher doses is more plausibly attributed to intrinsic phytochemical activity. High levels of polyphenols and related bioactive compounds may exert pro-oxidant effects under certain conditions, resulting in oxidative imbalance or mitochondrial dysfunction. This biphasic pattern—protective at lower doses and cytotoxic at higher doses—is consistent with hormetic responses described for plant-derived antioxidants. However, direct assessment of intracellular ROS or mitochondrial integrity was not performed and requires further investigation.

In contrast, CSE at concentrations of 25 *µg/ml* and below (12.5, 6.25, 3.12, and 1.56 *µg/ml*) maintained cell viability above 90%, with minimal or negative cell inhibition values, indicating a non-cytotoxic and potentially cytoprotective effect. These concentrations were statistically comparable to the negative control, suggesting that lower doses of CSE are biocompatible and safe for use in dermal fibroblasts. According to ISO 10993-5 standards, materials resulting in ≥80% cell viability are classified as non-cytotoxic [17], further confirming the safety of CSE at these concentrations *in vitro*.

This study suggests that *Coriandrum sativum* seed extract (CSE) may exert protective effects against UV-induced photoaging in human dermal fibroblasts by modulating markers associated with inflammation, matrix degradation, and cellular senescence. CSE has garnered attention for its potential anti-aging properties, particularly concerning the modulation of gene expression related to matrix metalloproteinase-1 (MMP-1) in UV-induced skin damage models. The modulation of inflammatory pathways, particularly NF-κB signaling, indicates a potential contribution of the extract to skin protective responses [10]. However, the present study did not directly examine upstream signaling cascades; therefore, the observed modulation of NF-κB and MMP-1 should be interpreted as associative rather than mechanistically confirmed.

Our findings indicate that CSE reduced HAase levels, with 25 *µg/ml* showing the most pronounced effect among the tested concentrations. HAase is an enzyme that degrades Hyaluronic Acid (HA) and contributes to various pathological conditions, including skin aging [18]. The role of HA and its degradation products in cellular signaling and skin homeostasis emphasizes the importance of regulating HAase levels to maintain skin integrity and prevent premature aging [19]. These findings are consistent with previous reports suggesting the potential pharmacological activity of CSE in modulating enzymatic processes related to skin aging. For instance, Ghazanfari *et al* reported that extracts of coriander seeds possess antioxidant properties [20], and Palmieri *et al* identified phytochemical components such as linalool that may help mitigate oxidative damage [21]. Nevertheless, whether the reduction in HAase levels observed in this study results from direct enzyme inhibition, antioxidant activity, or secondary signaling modulation was not specifically evaluated and remains to be clarified.

In addition, the present study did not directly assess intracellular Reactive Oxygen Species (ROS) levels, total antioxidant capacity (*e.g.*, DPPH or ABTS radical scavenging assays), or protein-level activation of NF-κB signaling components such as phosphorylated p65 or IκBα. Therefore, while gene expression changes suggest modulation of oxidative and inflammatory pathways, the precise biochemical mechanism underlying these effects remains to be confirmed. Future investigations integrating antioxidant assays, ROS quantification, and protein-based signaling analyses would provide stronger mechanistic validation.

Furthermore, our study showed that treatment with CSE at 25 *µg/ml* was associated with reduced expression of NF-κB and MMP-1, two critical markers involved in photoaging. The extract may contribute to the downregulation of NF-κB, a transcription factor involved in inflammatory and cellular stress responses. The anti-inflammatory activity of coriander extract has also been reported in previous research [22], including

studies demonstrating inhibition of NF-κB activation in macrophage models.

In parallel, CSE significantly suppressed *MMP-1* gene expression, which contributes to collagen degradation and extracellular matrix breakdown [23]. One pivotal study conducted by Salem *et al* demonstrated that coriander essential oil and nano formulations decreased MMP-1 expression *in vitro*. Although previous studies have implicated signaling pathways such as TGFβ/SMAD in MMP-1 regulation [10], these mechanisms were not experimentally assessed in the present work. These findings are also consistent with those of Huang *et al*, who reported that coriander leaf extract inhibited MMP-1 expression and transcription factors such as AP-1 involved in skin aging. Their research concluded that the antioxidant properties of coriander contribute to a decrease in MMP-1 levels, thereby promoting a healthier skin structure [24].

Moreover, as illustrated in figure 4, CSE was also effective in preventing UV-induced cellular senescence in BJ fibroblasts, with the 25 *μg/ml* concentration showing the strongest anti-senescent activity. This suggests a potential role in attenuating UV-induced senescence-associated alterations in dermal fibroblasts. Senescence assessment in the present study was limited to SA-β-gal staining, which, although widely used as a hallmark of cellular senescence, does not provide molecular confirmation of senescence pathway activation. Therefore, incorporation of additional markers such as p16INK4a, p21, or γH2AX in future studies would strengthen the validation of the anti-senescent effect. Consequently, the molecular basis of this effect remains to be elucidated. UV radiation is well recognized to induce oxidative stress, DNA damage, and senescence-related signaling in skin cells [25]. Previous mechanistic studies have demonstrated that environmental stressors, including cigarette smoke extract and UV exposure, activate pathways involving p53, p21, and p16, which contribute to cellular senescence [26,27]. In addition, proteomic analyses of UV-induced photodamage models have highlighted the involvement of oxidative stress–related pathways in senescence progression [28]. Although these mechanisms were not directly evaluated in the present study, they provide a biological context for interpreting the observed anti-senescent effects of CSE.

However, considering the methodological constraints described above, these findings should be interpreted as preliminary. Several limitations warrant acknowledgment. First, the CSE was not subjected to comprehensive phytochemical characterization (*e.g.*, quantitative phenolic/flavonoid analysis or chromategraphic profiling), which may affect reproducibility and limit precise mechanistic interpretation. Second, direct mechanistic validation was not conducted, as intracellular ROS levels, upstream NF-κB signaling components (*e.g.*, phosphorylated p65 or IκBα), and additional structural or oxidative stress markers such as collagen type I or malondialdehyde were not assessed. Third, some RNA samples exhibited relatively low purity ratios (λ260/280), which could influence qRT-PCR sensitivity despite normalization to GAPDH. In addition, melt curve analysis and amplification efficiency validation were not performed due to instrument limitations, which should be considered when interpreting the gene expression data. Finally, this study utilized a single BJ fibroblast monoculture model that does not capture dermal–epidermal interactions, immune components, or inter-individual variability present in human skin, and therefore extrapolation to *in vivo* or clinical settings should be undertaken with caution. Future studies are warranted to further support translational application, including the use of advanced models such as three-dimensional skin equivalents or organ-on-chip systems, clinical evaluation of safety and efficacy at physiologically relevant concentrations, assessment of active compound stability under cosmetic storage conditions, investigation of potential interactions with other cosmeceutical ingredients, and optimization of delivery systems to enhance dermal penetration and bioavailability.

Conclusion

The results demonstrate that *Coriandrum sativum* seed extract exerts anti-aging effects in UV-induced fibroblast cells through multiple mechanisms: inhibiting inflammatory pathways (NF-κB), reducing matrix degradation enzymes (HAase and MMP-1), and decreasing cellular senescence. Among the tested doses, 25 *μg/ml* showed the most consistent and significant protective effects, indicating it as the optimal concentration for anti-aging applications. These findings highlight the promise of CSE as a natural ingredient in topical formulations aimed at preventing or mitigating photoaging.

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Conflict of Interest

The authors have no conflict of interest.

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