

## Article

# Photoprotective Effects of Oral Coriander (*Coriandrum sativum* L.) Seed Oil Supplementation Against UV-Induced Skin Damage: Evidence from Two Randomized, Double-Blind, Placebo-Controlled Clinical Trials

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## Abstract

Skin is constantly exposed to UV radiation. While topical sunscreens are the main preventative measure, oral photoprotective agents are emerging as promising systemic adjuncts, offering uniform, continuous protection. This study presents the results of two clinical trials designed to evaluate the efficacy of supplementation with a standardized coriander (*Coriandrum sativum* L.) seed oil (CSO) in mitigating UV-induced skin damage, in comparison with a placebo. The first trial investigated the effects of CSO supplementation on women with reactive skin, assessing UVA+B-induced skin erythema and tumor necrosis factor-alpha (TNF- $\alpha$ ) release. The second trial included women of all skin types and, in addition to the outcomes mentioned above, examined UVA-induced lipoperoxidation. Measurements were taken before and after 56 days of supplementation. CSO supplementation led to a significant reduction in UV-induced skin erythema and associated TNF- $\alpha$  levels in both cohorts, with decreases of 11.8% and 24.1% in the reactive skin group and 18.1% and 18.7% in the cohort with all skin types, respectively. In women of all skin types, UV-induced skin lipoperoxidation was reduced by 31.9% at 4 h and by 69.9% at 24 h post-exposure. To the best of our knowledge, this is the first study reporting the photoprotective efficacy of CSO. This finding is attributed to CSO's high petroselinic acid content and its known anti-inflammatory properties.

**Keywords:** coriander seed oil; free fatty acids; petroselinic acid; erythema; inflammation; clinical trial

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## 1. Introduction

As the largest organ of the human body, the skin acts as a critical biological barrier that is constantly challenged by a wide array of external and internal exposome-related stressors throughout its lifespan [1–3]. Being ubiquitous in the environment, ultraviolet radiation (UVR) is one of the most deleterious environmental factors influencing skin biology and physiology to which everyone is inevitably exposed to [4]. UVR is subdivided into three distinct spectral regions: UVA (315–400 nm), UVB (280–315 nm), and UVC (100–

280 nm). While UVC radiation is effectively filtered by the stratospheric ozone layer, UVB and UVA reach the Earth's surface and penetrate the skin in a wavelength-dependent manner [5,6]. UVB, with a shorter wavelength, reaches the superficial layers of the skin, down to the basal layer of the epidermis. UVA radiation, conversely, penetrates deeper, extending into the dermis [7]. These distinct wavelength radiations have specific as well as overlapping and synergistic effects on the skin [8]. Exposure to UVA radiation results in indirect photosensitizing reactions, leading to the production of reactive oxygen and nitrogen species (ROS and RNS). These species can damage macromolecules like DNA, proteins, and lipids. UVB absorption by epidermal cells causes direct DNA damage and elevates oxidative stress. Specifically, UVB exposure activates the nuclear factor  $\kappa$ B (NF- $\kappa$ B) pathway in keratinocytes. This activation leads to the release of various pro-inflammatory mediators, including cytokines like interleukin  $1\beta$  (IL- $1\beta$ ), interleukin 6 (IL-6), and tumor necrosis factor alpha (TNF- $\alpha$ ), along with neuropeptides, histamine, prostaglandins, serotonin, and oxygen radicals [9,10]. Skin exposure to UVR collectively leads to inflammation, which is visibly apparent as erythema [9].

Historically, skin photoprotection has primarily relied on topical sunscreen products containing molecules capable of absorbing, reflecting or scattering UVR [11–15]. With the progression of global warming and the associated rise in the frequency and duration of intense solar radiation events worldwide, the need for effective photoprotection and consistent sunscreens application is expected to become increasingly critical. Nevertheless, despite the growing use of sunscreens, the global incidence of cutaneous melanoma continues to rise, suggesting that current preventive strategies may be insufficient or inconsistently applied [16]. Moreover, the protective efficacy of sunscreens is often undermined by limited public awareness of the risks associated with unintentional exposure to UVR, as well as the common misperception that temperature and cloud cover accurately reflect UV intensity [17].

Oral photoprotective agents therefore represent a promising adjunctive strategy for enhancing protection against UVR-induced damage [18]. Unlike topical photoprotection, which requires application prior to sun exposure and frequent reapplication, the oral photoprotective approach offers the advantage of enhancing the skin's physiological response to UVR while providing systemic and preventive benefits [19]. These oral photoprotective products usually contain one or more bioactives, aimed at counteracting the depletion of endogenous antioxidants following ultraviolet radiation (UVR) exposure [18,20–22]. Key photoprotective dietary compounds are recognized for their antioxidant, anti-inflammatory, and immunomodulatory properties, collectively boosting the skin's inherent resilience to UV-induced damage and photoaging [18,19]. These bioactive compounds include carotenoids and polyphenols. Carotenoids are potent antioxidants that inhibit the formation of ROS and reduce erythema, though they often necessitate high-dose, long-term administration. Polyphenols, on the other hand, neutralize free radicals and regulate inflammatory pathways. Additionally, vitamins such as C and E act synergistically to quench free radicals, while nicotinamide and vitamin D support DNA repair and modulate immune responses.

