Ind. J. Pharm. Edu. Res., 2026; 60(1s):s350-s357.
https://www.ijper.org

Original Article

Mechanism of Reactive Oxygen Species in the Photo-Cytotoxicity and Photo-Apoptotic Effects of Ciproquin on L929 Cells

Daoud Ali*, Saud Alarifi, Bader Almutairi, Mohammed Hasan Almarzoug, Saad Alakhtani

Department of Zoology, College of Science, King Saud University, Riyadh, SAUDI ARABIA.

ABSTRACT

Background: Photo-unstable drugs cause undesirable clinical side effects by acting as photo-toxin in the skin and other tissue under the exposure of sunshine. Ciproquin is an antibiotic that is used to treat bacterial infections and is a member of the powerful fluoroquinolone medication class. **Objectives:** In this work, we evaluated photochemical characteristics of ciproquin as well as its photocytotoxicity, apoptotic, and oxidative potential on the mouse fibroblast cell line (L929) over a 24-hr period at ambient UVB (1.40 mW/cm^2) intensity. **Materials and Methods:** Study conducted on mouse fibroblast cell line (L929), 24-hr exposure period, Ambient UVB radiation at 1.40 mW/cm^2 intensity, Evaluation of photochemical characteristics, photocytotoxicity, apoptotic, and oxidative potential, Comparison with dark control cells. **Results:** Ciproquin generated Hydroxyl radical ($^{\cdot}OH$), superoxide anion radical ($O_2^{\cdot-}$), and singlet oxygen (1O2) in a dose-dependent manner. Additionally, in comparison to dark control cells, ciproquin decreased reduced Glutathione (GSH) and increased Lipid Peroxide (LPO) levels in UV-B irritated L929 cells. Over a 24-hr period, L929 cells exposed to ambient UVB radiation intensity showed a significant ($p<0.05$) concentration-dependent impact of ciproquin. In L929 cells, it was found that caspase-3 activity and phosphatidylserine translocation were markedly ($p<0.05$) increased. **Conclusion:** The ability of ciproquin to act as both phototoxic and photo-apoptotic under UVB irradiation is thus supported by our findings.

Keywords: Ciproquin, L929 Cells, UVB Rays, Photocytotoxicity.

Correspondence:
Prof. Daoud Ali
Department of Zoology, College of Science, King Saud University, Riyadh-11451, SAUDI ARABIA.
Email: aalidaoud@ksu.edu.sa

Received: 13-06-2025;
Revised: 07-08-2025;
Accepted: 24-10-2025.

INTRODUCTION

Drugs of the fluoroquinolone class are often used in medical procedures and react when exposed to UV radiation from the sun. The class is known as fluoroquinolones because the majority of the drugs now used in treatment have at least one fluorine atom in their chemical composition. The fluoroquinolone most frequently used to treat a range of infections is ciprofloxacin. The safety of using this class of antibiotics in the general population, and particularly in the elderly, must be reevaluated in light of potentially serious toxicities linked to their usage in recent years.[1] Between 0.4% and 2.2% of patients who took ciproquin reported allergic reactions or skin problems. After being exposed to UVA light (320-400 nm), some patients also have phototoxicity effects. Many sunscreens only include UVB blockers, which means they don't prevent UVA rays. Products with UVA blockers, such as Parsol 1789 or octocrylene, may provide some protection against phototoxicity from fluoroquinolones, yet their efficacy in doing so has not been thoroughly evaluated. One of the elements that contributes to the development of negative pharmacological side effects is the UVR.[2] Understanding the consequences of fluoroquinolone drugs for human safety is essential due to their wide range of potential and new applications.[3] The mouse fibroblast (L929) cell line is derived from a clone of normal subcutaneous areolar and adipose tissue male mouse. In this work, we examine how photoxic the drug ciproquin is to L929 cells. According to several studies, ciproquin can cause negative cutaneous effects when combined with UV or visible light.[4] The fluoroquinolone treatment reduced the amount of immunotoxic enzymes and antioxidants in the rat tissues.[5] According to Sabharwal *et al.*,[6] mitochondrial-generated Reactive Oxygen Species (ROS) are involved in stress signaling in healthy cells and have a role in the start of nuclear or mitochondrial DNA alterations that encourage neoplastic transformation. Not many research have demonstrated the detrimental effects of ciproquin on L929 cells when exposed to UV-B light. In this work, we assessed the impact of ciproquin on L929 cells' susceptibility and toxicity to UVB irradiation.

DOI: 10.5530/ijper.20262410

Copyright Information :
Copyright Author (s) 2026 Distributed under Creative Commons CC-BY 4.0

Publishing Partner : Manuscript Technomedia. [www.mstechnomedia.com]

MATERIALS AND METHODS

Chemical and Reagents

Sigma Aldrich USA was the supplier of ciproquin (N8878), Chlorpromazine (C8138), RNO, NBT, TBA, trichloroacetic acid, NADPH, and L-histidine. Additional chemicals were bought from the Riyadh, Saudi Arabia, Local Market.

Experimental design

Milli-Q double-distilled deionized water was used to dissolve Ciproquin. The tests were conducted with a 30-min exposure to UV-B light.

