

Original article

Effects of Ultraviolet-C Radiation on Skin Trace Element Concentrations (Copper, Zinc, and Iron) in Wistar Rats: An Atomic Absorption Spectrometry Study

Sumaya Alusta*¹ , Eman Alsqyar² , Antisar Ibashouk³ ¹Department of Physics to divide the Physiotherapy Department, Faculty of Health Sciences, Al Ajilat, University of Zawia, Libya²Department of Dermatology and Venereology to divide the Physiotherapy department, Faculty of Health Sciences, Al Ajilat University of Zawia, Libya³Department of Chemistry Science to divide the Physiotherapy department, Faculty of Health Sciences/ Al Ajilat University of Zawia, LibyaCorresponding author email: s.astay@zu.edu.ly

Abstract

Ultraviolet-C (UVC) radiation is a known inducer of oxidative stress and cutaneous injury. However, its specific impact on essential trace element homeostasis in dermal tissue remains inadequately characterized. This study investigated the effects of artificial UVC radiation (254 nm) on the concentrations of copper (Cu), zinc (Zn), and iron (Fe) in the skin of Wistar rats. Forty adult Wistar rats were allocated into one control group and three experimental groups exposed to UVC radiation for 8, 16, or 24 days (8 hours daily). Trace element concentrations in skin homogenates were quantified using atomic absorption spectrometry (AAS). Statistical comparisons between control and exposed groups were performed using Student's *t*-test, with significance set at $p < 0.05$. UVC exposure induced a significant, duration-dependent decline in all three trace elements. Compared to controls, Cu levels decreased from 0.20 ± 0.04 mg/g to 0.03 ± 0.01 mg/g ($p < 0.05$), Zn levels from 15.5 ± 1.56 mg/g to 6.32 ± 0.48 mg/g ($p < 0.05$), and Fe levels from 4.66 ± 1.23 mg/g to 0.32 ± 0.12 mg/g ($p < 0.05$) after 24 days. Iron exhibited the most pronounced reduction. Prolonged UVC radiation significantly depletes essential trace elements in rat skin in a time-dependent manner. These findings provide quantitative evidence for UVC-induced micronutrient dyshomeostasis, which may contribute to UV-mediated dermatological pathology.

Keywords. Ultraviolet C, Trace Elements, Copper, Zinc, Skin.

Received: 14/02/26

Accepted: 11/04/26

Published: 20/04/26

Copyright: Author (s) 2026.

Distributed under Creative Commons CC-BY 4.0

Introduction

Radiation is defined as the emission or propagation of radiant energy in the form of electromagnetic waves or particles. Ultraviolet (UV) radiation, a component of the solar spectrum, is subclassified into UVA (315–400 nm), UVB (280–315 nm), and UVC (100–280 nm) based on wavelength (1). Although terrestrial UVC is almost entirely absorbed by the stratospheric ozone layer, artificial sources such as germicidal lamps are increasingly used in clinical, occupational, and laboratory settings, posing potential health hazards (2). UVC radiation is highly genotoxic, primarily causing the formation of cyclobutane pyrimidine dimers and generating reactive oxygen species (ROS) that induce oxidative stress, protein denaturation, and lipid peroxidation (3). The skin, as the primary interface with the external environment, is particularly vulnerable to these effects.

Trace elements, including copper (Cu), zinc (Zn), and iron (Fe), are essential micronutrients that serve as catalytic cofactors for numerous enzymatic reactions critical to skin integrity. Cu and Zn are integral components of superoxide dismutase (SOD), a key antioxidant enzyme (4). Fe is a central constituent of catalase and cytochrome P450 enzymes, involved in both antioxidant defense and metabolic processes (5). Disruption of trace element homeostasis is implicated in impaired wound healing, increased photosensitivity, and accelerated photoaging (6).

Despite the known deleterious effects of UV radiation, quantitative data regarding its impact on skin trace element concentrations remain limited. This study aimed to evaluate the hypothesis that chronic exposure to artificial UVC radiation (254 nm) significantly alters the concentrations of Cu, Zn, and Fe in rat skin, with effects proportional to exposure duration.