Among food bioactives, essential fatty acids are key components of cell membranes, playing a crucial role in maintaining the epidermal barrier function [23]. As the human body cannot synthesize them, unsaturated fatty acids must be obtained through the diet. These fatty acids have demonstrated anti-inflammatory properties and the ability to enhance skin repair processes [24]. In this study, we aimed to investigate the photoprotective efficacy of supplementation with a standardized coriander (*Coriandrum sativum* L.) seed oil (CSO). Coriander, a medicinal herb originating in the Near East and Mediterranean, is widely recognized for its various biological activities. These activities notably include antioxidant, antimicrobial, and anti-inflammatory properties [25]. CSO is

a virgin oil of coriander seeds, sourced in the southwestern area of France, and obtained by mechanical pressing using twin-screw extrusion technology, a gentle eco-extraction process without solvent to ensure the protection of the bioactive compounds. CSO is primarily composed of petroselinic acid (65–80% of total fatty acids), a monounsaturated omega-12 positional isomer of oleic acid with demonstrated anti-inflammatory properties [25,26]. Petroselinic fatty acid is considered a rare fatty acid characteristic of the lipid fraction of the Apiaceae family plants [27]. Practically absent in the leaves and other parts of these plants, it constitutes about 80% of the total fatty acids in their seeds. The most significant dietary sources of petroselinic acid include the seed oils of Coriander (*Coriandrum sativum* L.), Anise (*Pimpinella anisum*), Fennel (*Foeniculum vulgare*), Parsley (*Petroselinum crispum*), Caraway (*Carum carvi*), Celery (*Apium graveolens*), Cumin (*Cuminum cyminum*), and Dill (*Anethum graveolens*). CSO is also a natural source of linoleic acid and its derivatives (13–16% of total fatty acids) and phytosterols, like stigmasterol (21–30% of total sterols) and  $\beta$ -sitosterol (24–37% of total sterols). It also contains tocopherols, especially  $\gamma$ -tocotrienol (>70% of total tocopherols). Petroselinic acid-rich CSO has been previously shown to mitigate skin reactivity in healthy women with sensitive skin, likely due to its ability to temper inflammatory responses [28].

To investigate the systemic effects of CSO on key biophysical parameters of skin photoprotection, two independent randomized, placebo-controlled clinical trials were conducted on women with normal or reactive skin. Specifically, the studies assessed the effectiveness of oral CSO supplementation in preventing UV-induced skin erythema. The underlying mechanism of action was explored by quantifying the release of TNF- $\alpha$  and malondialdehyde by skin cells.

## 2. Materials and Methods

### 2.1. Trials Design

The photoprotective efficacy of CSO was evaluated through two independent clinical studies. Both studies were randomized, parallel-group, double-blind, placebo-controlled trials conducted at Complife Italia S.r.l. facilities (San Martino Siccomario, Italy). The first trial, involving women with reactive skin, was carried out from October 2021 to January 2022. The second trial, which included women of various skin types, was performed from September 2024 to January 2025. The protocols were approved by an independent ethics clinical investigation committee (Società Scientifica Italiana per le Indagini Cliniche Non Farmacologiche) in July 2021 (ref. no. 2021/04) and June 2024 (ref. no. 2024/06). Both trials were conducted in full compliance with the principles of the 1975 Declaration of Helsinki revised in 2013. Before recruitment, all participants received detailed information and provided written informed consent. The trials protocols were registered on [www.clinicaltrials.gov](https://www.clinicaltrials.gov) under NCT06037291 ([https://www.clinicaltrials.gov/study/NCT06037291?term=AREA%5BBasicSearch%5D\(CORIANDE\)&rank=3](https://www.clinicaltrials.gov/study/NCT06037291?term=AREA%5BBasicSearch%5D(CORIANDE)&rank=3); accessed on 6 November 2025) and NCT06571409 ([https://www.clinicaltrials.gov/study/NCT06571409?term=AREA%5BBasicSearch%5D\(AREA%5BBasicSearch%5D\(CORIANDE\)\)&rank=4](https://www.clinicaltrials.gov/study/NCT06571409?term=AREA%5BBasicSearch%5D(AREA%5BBasicSearch%5D(CORIANDE))&rank=4); accessed on 6 November 2025).

Subjects attended clinic visit at baseline (D0) and after 56 days (D56) of product use. Skin erythema and inflammation were measured 24 h after UVA+B exposure at D0 and D56 in both trials, while skin lipoperoxidation was measured 4 and 24 h after UVA exposure at D0 and D56 in the second trial. A supplementation period of 56 days is a standard timeframe used in clinical trials evaluating the effects of food supplements on skin biophysical parameters and UV-induced damage, encompassing the kinetics of skin cell turnover.

### 2.2. Interventions and Randomization

Participants received either coriander seed oil (CSO) or a placebo in visually indistinguishable, opaque soft gelatin (bovine) capsules. The active capsules were filled with 200 mg of CSO (Sepibliss™, Seppic, La Garenne Colombes, France), a pure virgin oil produced by mechanical pressing of *Coriandrum sativum* L. seeds. This oil is notable for its high content of petroselinic acid (60–75%), alongside linoleic acid (12–19%) and oleic acid (7–15%). A daily dose of 200 mg of CSO was previously shown to be safe and effective in a previous pilot clinical study investigating CSO's benefits on sensitive skin [28]. The placebo capsules contained 200 mg of a standardized sunflower oil, characterized by 50–72% linoleic acid and 15–85% oleic acid. Sunflower oil is a comparable fatty acid-based vehicle that is visually and texturally indistinguishable from CSO, but crucially lacks petroselinic acid, the key active component of CSO. Participants took one capsule with breakfast each morning throughout the 56-day study period.

Randomization into the CSO or placebo group was achieved using a restricted and balanced 1:1 ratio list. This list was computer-generated using the “Wey’s urn” algorithm (PASS 11, version 11.0.8, PASS, LLC, Kaysville, UT, USA). The group assignments were kept concealed throughout the study in sequentially numbered, sealed, opaque envelopes. A double-blind approach was maintained, ensuring that participants, investigators, and collaborators remained unaware of the group assignments.

### 2.3. Sample Size, Participants and Compliance with Treatment

The sample size was calculated based on previously published clinical studies evaluating the effects of nutraceuticals on UVR-induced skin damage [20,22]. These studies indicate that a sample size of approximately 30 participants per group is generally sufficient to detect clinically meaningful differences with adequate statistical power. In alignment with these findings, and to account for an anticipated dropout rate, we enrolled 33 subjects per treatment arm in the cohort comprising all skin types, and 40 subjects per treatment arm in the reactive skin cohort.