The UV340B UV light meter (China) was used to measure the UV-B radiation intensities in sunshine. UV-B mean intensities employed in the experiment were 1.40±0.6 mW/cm^2. During the exposure periods, the temperature ranged from 27 to 36°C and the relative humidity from 38 to 45%.

Corning glass petri plates (15 × 60 mm) were used to generate two sets of reaction mixtures with three replicates. One set was left in the dark as an unexposed control, and the other set was exposed to UV-B rays with the lid open. To prevent thermal change during exposure to UV-B radiation, petri dishes were maintained on a platform encircled by ice packs (Polar Tech Industries, USA). Two replicates were used to repeat the experiment.

Photochemical assays

Measurement of singlet oxygen ($^1O_2$) generation

Reaction mixture having N, N-dimethyl-p-nitrosoaniline (RNO, 3.5×10^{-e} M), histidine (10^{-M} M) and nalidixic acid, chlorpromazine and ciproquin (80 mg/L) in 0.01 M phosphate buffer (pH 7.4) was exposed under UVA, UVB, sunlight for 60 min. The bleaching of RNO was recorded spectrophotometrically at 440 nm against the control.[7]

Measurement of superoxide (O^{2-}) generation

Reaction mixture having nitro blue tetrazolium (NBT, 1.67×10^{-e} M) and nalidixic acid, chlorpromazine and ciproquin (80 mg/l) in 0.01 M carbonate buffer (pH 10.0) was exposed under UVA, UVB, sunlight for 10 min. Reduction of NBT to nitro blue formazan was observed at 560 nm.[7]

Measurement of image1OH radicals

The generation of hydroxyl (image1OH) radicals was determined according method.[8] The samples were then assayed for formaldehyde formation by the method proposed by Nash.[9] The production of formaldehyde was monitored at 412 nm. Further the quenching of image1OH was performed by adding mannitol (0.5 M) and sodium benzoate (0.5 M) as specific quenchers.

Cell culture

The mouse fibroblast cell line L929 was grown in DMEM culture medium with 10% FBS and antibiotic-antimycotic solution (1%) at CO_2 (5%) and RH (95%) at 37°C in CO_2 incubator.

Photo toxicity assay

MTT assay

The MTT assay is based on the protocol described by Ali *et al.*,[7] with minor modifications. The absorbance was recorded at 530 nm by using multi-well microplate reader (Omega Fluostar). Chlorpromazine (5.0 µg/mL) and l-histidine (100 µg/mL) were used as positive and negative controls, respectively.

Neutral Red Uptake (NRU) assay

The NRU (neutral red uptake) assay is based on the initial protocol described by Borenfreund and Puerner[10] and determines the accumulation of the neutral red dye in the live lysosomes. The absorbance was recorded at 540 nm by using multi-well micro plate reader (Omega Fluostar).

Determination of intracellular ROS generation

The production of ROS in L929 cells because of ciproquin (8 µg/mL) exposure to UVR irradiation was carried out using H_2DCFDA in accordance with the procedures outlined by Ali *et al.*[11]

Oxidative stress

Cell lysate

Following 20 min of exposure to ciproquin (8 µg/mL) under UVB (1.4 mW/cm^2) irradiation, L929 cells were cultured in a CO_2 incubator for the entire night. Following exposure, cells were scraped and washed with PBS before being gathered in an Eppendorf tube. Cell lysate, or the supernatant, was placed on ice for additional testing for Lipid Peroxide (LPO) and reduced Glutathione (GSH) after lysis buffer was combined with scraped cells and centrifuged at 13000 rpm for 15 min at 4°C. The Bradford method was used to determine the amount of total protein in the cell lysate, with bovine serum albumin serving as the standard.[12]

GSH test

The GSH content was evaluated according to Ellman's method.[13] Cell lysate (100 µL) was added to Trichloroacetic Acid (TCA) (5%, TCA 900 µL) and centrifuged at 3000 g for 10 min at 4°C. Again, 500 µL of supernatant was added to DTNB (0.01%, 1.5 mL), and the OD of the mixture was observed at 412 nm. The quantity of GSH was represented as a mole/mg protein.

MDA test

LPO was measured according to the Alarifi *et al.*, method.[14] Briefly, 1.9 mL PBS (0.1 M, pH 7.4) was added to 100 µL cell lysate and left at 37°C for 60 min. After 60 min, TCA (5%) was added,

and the mixture centrifuged at 3000 rpm for 15 min at room temperature. The supernatant was mixed with thiobarbituric acid (1%, 1 mL) and incubated in a water bath at 100ºC for 30 min. A pink color developed, and the OD was measured at 532 nm and expressed as n mol MDA/mg protein.

Evaluation of caspase-3 activity

Ciproquin (8 μg/mL) was exposed to UVB (1.4 mW/cm²) irradiation for 20 min, and the cells were then cultured in a CO_2 incubator for the entire night to determine the impact on caspase-3 activity in L929 cells. Following the manufacturer's instructions, a colorimetric kit (Cayman Chemical) was used to measure the amount of caspase-3 activity in L929 cells.