Materials and Methods

Experimental Animals

Forty adult male Wistar rats (*Rattus norvegicus*) weighing approximately 200–250 g were obtained from the institutional animal facility. Animals were housed under standard laboratory conditions ($22 \pm 2^\circ\text{C}$, 12-hour light/dark cycle) with ad libitum access to water and standard pellet diet. All experimental procedures complied with institutional guidelines for animal care and use.

Experimental Design

Rats were randomly assigned to four groups (n = 10 per group):

- **Group I (Control):** No UVC exposure.
- **Group II (UVC-8):** Exposed to UVC radiation for 8 days.
- **Group III (UVC-16):** Exposed to UVC radiation for 16 days.
- **Group IV (UVC-24):** Exposed to UVC radiation for 24 days.

UVC Irradiation Protocol

A germicidal UVC lamp emitting at 254 nm (wavelength peak) was positioned 30 cm above the animals' dorsal skin. The experimental groups received 8 hours of continuous exposure per day for their respective durations. The control group was housed identically but shielded from UVC exposure.

Sample Collection and Trace Element Analysis

Following the completion of each exposure period, animals were euthanized, and full-thickness skin samples (approximately 1 cm²) were excised from the irradiated dorsal region. Samples were digested in acid-washed vessels using a mixture of nitric acid (HNO₃) and hydrogen peroxide (H₂O₂). The concentrations of copper, zinc, and iron (expressed as mg/g wet tissue) were determined using an atomic absorption spectrometer (AAS) with appropriate hollow cathode lamps and calibration standards.

Statistical Analysis

Data were expressed as mean \pm standard deviation (SD). Statistical comparisons between each experimental group and the control group were performed using an unpaired two-tailed Student's t-test. A p-value of less than 0.05 was considered statistically significant. All analyses were conducted using standard statistical software.

Results

Copper Concentration

Exposure to UVC radiation resulted in a progressive, statistically significant reduction in skin copper levels (Table 1). The mean Cu concentration in the control group was 0.20 ± 0.04 mg/g. After 8, 16, and 24 days of UVC exposure, concentrations decreased to 0.11 ± 0.02 mg/g (45% reduction), 0.06 ± 0.01 mg/g (70% reduction), and 0.03 ± 0.01 mg/g (85% reduction), respectively. All differences from control were statistically significant ($p < 0.05$), with t-values of 4.5, 14.0, and 17.0, respectively.

Table 1. Copper concentration (mg/g) in rat skin following UVC exposure.

| Group | Mean \pm SD (mg/g) | Minimum | Maximum | t-value | p-value |
|-------------|----------------------|---------|---------|---------|------------|
| Control | 0.20 ± 0.04 | 0.14 | 0.32 | — | — |
| UVC-8 days | 0.11 ± 0.02 | 0.06 | 0.16 | 4.5 | $< 0.05^*$ |
| UVC-16 days | 0.06 ± 0.01 | 0.04 | 0.10 | 14.0 | $< 0.05^*$ |
| UVC-24 days | 0.03 ± 0.01 | 0.01 | 0.06 | 17.0 | $< 0.05^*$ |

*Statistically significant compared to control (t-test, $p < 0.05$).

Zinc Concentration

A similar duration-dependent decline was observed for zinc (Table 2). The control group exhibited a mean Zn level of 15.5 ± 1.56 mg/g. Following 8, 16, and 24 days of UVC exposure, Zn concentrations decreased to 11.25 ± 0.89 mg/g (27%

reduction), 8.83 ± 0.51 mg/g (43% reduction), and 6.32 ± 0.48 mg/g (59% reduction), respectively. All reductions were statistically significant ($p < 0.05$). The t-values increased progressively (7.4, 12.8, and 18.0), indicating a strong exposure-response relationship.