The first trial included healthy women, aged 18–65, with phototypes I–IV and reactive skin. Skin reactivity was confirmed by a moderate to intense stinging sensation after the topical lactic acid application [29]. A comprehensive list of inclusion and exclusion criteria can be found in the Supplementary Materials (Table S1). The second trial enrolled healthy women aged between 35 and 65 years old with phototype I to III and all skin types. The complete inclusion and exclusion list is reported in the Supplementary Materials (Table S2). Treatment compliance was assessed by the principal investigator through counting the remaining pills in each bottle after 56 days of treatment. The average overall compliance rate was required to be ≥80%. Moreover, participants were asked to fill in a daily diary specifying their daily food and drink consumption during the first 14 days of the trial to ensure the stability of their dietary habits.

### 2.4. Outcomes Assessment

All outcomes were assessed under controlled environmental conditions (temperature:  $22 \pm 4$  °C, room humidity:  $50 \pm 10\%$ ). Prior to any measurement, volunteers underwent a 15–20 min acclimatization period to these ambient conditions.

#### 2.4.1. UVA+B Exposure and Measurement of Erythema Index

Skin erythema was induced at D0 and D56 by exposing selected skin areas of the volunteers’ back (surface of  $0.8 \times 0.8$  cm) to UVA + UVB radiation using a Multiport 601–300 W Solar simulator (Solar Light Company, LLC, Glenside, PA, USA). The radiation dose was standardized to 1.5 times the minimal erythema dose (MED). The MED is defined as the lowest dose of ultraviolet radiation that results in the first clearly visible, unambiguous erythema with defined borders appearing across the majority of the

exposed area, observed  $20 \pm 4$  h post-exposure. A PMA 2100 radiometer equipped with a PMA 2103 biologically weighted erythema sensor (Solar Light Company, LLC, PA, USA) was used to adjust this dose. The solar simulator's spectral output adhered to the specifications for assessing the sun protection factor (SPF) of sunscreens, as outlined in ISO 24444 [30].

Erythema (hemoglobin content) was quantified 24 h post-UV exposure using a Mexameter® MX 18 (Courage + Khazaka Electronic GmbH, Cologne, Germany). The skin erythema index was then determined by subtracting the measurement taken on the non-irradiated skin area from the measurement on the adjacent irradiated area. The maximal inflammatory response, typically peaking between 12 and 24 h after UV exposure [20], was therefore captured by the 24 h time point measurement.

#### 2.4.2. Quantification of TNF- $\alpha$ After UVA+B Exposure

For TNF- $\alpha$  quantification, 20 out of 40 participants per group from the reactive skin cohort and 22 out of 33 participants per group from the all-skin types cohort were randomly selected. At D0 and D56, ten tape strippings (Corneofix®, Courage + Khazaka Electronic GmbH, Cologne, Germany) were collected from a  $2\text{ cm} \times 2\text{ cm}$  skin surface 24 h post-UV exposure. Strippings were taken from both the irradiated area (used for erythema index evaluation) and an adjacent non-irradiated area, then stored at  $-80\text{ }^{\circ}\text{C}$ . TNF- $\alpha$  in the pooled extraction solution from these ten strippings per participant was quantified by ELISA (EliKine™ Human TNF- $\alpha$  ELISA Kit, Abbkine, Atlanta, GA, USA) according to the manufacturer instructions. The analysis focused on UV-induced TNF- $\alpha$  variation, calculated by subtracting the non-irradiated area's value from the irradiated area's value.

#### 2.4.3. UVA Exposure and Measurement of Lipoperoxidation

UV-induced skin lipoperoxidation was analyzed in 22 out of the 33 participants per group in the cohort of volunteers with all skin types, selected by a randomization list.

At D0 and D56, selected skin areas of the volunteers' back (surface of  $0.8 \times 0.8\text{ cm}$ ) were exposed to UVA exposure using a Multiport 601–300W Solar simulator (Solar Light Company, LLC, PA, USA). These areas were distinct from those used for UVA+B irradiation. The radiation dose was adjusted to  $5\text{ J/cm}^2$  using a PMA 2100 radiometer equipped with a PMA 2113 UVA sensor (Solar Light Company, LLC, PA, USA). The spectral output of the solar simulator complied with the specifications outlined in ISO 24442 for the assessment of UVA protection factor (UVA PF) of sunscreens [31].

At D0 and D56, ten tape strippings (Corneofix®, Courage + Khazaka Electronic GmbH, Cologne, Germany) were collected from a  $2\text{ cm} \times 2\text{ cm}$  skin surface 4 h and 24 h post-UV exposure. These time points were selected to capture both the early oxidative burst and the later, sustained oxidative response. Strippings were taken from both the irradiated area and an adjacent non-irradiated area. The first eight strippings were discarded, while stripping no. 9 and 10 were collected and stored at  $-80\text{ }^{\circ}\text{C}$ . The malondialdehyde (MDA) assay was used to measure the oxidative damage to cellular lipids. The MDA assay relies on the ability of the chromogen N-methyl-2-phenylindole (NMPI) to react with MDA at  $45\text{ }^{\circ}\text{C}$  in acidic conditions, forming a stable chromophore with an absorption peak between 540 and 590 nm [32]. Each well of a 12-well plate was filled with 500  $\mu\text{L}$  of a 0.5 mmol/L  $\text{CuSO}_4$  aqueous solution and incubated at  $37\text{ }^{\circ}\text{C}$  in a microplate incubator/shaker under continuous agitation for 1 h. After incubation, 1.3 mL of R1 solution (2.13 mg NMPI dissolved in 1 mL acetonitrile) and 0.3 mL of 37% HCl were added. Samples were then incubated at  $45\text{ }^{\circ}\text{C}$  for an additional 60 min under constant agitation. The reaction was quenched by placing the samples on ice for 10 min, followed by 10 min at room temperature. Subsequently, 1 mL of the mixture was centrifuged at

120× g for 10 min. Absorbance was measured at 586 nm. A standard calibration curve with increasing MDA concentrations was employed for quantification. The analysis focused on UV-induced MDA variation, calculated by subtracting the non-irradiated area's value from the irradiated area's value.