Translocation of phosphatidylserine using Annexin V-fluorescein-isothiocyanate (FITC) staining

L929 cells were exposed to ciproquin (8 μg/mL) under UVB (1.4 mW/cm²) irradiation for 20 min to examine the translocation of PS (an indicator of early apoptosis). The cells were then cultured in a CO_2 incubator for the entire night. Following the guidelines provided by the kit's manufacturer (Santa Cruz Biotechnology, Santa Cruz, CA, USA), staining was carried out. Briefly, cells were suspended in 200 µL of binding buffer and allowed to sit at room temperature for 30 min while being incubated with 5 µL of Annexin V-FITC. Following the addition of 10 µL of propidium iodide, stained cells were seen under a confocal microscope.

Statistical analysis

For every experiment, a minimum of three separate experiments were conducted in duplicate. One-way Analysis of Variance (ANOVA) was used to evaluate the data, which were presented as Mean (±S.E.). A *p*-value of greater than 0.05 was deemed statistically significant.

RESULTS

Cell viability

The viability of L929 cells following a 24-hr exposure to UVB light and ciproquin (0, 1, 8, 20, 40, 80, and 120 µg/mL were represented in Figures (1A and 1B). Ciproquin under UVB irradiation caused cytotoxicity in cells in a concentration-dependent manner, as seen in Figures 1A and 1B. Cells exposed to UVB light showed the maximum toxicity of ciproquin at 120 µg/mL (Figures 1A and 1B).

UVB-photosensitized ciproquin generates $^1O_2$, $O_2^{\bullet-}$, and $^\bullet OH$

Photosensitized ciproquin generated $^1O_2$, $O_2^{\bullet-}$, and $^\bullet OH$ in a concentration-dependent manner (Figures 2-4). At lower concentrations, free radicals like $^1O_2$, $O_2^{\bullet-}$, and $^\bullet OH$ were not significant; however, at greater concentrations, they were (Figures 2-4). In all ciproquin dosages, a considerable amount of $O_2^{\bullet-}$ was generated during UVB irradiation (Figure 3). $^\bullet OH$ radical generation peaked at 120 µg/mL and peaked at 1 µg/mL in cells exposed to UVB (Figure 4).

UVB-excited ciproquin enhances intracellular ROS production

Photoexcited ciproquin generated intracellular ROS in a concentration-dependent manner (Figures 5a-d). Percent DCF fluorescence was determined under UVB irradiations (Figures 5a-d). Dark control samples did not show DCF fluorescence, while experimental sets showed significant increase in fluorescence due to increased oxidative stress and intracellular ROS generation (Figures 5a-d). The ROS generation was increase as exposure concentration increased (Figures 5a-d). Maximum ROS were observed at ciproquin (8 µg/ mL) under UVB exposure (Figures 5c and d).

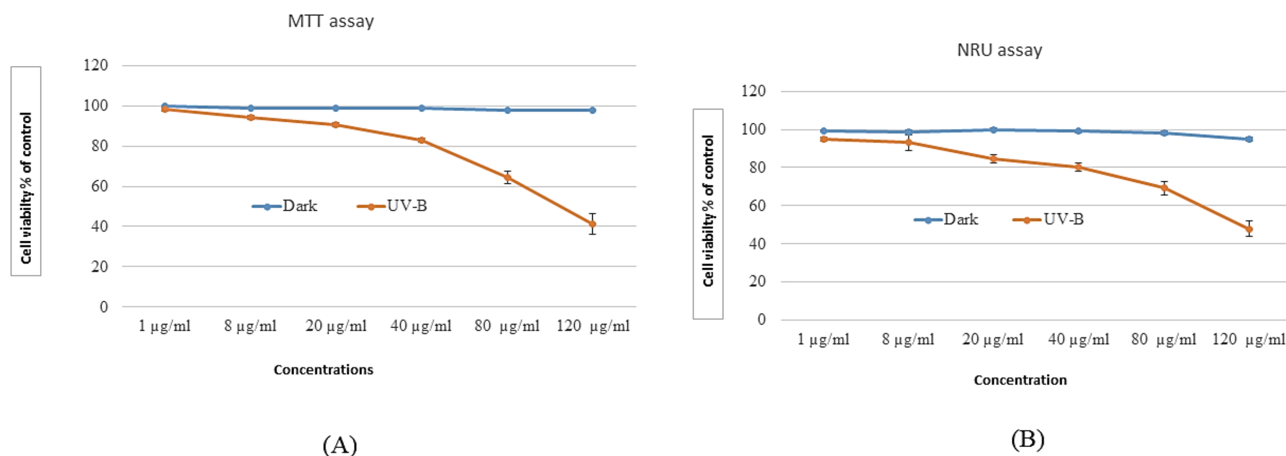

Figure 1: Cytotoxicity of ciproquin on L929 cells under UVB (1.4 mW/cm²) by using (A) MTT (B) NRU tests. Each value represents the mean±SE of three experiments. *$p<0.05$ vs. control.