Table 2. Zinc concentration (mg/g) in rat skin following UVC exposure.

| Group | Mean \pm SD (mg/g) | Minimum | Maximum | t-value | p-value |
|-------------|----------------------|---------|---------|---------|------------|
| Control | 15.5 ± 1.56 | 12.0 | 18.0 | — | — |
| UVC-8 days | 11.25 ± 0.89 | 9.5 | 12.8 | 7.4 | $< 0.05^*$ |
| UVC-16 days | 8.83 ± 0.51 | 7.8 | 9.8 | 12.8 | $< 0.05^*$ |
| UVC-24 days | 6.32 ± 0.48 | 5.5 | 7.0 | 18.0 | $< 0.05^*$ |

*Statistically significant compared to control (t-test, $p < 0.05$).

Iron Concentration

Iron levels exhibited the most pronounced reduction among the three trace elements analyzed (Table 3). The control group mean was 4.66 ± 1.23 mg/g. After 8, 16, and 24 days of UVC exposure, Fe concentrations fell sharply to 2.24 ± 0.23 mg/g (52% reduction), 1.49 ± 0.19 mg/g (68% reduction), and 0.32 ± 0.12 mg/g (93% reduction), respectively. All differences were statistically significant ($p < 0.05$), with t-values of 6.2, 8.1, and 11.1.

Table 3. Iron concentration (mg/g) in rat skin following UVC exposure.

| Group | Mean \pm SD (mg/g) | Minimum | Maximum | t-value | p-value |
|-------------|----------------------|---------|---------|---------|------------|
| Control | 4.66 ± 1.23 | 3.0 | 7.0 | — | — |
| UVC-8 days | 2.24 ± 0.23 | 1.9 | 2.6 | 6.2 | $< 0.05^*$ |
| UVC-16 days | 1.49 ± 0.19 | 1.2 | 1.8 | 8.1 | $< 0.05^*$ |
| UVC-24 days | 0.32 ± 0.12 | 0.17 | 0.50 | 11.1 | $< 0.05^*$ |

*Statistically significant compared to control (t-test, $p < 0.05$).

Discussion

The present study demonstrates that chronic exposure to artificial UVC radiation (254 nm) induces a significant, time-dependent depletion of the essential trace elements copper, zinc, and iron in the skin of Wistar rats. All experimental groups exhibited statistically significant reductions relative to controls ($p < 0.05$), with the most prolonged exposure (24 days) producing the greatest elemental loss. These findings provide quantitative evidence linking UVC radiation duration to cutaneous micronutrient dyshomeostasis.

The observed depletion of trace elements can be mechanistically attributed to UVC-induced oxidative stress. UVC radiation is a potent generator of reactive oxygen species (ROS), including superoxide anions (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals ($\bullet OH$) (3,7). These ROS species directly damage cellular membranes, proteins, and nucleic acids. Critically, the binding sites and storage proteins for trace elements such as metallothioneins (for Zn and Cu) and ferritin (for Fe) are susceptible to oxidative modification, potentially leading to metal release and subsequent excretion or redistribution (8).

Copper and zinc are essential cofactors for the antioxidant enzyme Cu/Zn-superoxide dismutase (SOD1), which catalyzes the dismutation of superoxide radicals (4). Under sustained oxidative challenge, increased ROS production may lead to enhanced consumption or oxidative inactivation of SOD1, resulting in a net loss of its associated metals from the tissue. Similarly, iron is a critical component of catalase, which decomposes H_2O_2 , and of heme-containing cytochromes (5). Prolonged UVC exposure may overwhelm these antioxidant systems, leading to enzymatic degradation and liberation of the metal cofactors.

Iron exhibited the most dramatic reduction (93% loss after 24 days). This finding is particularly noteworthy because iron is highly redox-active and can catalyze the Fenton reaction ($Fe^{2+} + H_2O_2 \rightarrow Fe^{3+} + \bullet OH + OH^-$), generating the highly damaging hydroxyl radical (9). The rapid decline in tissue iron may represent an adaptive protective response: the organism may actively sequester or eliminate iron from chronically irradiated skin to minimize Fenton chemistry and subsequent oxidative amplification (10).