#### 2.4.4. Self-Assessment Questionnaire

Upon study completion (D56), participants were asked to evaluate the tested products via a questionnaire. This assessment, administered before any objective outcome measurements to prevent bias, captured their perceptions of the product's efficacy on skin condition, including redness, reactivity, and sun sensitivity. Answers were recorded on a 4-point grading scale (completely agree, agree, disagree and completely disagree) and results are presented as the percentage of participants in agreement.

#### 2.5. Statistical Analysis

All participants in the reactive skin cohort completed the study, while two volunteers in the CSO group of the all-skin types cohort discontinued their participation. Therefore, the results reported herein reflect a per-protocol (PP) analysis, including all randomized subjects who completed the entire study.

UV-induced variations in erythema index, TNF- $\alpha$  concentration, and MDA concentration were computed for each participant and measurement time. Subsequently, group-specific means and standard errors of the mean (SEM) were calculated for each parameter at each time point. Intra-group comparisons for erythema index and TNF- $\alpha$  concentration employed either a paired *t*-test or Wilcoxon matched-pairs signed-rank test, depending on data normality. Similarly, inter-group comparisons used an unpaired *t*-test or Mann–Whitney test. For MDA concentration, intra-group statistical assessment involved one-way ANOVA with subsequent Šidák's correction when data met normality assumptions; otherwise, the Kruskal–Wallis test followed by Dunn's post hoc test was employed. Variations were considered statistically significant when *p* value was <0.05. All statistical analyses were performed using GraphPad Prism 10.1.2 software (GraphPad Software, Boston, MA, USA).

### 3. Results

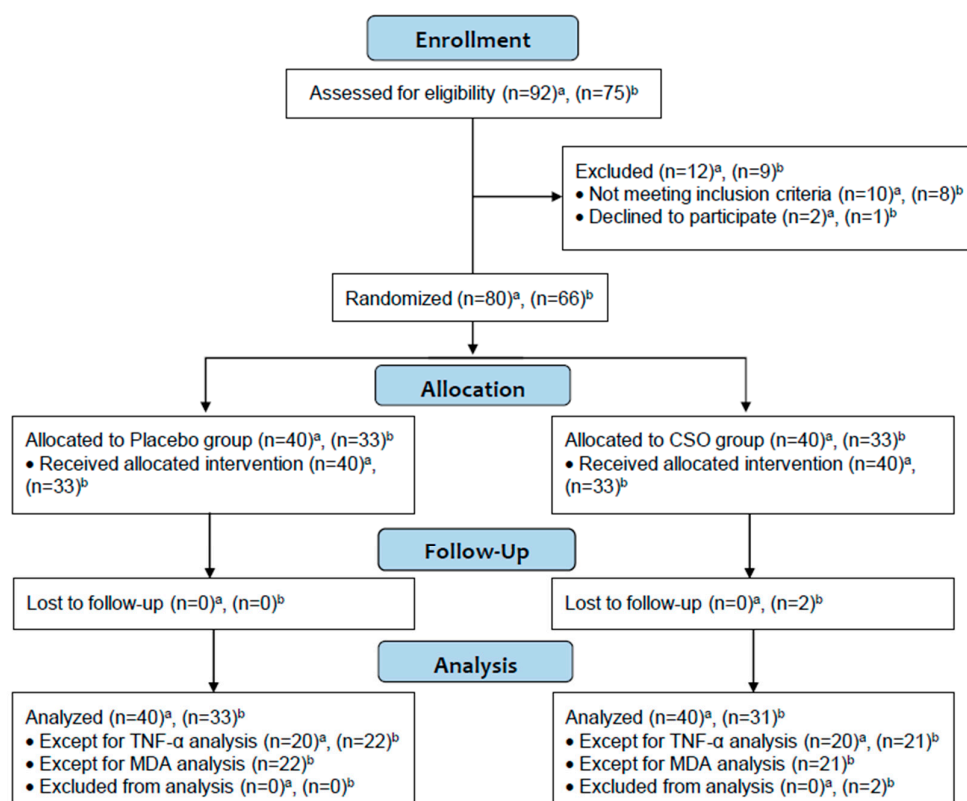
#### 3.1. Participants Characteristics, Tolerability and Compliance with Treatment

In the first trial (cohort with reactive skin), 92 volunteers were screened; 10 were excluded for not meeting the inclusion criteria and 2 volunteers declined to participate. A total of 40 participants were then randomized in the CSO and in the placebo group (Figure 1). The second trial (cohort with all skin types) screened 75 volunteers with 8 volunteers being excluded for not meeting the inclusion criteria and 1 volunteer declined to participate. This trial randomized a total of 33 participants in the CSO and in the placebo group. Two participants dropped out in the CSO group in the second trial (Figure 1).

Baseline characteristics, encompassing key demographic variables such as age, Fitzpatrick phototype, and skin type, are detailed for both experimental cohorts across the two clinical trials in Table 1. These data confirm the initial homogeneity between groups and the absence of potential confounding variables that could influence the interpretation of study outcomes.

All investigational products demonstrated excellent tolerability, with no adverse events or skin reactions reported by either clinical investigators or study participants throughout the entire duration of the trial.

Treatment compliance exceeded 95% in both trials and across both the CSO and placebo groups. This high adherence rate reflects the favorable tolerability profile of the tested formulations.



**Figure 1.** Participants flow diagram. <sup>a</sup> Cohort with reactive skin, <sup>b</sup> Cohort with all skin types.