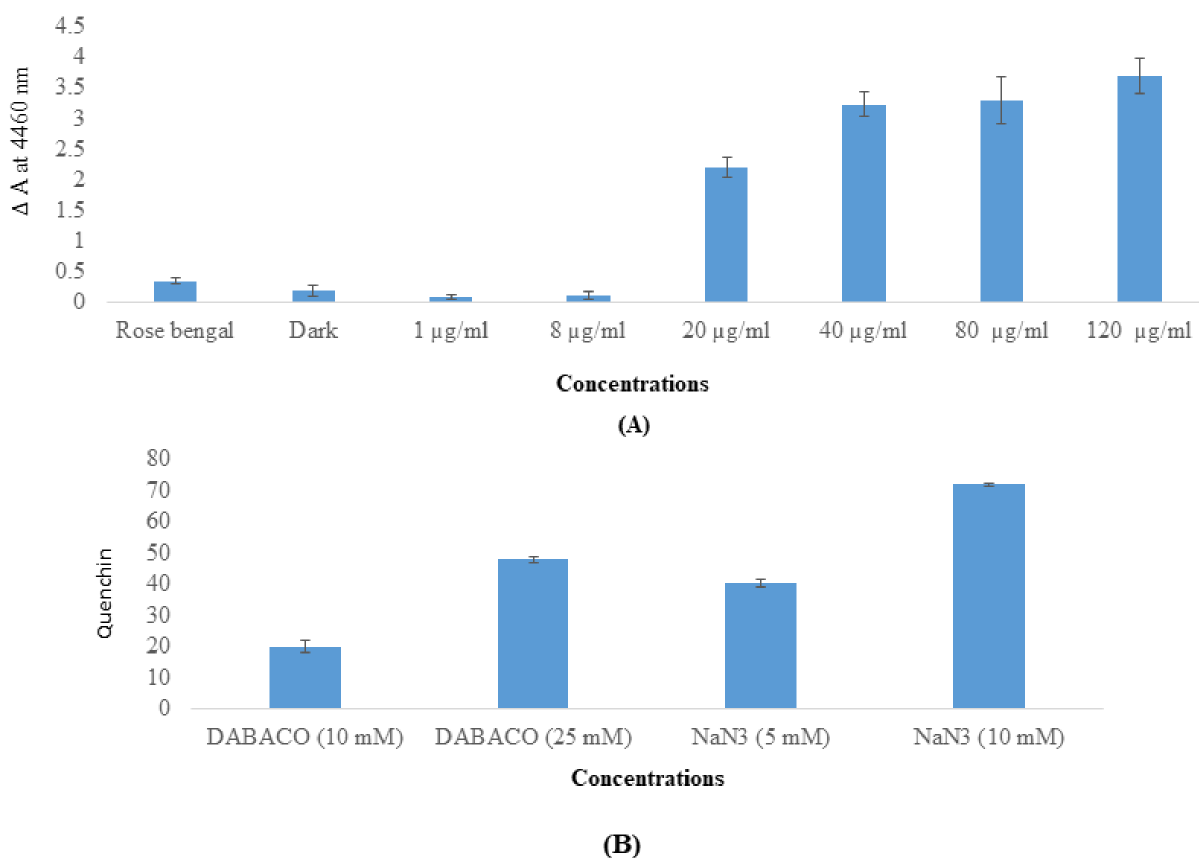

Figure 2: (A) Photochemical generation of $^1O_2$ under UVB (1.4 mW/cm^2) by ciproquin at various concentrations. Rose Bengal used as positive control. (B) Percent photochemical quenching of $^1O_2$ under UVB (1.4 mW/cm^2) at ciproquin (80 µg/mL). Each value represents the mean±SE of three experiments. **p*<0.05 vs. control.

Oxidative stress assays

Oxidative stress was assessed by measuring the levels of LPO and GSH enzymes in L929 cell line after treatment with ciproquin (8 µg/ mL) under UVB irradiation. Lipid peroxidation is a complex chemical process that leads to the oxidative degradation of lipids, resulting in the formation of peroxide and hydroperoxide derivatives. It occurs when free radicals, specifically Reactive Oxygen Species (ROS), interact with lipids within cell membranes. This reaction leads to the formation of lipid radicals, referred to as lipid peroxides or lipid oxidation products, which in turn react with other oxidizing agents, leading to a chain reaction that results in oxidative stress and cell damage. The results showed increased activity of lipid peroxidation of the L929 cell line after treatment with ciproquin (8 µg/ mL) under UVB irradiation as compared to control cells. The high LPO activity was found at concentrations of 8 µg/mL under UVB irradiation and less of a concentration 8 µg/ mL under dark exposure (Figure 6). GSH levels were quantified and statistically compared to those in control cells. The total GSH levels (mM/mg) showed no significant changes (*p*>0.05) across most groups. However, a significant reduction (*p*<0.05) in total GSH levels was observed in cells exposed to ciproquin (8 µg/ mL) under UVB irradiation (Figure 6).

Caspase-3 activity

Caspase-3 activity in the L929 cell line after treatment with ciproquin (8 µg/ mL) under UVB irradiation increased significantly (Figure 7). Experimental sets for caspase-3 activity were compared with the control sets and showed significant increase in enzyme activity (Figure 7).

Phosphatidylserine translocation

Cells showed increased apoptosis under the L929 cell line after treatment with ciproquin (8 µg/mL) under UVB irradiation (Figure 8). Control sets showed green live cells while UVB -exposed cells showed apoptotic cells which appeared orange-green due to merging of both red and green dye (Figure 8). Some necrotic cells (bright red) were also observed in the L929 cell line after treatment with ciproquin (8 µg/mL) under UVB irradiation.