The progressive decline in zinc (59% loss) is also clinically significant. Zinc plays crucial roles in DNA repair, cell proliferation, and the maintenance of epidermal barrier function (11). Its depletion would be expected to impair keratinocyte proliferation and delay repair of UV-induced DNA lesions. Copper depletion (85% loss), while less discussed in the UV literature, may impair collagen cross-linking via lysyl oxidase activity, potentially accelerating photoaging and reducing dermal tensile strength (12).

These results align with previous studies demonstrating UVB-induced reductions in skin antioxidant enzymes (13), but extend the literature by providing specific quantitative data on the elemental losses themselves using AAS. The clear exposure-response relationship observed for all three elements, with t-values increasing progressively from 8 to 24 days, strongly supports a causal link between cumulative UVC dose and trace element depletion. The statistical significance achieved across all experimental groups ($p < 0.05$) further reinforces the robustness of these findings. The use of a control group and consistent exposure conditions strengthens internal validity.

Conclusion

In conclusion, this study provides definitive evidence that exposure to UVC radiation (254 nm) for 8 hours daily over 8 to 24 days causes a statistically significant and duration-dependent decrease in the concentrations of copper, zinc, and iron in the skin of Wistar rats. The effects are most severe after 24 days of exposure, with iron exhibiting the greatest proportional loss (93% reduction). These results confirm the harmful biological effects of UVC radiation on cutaneous trace element homeostasis and highlight the relationship between exposure duration and biochemical changes in the skin. Future research should investigate whether antioxidant or metal supplementation strategies can mitigate these elemental losses and associated dermal pathology.

Conflict of interest. Nil

References

1. Diffey BL. Sources and measurement of ultraviolet radiation. *Methods*. 2002;28(1):4-13.
2. Cutler TD, Zimmerman JJ. Ultraviolet irradiation and the mechanisms underlying its inactivation of infectious agents. *Anim Health Res Rev*. 2011;12(1):15-23.
3. de Jager TL, Cockrell AE, Du Plessis SS. Ultraviolet light induced generation of reactive oxygen species. *Adv Exp Med Biol*. 2017;996:15-23.
4. Fukai T, Ushio-Fukai M. Superoxide dismutases: role in redox signaling, vascular function, and diseases. *Antioxid Redox Signal*. 2011;15(6):1583-606.
5. Dixon SJ, Stockwell BR. The role of iron and reactive oxygen species in cell death. *Nat Chem Biol*. 2014;10(1):9-17.
6. Rostan EF, DeBuys HV, Madey DL, Pinnell SR. Evidence supporting zinc as an important antioxidant for skin. *Int J Dermatol*. 2002;41(9):606-11.
7. Ichihashi M, Ueda M, Budiyanoto A, Bito T, Oka M, Fukunaga M, et al. UV-induced skin damage. *Toxicology*. 2003;189(1-2):21-39.
8. Maret W. The redox biology of redox-inert zinc ions. *Free Radic Biol Med*. 2019;134:311-26.
9. Winterbourn CC. Toxicity of iron and hydrogen peroxide: the Fenton reaction. *Toxicol Lett*. 1995;82-83:969-74.
10. Reelfs O, Tyrrell RM, Pourzand C. Ultraviolet A radiation-induced immediate iron release is a key modulator of the activation of NF- κ B in human skin fibroblasts. *J Invest Dermatol*. 2004;122(6):1440-7.
11. Ogawa Y, Kinoshita M, Shimada S, Kawamura T. Zinc in keratinocytes and langerhans cells: relevance to the epidermal homeostasis. *J Dermatol Sci*. 2018;90(1):3-9.
12. Rucker RB, Kosonen T, Clegg MS, Mitchell AE, Rucker BR, Uriu-Hare JY, et al. Copper, lysyl oxidase, and extracellular matrix protein cross-linking. *Am J Clin Nutr*. 1998;67(5 Suppl):996S-1002S.
13. Shindo Y, Witt E, Han D, Epstein W, Packer L. Enzymic and non-enzymic antioxidants in epidermis and dermis of human skin. *J Invest Dermatol*. 1994;102(1):122-7.