**Table 1.** Demographics and baseline characteristics of study participants.

|                             | Reactive Skin |             | All Skin Types |              |
|-----------------------------|---------------|-------------|----------------|--------------|
|                             | PL            | CSO         | PL             | CSO          |
| Age [years]                 | 42.9 ± 2.2    | 41.0 ± 2.3  | 51.4 ± 1.2     | 53.3 ± 1.3   |
| Phototype [% (no.)]         |               |             |                |              |
| I                           | -             | -           | 3.0% (1)       | -            |
| II                          | 27.5% (11)    | 32.5% (13)  | 6.0% (2)       | 25.8% (8)    |
| III                         | 40.0% (16)    | 60.0% (24)  | 91.0% (30)     | 74.2% (23)   |
| IV                          | 32.5% (13)    | 7.5% (3)    | -              | -            |
| Skin erythema index [a.u.]  | 124.6 ± 5.8   | 133.6 ± 7.8 | 140.2 ± 10.0   | 127.0 ± 10.8 |
| TNF-α concentration [pg/mL] | 2.34 ± 0.22   | 2.18 ± 0.19 | 2.01 ± 0.07    | 2.00 ± 0.09  |
| MDA concentration [μM]      |               |             |                |              |
| Basal                       | n.d.          | n.d.        | 2.46 ± 0.07    | 2.47 ± 0.07  |
| 4 h post UV stress          | n.d.          | n.d.        | 4.15 ± 0.16    | 4.13 ± 0.14  |
| 24 h post UV stress         | n.d.          | n.d.        | 3.29 ± 0.11    | 3.15 ± 0.12  |

Continuous data are expressed as mean ± SEM; categorical data are expressed as counts and percentages. n.d. Not determined. PL: placebo, CSO: Coriander Seed Oil.

### 3.2. Skin Response to UVA+B-Induced Stress

In women with reactive skin, the erythema index significantly decreased by 11.8% in the CSO group after 56 days of supplementation, whereas it significantly increased by 13.2% in the placebo group (Table 2, Figure 2a). The variation in erythema index was significantly lower in the CSO group compared to the placebo group. Similarly, following CSO supplementation, UV-induced TNF- $\alpha$  levels significantly decreased by 24.1% in the CSO group, while a slight, non-significant increase of 5.2% was observed in the placebo group (Table 2, Figure 2c). The reduction in TNF- $\alpha$  levels observed in the CSO group was statistically significant compared to the placebo group.

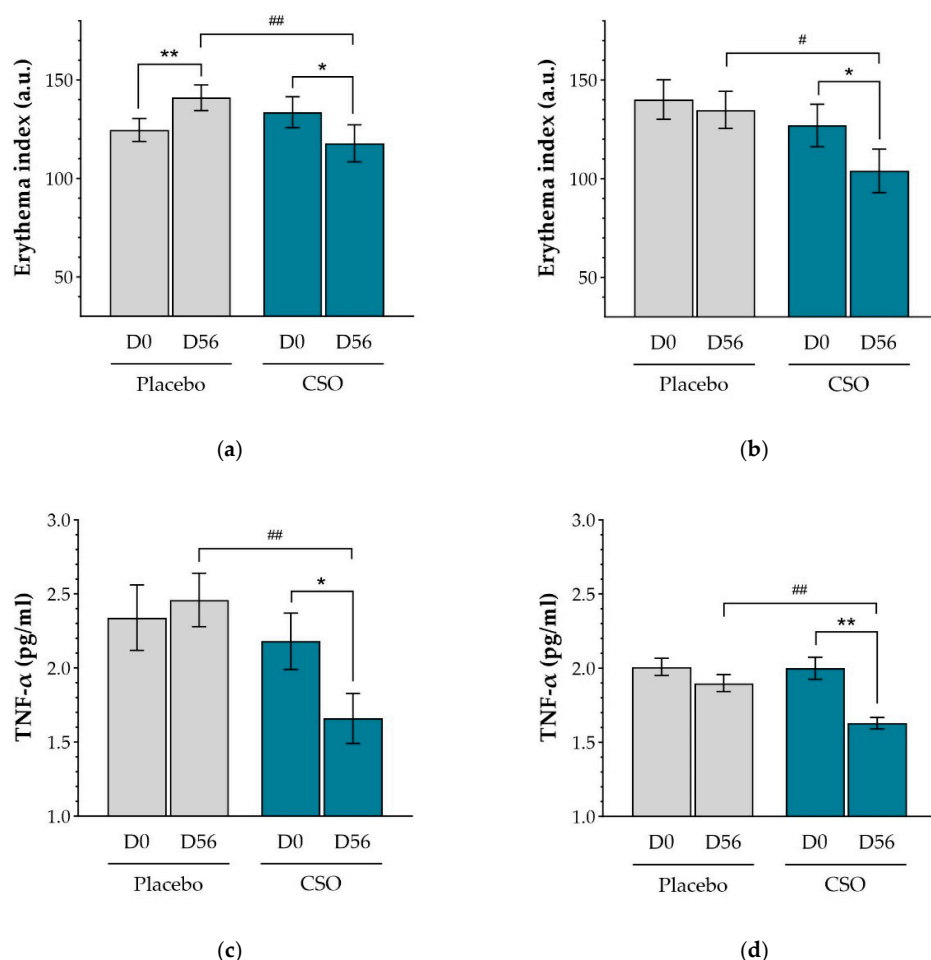
In women with various skin types, the erythema index significantly decreased by 18.1% in the CSO group at D56, while a slight, non-significant reduction of 3.8% was observed in the placebo group (Table 2, Figure 2b). The difference in erythema index between the CSO and placebo groups was statistically significant. Similar results were observed for TNF- $\alpha$  levels, which significantly decreased by 18.7% in the CSO group while it was slightly but not significantly decreased by 5.5% in the placebo group (Table 2, Figure 2d). The difference in TNF- $\alpha$  levels between the CSO and placebo groups was statistically significant.