DISCUSSION

The current study investigates the new understanding of how photosensitized ciproquin under UVB irradiation produces intracellular ROS, which causes photocell toxicity, oxidative stress, and apoptosis. The mouse fibroblast (L929) cell line may eventually undergo apoptosis as a result of all these occurrences. Ciproquin was photosensitized by ambient UVB radiation,

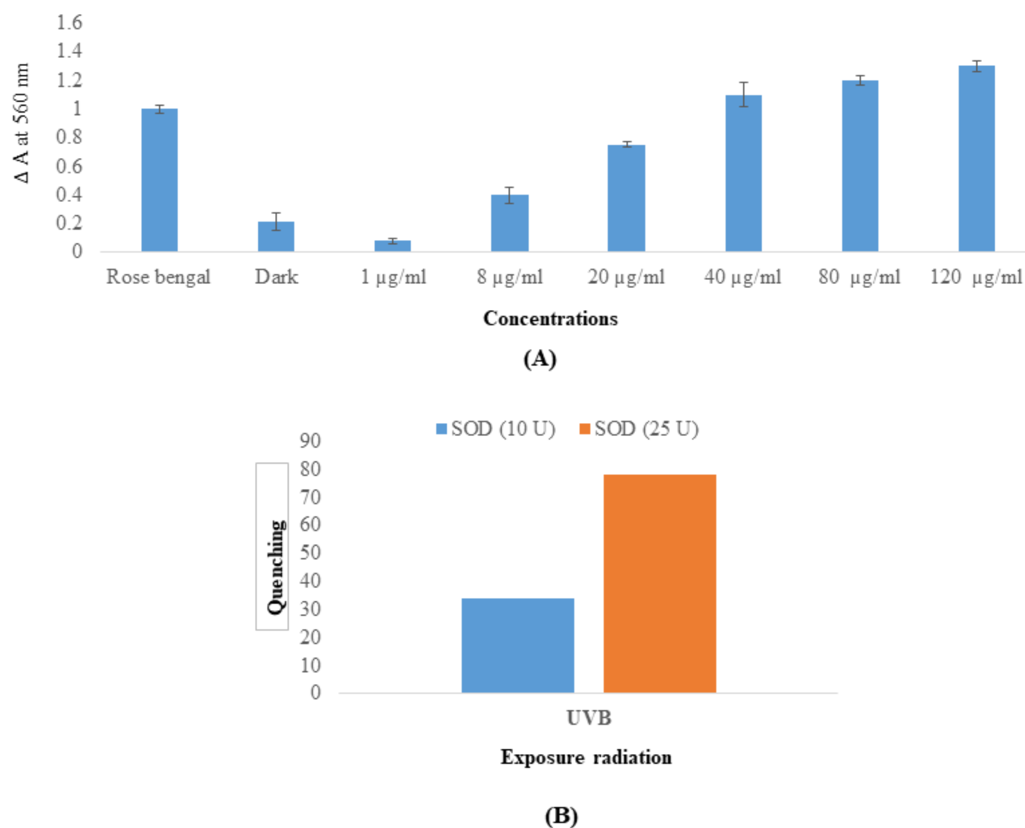

Figure 3: (A) Photochemical generation of O_2^- under UVB (1.4 mW/cm^2) by ciproquin at various concentrations. Rose Bengal used as positive control. (B) Percent photochemical quenching of O_2^- under UVB (1.4 mW/cm^2) at ciproquin (80 µg/mL). Each value represents the mean±SE of three experiments. *p<0.05 vs. control.