**Table 2.** Erythema index and TNF- $\alpha$  release following UVA+B exposure.

|                          |                |                      | D0               | D56 ( $\Delta\%$ vs. D0)<br><i>p</i> vs. D0   | <i>p</i> vs. PL  |
|--------------------------|----------------|----------------------|------------------|-----------------------------------------------|------------------|
| Erythema index<br>(a.u.) | Reactive skin  | PL ( <i>n</i> = 40)  | 124.6 $\pm$ 5.8  | 141.0 $\pm$ 6.6 (+13.2%)<br><i>p</i> = 0.004  | <i>p</i> = 0.006 |
|                          |                | CSO ( <i>n</i> = 40) | 133.6 $\pm$ 7.8  | 117.8 $\pm$ 9.4 (−11.8%)<br><i>p</i> = 0.021  |                  |
|                          | All skin types | PL ( <i>n</i> = 33)  | 140.2 $\pm$ 10.0 | 134.9 $\pm$ 9.4 (−3.8%)<br>ns                 | <i>p</i> = 0.013 |
|                          |                | CSO ( <i>n</i> = 31) | 127.0 $\pm$ 10.8 | 104.0 $\pm$ 11.1 (−18.1%)<br><i>p</i> = 0.033 |                  |
| TNF- $\alpha$<br>(pg/mL) | Reactive skin  | PL ( <i>n</i> = 20)  | 2.34 $\pm$ 0.22  | 2.46 $\pm$ 0.18 (+5.2%)<br>ns                 | <i>p</i> = 0.003 |
|                          |                | CSO ( <i>n</i> = 20) | 2.18 $\pm$ 0.19  | 1.66 $\pm$ 0.17 (−24.1%)<br><i>p</i> = 0.037  |                  |
|                          | All skin types | PL ( <i>n</i> = 22)  | 2.01 $\pm$ 0.07  | 1.90 $\pm$ 0.07 (−5.5%)<br>ns                 | <i>p</i> = 0.002 |
|                          |                | CSO ( <i>n</i> = 21) | 2.00 $\pm$ 0.09  | 1.63 $\pm$ 0.05 (−18.7%)<br><i>p</i> = 0.003  |                  |

Data are expressed as mean  $\pm$  SEM. PL: placebo, CSO: Coriander Seed Oil. ns: not significant.

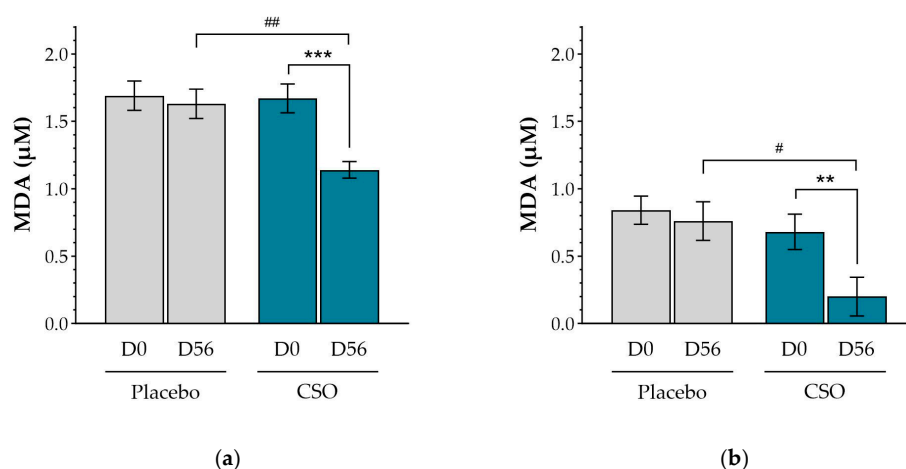




**Figure 2.** Skin response to UVA+B-induced stress. Erythema index in (a) women with reactive skin and (b) women of various skin types, TNF- $\alpha$  release in (c) women with reactive skin and (d) women of various skin types. Data are expressed as mean  $\pm$  SEM. The intragroup (vs. baseline) statistical analysis is denoted by the symbol \*, while the intergroup (CSO vs. placebo) statistical analysis is denoted by the symbol #, as follows: \*/#  $p < 0.05$ , \*\*/#  $p < 0.01$ .

### 3.3. Skin Antioxidant Capacity (Cohort with All Skin Types)

At baseline, UVA exposure resulted in a marked increase in lipoperoxidation in both groups, as reflected by elevated MDA levels (Figure 3). At D56, UVA-induced lipoperoxidation was significantly reduced in the CSO group at both T4h and T24h (−31.9% and −69.9% vs. baseline, respectively), whereas a slight, not significant reduction was observed in the placebo group (−3.7% at T4h and −8.8% at T24h, Table 3). Differences between the CSO and placebo groups were statistically significant both at T4h ( $p < 0.01$ ) and T24h ( $p < 0.05$ ).



**Figure 3.** MDA concentration at (a) 4 h and (b) 24 h after UV exposure. Data are expressed as mean  $\pm$  SEM. The intragroup (vs. baseline) statistical analysis is denoted by the symbol \*, while the intergroup (CSO vs. placebo) statistical analysis is denoted by the symbol #, as follows: #  $p < 0.05$ , \*\*/##  $p < 0.01$ , \*\*\*  $p < 0.001$ .

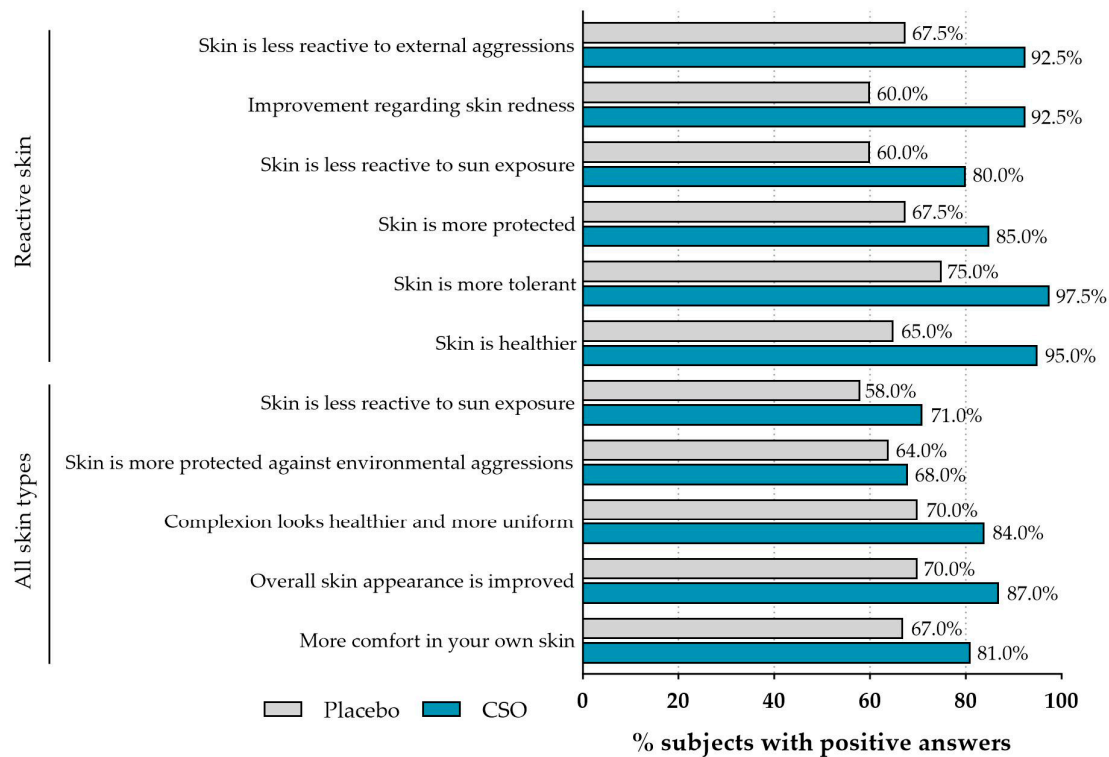
**Table 3.** MDA concentration following UVA exposure.

|                  | D0              |                 | D56                                     |             |                                         |             |
|------------------|-----------------|-----------------|-----------------------------------------|-------------|-----------------------------------------|-------------|
|                  | 4 h             | 24 h            | 4 h ( $\Delta\%$ vs. D0)<br>$p$ vs. D0  | $p$ vs. PL  | 24 h ( $\Delta\%$ vs. D0)<br>$p$ vs. D0 | $p$ vs. PL  |
| PL ( $n = 22$ )  | 1.69 $\pm$ 0.11 | 0.84 $\pm$ 0.10 | 1.63 $\pm$ 0.11 (−3.7%)<br>ns           |             | 0.76 $\pm$ 0.14 (−8.8%)<br>ns           |             |
| CSO ( $n = 21$ ) | 1.67 $\pm$ 0.11 | 0.68 $\pm$ 0.13 | 1.14 $\pm$ 0.06 (−31.9%)<br>$p < 0.001$ | $p = 0.002$ | 0.20 $\pm$ 0.14 (−69.9%)<br>$p = 0.001$ | $p = 0.011$ |

Data are expressed as mean  $\pm$  SEM. PL: placebo, CSO: Coriander Seed Oil. ns: not significant.

### 3.4. Self-Assessment Questionnaire

The efficacy of CSO supplementation in mitigating UV-induced skin alterations was not only demonstrated through objective clinical and biochemical markers but also perceived subjectively by participants, as illustrated in Figure 4. A higher proportion of individuals in the CSO group reported improvements in skin condition, including reduced redness, reactivity to external aggressions, and sensitivity to sun exposure, compared to those in the placebo group.



**Figure 4.** Perceived Skin Benefits.

#### 4. Discussion

Solar ultraviolet radiation (UVR) is one of the most important environmental factors influencing skin physiology. Rising UVR levels, primarily due to climate change, compromise skin health by intensifying biomechanical damage and diminishing the skin's inherent resilience, thereby impairing its vital barrier function [33].

The increasing awareness of the limitations of topical sunscreens, such as inadequate application, short duration of effectiveness, and user non-compliance, has prompted growing interest in systemic approaches to photoprotection [18,34–36]. Oral photoprotectors offer the advantage of providing uniform, full-body coverage and continuous protection, independent of user behavior. These products exert their photoprotective efficacy through multiple mechanisms of action, targeting diverse cellular and molecular signaling pathways. Their activity encompasses the scavenging of reactive oxygen species, suppression of pro-inflammatory cytokines, and modulation of immune responses, collectively contributing to antioxidant, anti-inflammatory, and immunomodulatory effects at the skin level [19,37]. In particular, unsaturated fatty acids are gaining attention for their potential in maintaining skin health and addressing various skin disorders, particularly those mediated by UVR [24,38].

The findings of this study provide clinical evidence supporting the efficacy of oral supplementation with standardized coriander seed oil (CSO) in mitigating UV-induced skin damage. Compared with a placebo, supplementation with CSO for 56 days significantly reduced UVA+B-induced skin erythema and the associated release of TNF- $\alpha$ , demonstrating notable anti-inflammatory properties. Women with reactive skin showed an increased erythematous response in the placebo group, which confirms their heightened susceptibility to skin damage caused by UV radiation [7]. Interestingly, CSO supplementation led to a comparable reduction of about 12–18% of UV-induced skin

erythema in both study populations, reinforcing the systemic photoprotective potential of CSO regardless of baseline skin sensitivity.