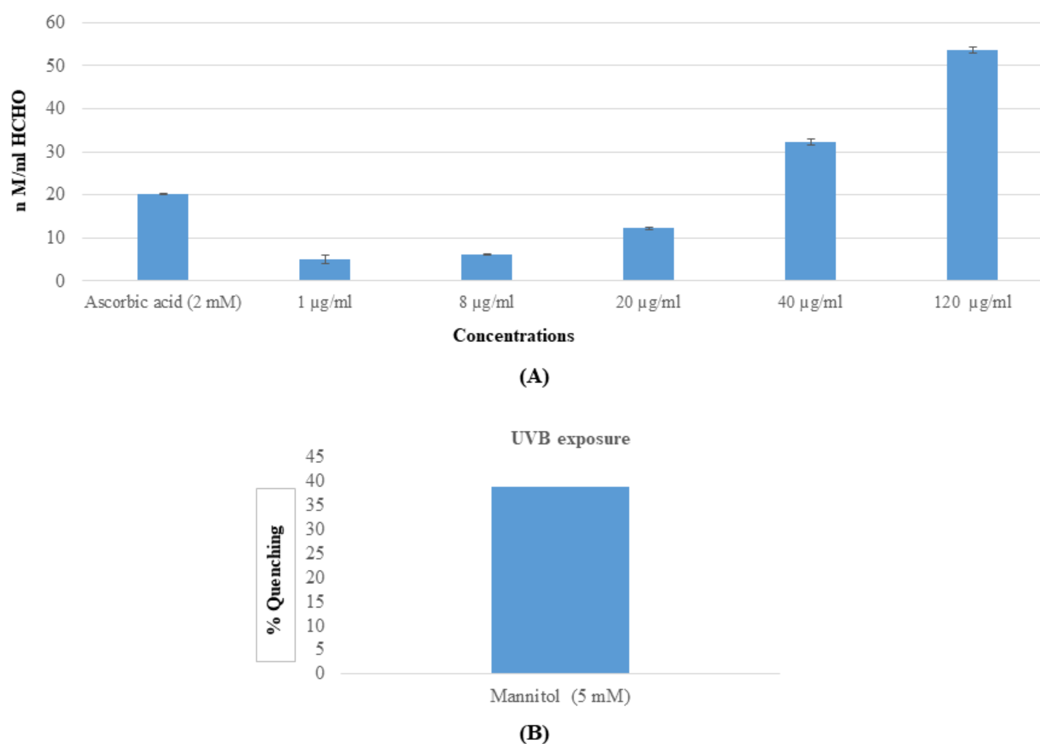

Figure 4: (A) Photochemical generation of *OH under UVB (1.4 mW/cm^2) by ciproquin at various concentrations. Ascorbic acid used as positive control. (B) Percent photochemical quenching of *OH under UVB (1.4 mW/cm^2) at ciproquin (80 µg/mL). Each value represents the mean±SE of three experiments. *p<0.05 vs. positive control.

which led to the production of free radicals like $^1O_2$, $O_2^{\bullet-}$, and $^{\bullet}OH$. Normal cell functions include the generation of ROS and their concomitant quenching by the cellular defense system. Excessive intracellular ROS production can interact with other macromolecules, the nucleus, and membrane-bound organelles, eventually damaging the cell. Photosensitized medications may not always cause apoptosis in response to non-phototoxic UVR radiation doses. On the other hand, higher UV radiation dosages could start the deadly process that could lead to apoptosis.[11] When a photolabile medicine interacts with an appropriate dosage of UVB radiation, adverse phototoxic responses are expected. The production of photoproducts when exposed to UV light is a crucial area of study to determine if the resulting compounds are safer or more phototoxic than their parent compounds. Ciproquin produced $O_2^{\bullet-}$ and $^1O_2$ when exposed to non-lethal UVB levels, demonstrating the involvement of both type I and

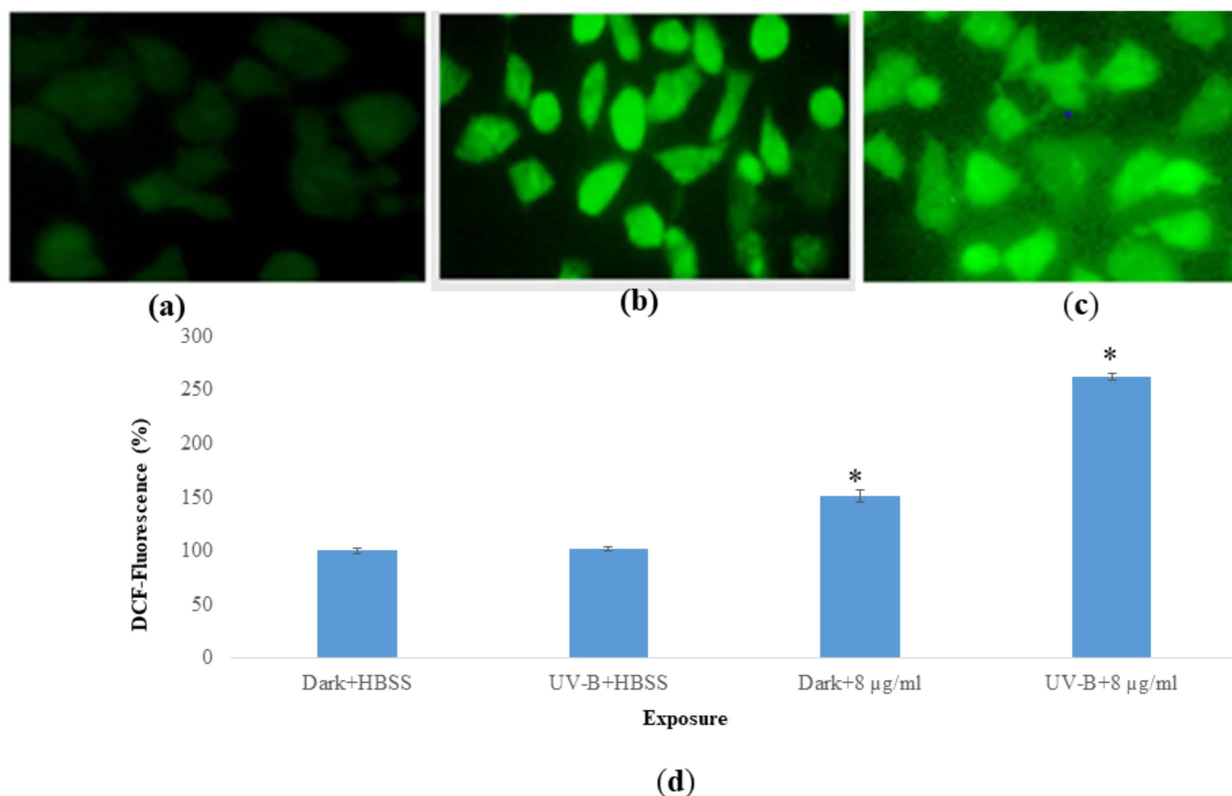

Figure 5: Production of intracellular ROS in L929 cells due to ciproquin under UVB (1.4 mW/cm²) (a), L929 cells under dark (b), L929 cells at UVB+HBSS (c), L929 cells at dark+8 µg/mL (d), L929 cells at UVB (1.4 mW/cm²)+8 µg/mL (e). Percent of DCF fluorescence intensity in L929 cells for 24 hr. Each value represents the mean±SE of three experiments. * $p<0.05$ vs. control.