In addition to attenuating erythema and inflammation, CSO supplementation demonstrated a significant effect in improving the skin antioxidant capacity, as evidenced by a strong decrease in skin lipoperoxidation. After 56 days of supplementation, while no significant change was observed in the placebo group, UVA-induced MDA levels were strongly reduced by about 32% and 70% after 4 h and 24 h of exposure, respectively, in women with various skin types. Products of lipoperoxidation, such as MDA, have been demonstrated to induce nucleic acid and protein oxidation, further increasing oxidative stress [8]. These compounds also enhance inflammation by stimulating pro-inflammatory genes and producing cytokines, and significantly contribute to the pathogenesis of various skin diseases [39].

These outcomes align with prior reports suggesting that oral food bioactives can modulate erythematous responses by mitigating UV-induced skin redness [20,22,40]. In particular, a recent study showed a significant reduction in UVB-induced erythema intensity following oral supplementation with a blend of *Polypodium leucotomos* extract, red orange extract and vitamins A, C, D, and E. These benefits were attributed to the combined antioxidant and anti-inflammatory properties of the polyphenols and the supporting vitamins [22]. Reductions in the pro-inflammatory cytokine TNF- $\alpha$  further support the anti-inflammatory properties of CSO and suggest a potential role in modulating cytokine-mediated signaling pathways involved in UV-induced skin inflammation. These findings are in accordance with a previous in vitro study reporting the beneficial role of CSO in reducing NF- $\kappa$ B activation mediated by TNF- $\alpha$  [41]. In this study, CSO has also been demonstrated to regulate the activation of TRPA1 (transient receptor potential ankyrin 1), a sensor playing a functional role in nociception and neurogenic inflammation. TRPA1 is already well-recognized for its role in the pathophysiology of sunburn caused by UVB radiation [42].

Self-assessment data corroborated the objective findings, with a higher proportion of participants in the CSO groups reporting improved skin comfort, reduced reactivity, and decreased sun sensitivity, especially in women with reactive skin. These perceptions, collected prior to objective assessments, suggest a real-world benefit of the intervention that complements the biochemical and clinical outcomes.

CSO's photoprotective properties are primarily attributed to petroselinic acid, a rare fatty acid with potential anti-inflammatory effects [25,27,28]. In rodents, petroselinic acid was shown to be absorbed and incorporated in tissue lipids, allowing it to exert a systemic anti-inflammatory action. Indeed, the ingestion of coriander oil has been shown to significantly decrease levels of arachidonic acid, a polyunsaturated fatty acid released following irritation or injury and known to be involved in activating inflammatory pathways [26]. CSO is also a natural source of beneficial compounds, including phytosterols like  $\beta$ -sitosterol and stigmasterol, both known for their anti-inflammatory effects [25,43]. Furthermore, CSO contains vitamin E, specifically the  $\gamma$ -tocotrienol form. Dietary supplementation with tocotrienols enhances the body's antioxidant defenses, thereby protecting the skin from oxidative stress caused by environmental aggressors [24,27]. These molecules also exhibit potent anti-inflammatory properties by suppressing the expression of pro-inflammatory cytokines, such as TNF- $\alpha$  [44].

To the best of our knowledge, this study is the first to evaluate the photoprotective efficacy of *Coriandrum sativum* L. seed oil in a controlled clinical setting using a robust, double-blind, randomized, placebo-controlled design. The use of two distinct but complementary cohorts allowed for a broader assessment of CSO's efficacy across different skin phenotypes. Furthermore, the study employed a multi-endpoint evaluation strategy, incorporating objective clinical (erythema index), biochemical (TNF- $\alpha$  and MDA

quantification), and subjective (self-assessment questionnaire) outcomes. This comprehensive approach strengthens the internal validity of the findings and provides mechanistic insight into CSO's anti-inflammatory and antioxidant actions. Additionally, high treatment compliance and absence of adverse events underscore the product's favorable tolerability and feasibility for long-term use. Nevertheless, while CSO's effectiveness has been consistently shown in these two studies, future clinical trials should include a larger subject pool to validate its photoprotective potential.

## 5. Conclusions

The findings of this study provide novel clinical evidence supporting the photoprotective efficacy of oral supplementation with standardized coriander seed oil, particularly in mitigating UV-induced skin damage. Across two randomized, placebo-controlled trials involving women with both normal and reactive skin types, CSO demonstrated significant reductions in erythema formation, pro-inflammatory cytokine (TNF- $\alpha$ ) expression, and lipid peroxidation (MDA levels), underscoring its anti-inflammatory and antioxidant properties. These results suggest that CSO, which is rich in petroselinic acid and other bioactive fatty acids, may represent a promising adjunctive strategy for systemic photoprotection. While further studies are warranted to confirm long-term benefits and to explore molecular mechanisms in greater depth, this investigation offers a solid foundation for the integration of CSO into holistic approaches aimed at reinforcing the skin's resilience to environmental stressors.

**Supplementary Materials:** The following supporting information can be downloaded at <https://www.mdpi.com/article/10.3390/cosmetics12060285/s1>, Table S1: Inclusion and exclusion criteria (cohort with reactive skin); Table S2: Inclusion and exclusion criteria (cohort with all skin types).

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**Data Availability Statement:** The data presented in this study are available on request from the corresponding author. The data are not publicly available since they are the property of the sponsor of the study (Seppic, France).

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## Abbreviations

The following abbreviations are used in this manuscript:

|               |                             |
|---------------|-----------------------------|
| CSO           | Coriander seed oil          |
| PL            | Placebo                     |
| TNF- $\alpha$ | tumor necrosis factor-alpha |
| UVR           | Ultraviolet radiation       |
| UVA           | Ultraviolet A               |
| UVB           | Ultraviolet B               |
| UVC           | Ultraviolet C               |
| MDA           | Malondialdehyde             |

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