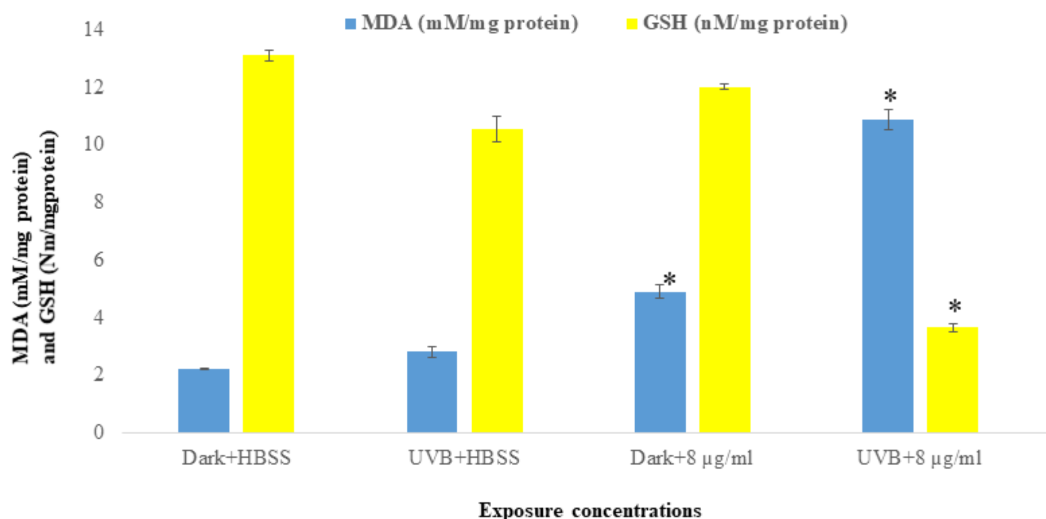

Figure 6: Ciproquin under UVB (1.4 mW/cm²) induced LPO and Glutathione (GSH) in L929 cells for 24 hr. Each value represents the mean±SE of three experiments. * $p<0.05$ vs. control.

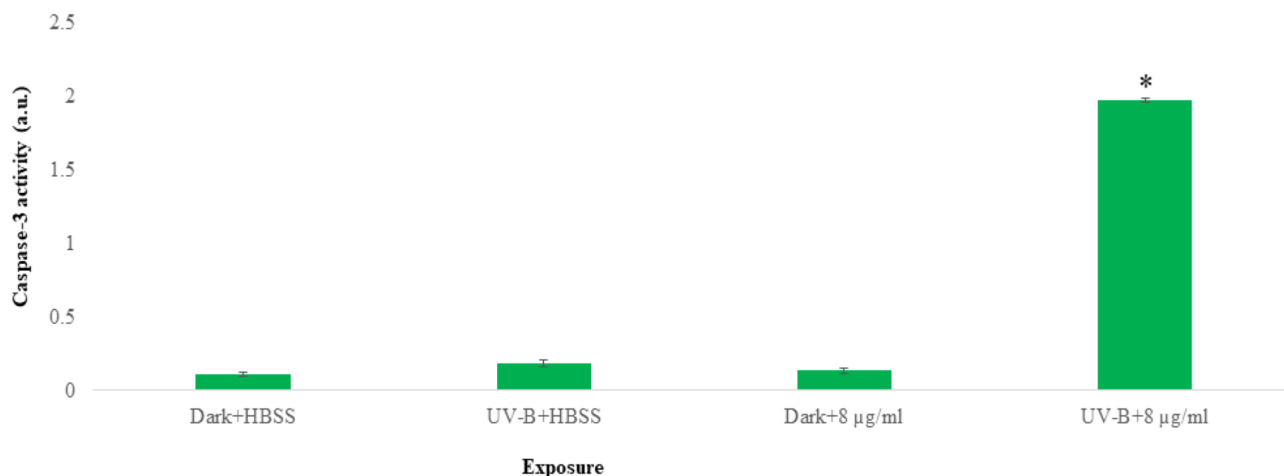

Figure 7: Ciproquin under UVB (1.4 mW/cm^2) induced caspase-3 in L929 cells for 24 hr. Each value represents the mean±SE of three experiments. * $p<0.05$ vs. control.

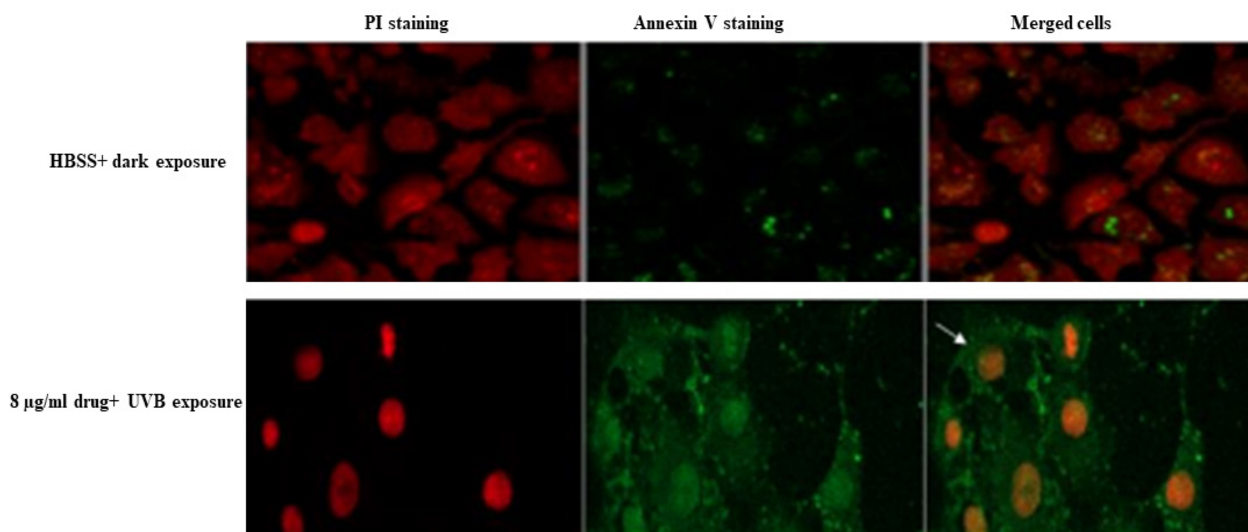

Figure 8: Phosphatidylserine translocation in L929 cells after treatment of ciproquin drug for 24 hr. Arrow is showing early and late apoptotic L929 cells.

type II photodynamic processes. Fibroblast cells may have higher intracellular ROS levels as a result of this. Additionally, chemicals unique to mitochondria and lysosomes increase photoinduced apoptosis.[15] Ciproquin exposure under UVB -exposure for 24 hr had considerable cytotoxicity and apoptotic effects on fibroblast cells in the current experiment. Furthermore, a notable rise in LPO and a decline in glutathione were noted. Additionally, a notable rise in intracellular ROS production was obtained. Other research revealed that ciproquin exposure under UVB exposure led to toxicity, which is in line with our findings.[16] Ciproquin exposed to UVB generated free radicals (ROS), which cause cell degeneration. By monitoring phosphatidylserine translocation and employing annexin-V-FITC labeling, we have verified the apoptotic response of ciproquin during UVB exposure. Based on the aforementioned results, we verified that ciproquin exposure to UVB caused toxicity because of its concentration in cells. Ciproquin exposed to UVB exhibited cytotoxicity and apoptosis, most likely as a result of the exposure's effects and the oxidative stress and ROS it induced. Based on our research, we found that L929 cells are more susceptible to ciproquin when exposed to UVB. In the future, we will investigate the toxicity of ciproquin in an *in vivo* model of UVB exposure.

ACKNOWLEDGEMENT

This project was funded by the National Plan for Science, Technology and Innovation (MAARIFAH), King Abdulaziz City for Science and Technology, Kingdom of Saudi Arabia, Award Number (2-17-01-001-0031).

ABBREVIATIONS

ROS: Reactive Oxygen Species; **UVB:** Ultraviolet B; **L929:** Mouse fibroblast cell line; **•OH:** Hydroxyl radical; **O2•−:** Superoxide anion radical; **1O2:** Singlet oxygen; **GSH:** Reduced Glutathione; **LPO:** Lipid Peroxide; **UVA:** Ultraviolet A (320-400 nm); **UVR:**

Ultraviolet Radiation; **DNA:** Deoxyribonucleic Acid; **RNO:** N, N-dimethyl-p-nitrosoaniline; **NBT:** Nitro blue tetrazolium; **TBA:** Thiobarbituric acid; **NADPH:** Nicotinamide adenine dinucleotide phosphate; **DMEM:** Dulbecco's Modified Eagle Medium; **FBS:** Fetal Bovine Serum; **CO2:** Carbon dioxide; **RH:** Relative Humidity; **MTT:** 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide; **DMSO:** Dimethyl sulfoxide; **HBSS:** Hanks' Balanced Salt Solution; **NRU:** Neutral Red Uptake; **H2DCFDA:** 2′,7′-dichlorodihydrofluorescein diacetate; **DCF:** Dichlorofluorescein; **PBS:** Phosphate Buffered Saline; **TCA:** Trichloroacetic Acid; **DTNB:** 5,5′-dithiobis(2-nitrobenzoic acid); **OD:** Optical Density; **MDA:** Malondialdehyde; **FITC:** Fluorescein-isothiocyanate; **PS:** Phosphatidylserine; **ANOVA:** Analysis of Variance; **S.E.:** Standard Error; **MAARIFAH:** National Plan for Science, Technology and Innovation; **PMID:** PubMed Identifier; **DOI:** Digital Object Identifier; **UV:** Ultraviolet; nm: Nanometers; **mW/cm²:** Milliwatts per square centimeter; **µg/mL:** Micrograms per milliliter; **µM:** Micromolar; **M:** Molar; **pH:** Potential of Hydrogen; **rpm:** Revolutions per minute; **hr:** Hour; **min:** Minutes; **ºC**: Degrees Celsius; **mg/mL:** Milligrams per milliliter; **mM/mg:** Millimolar per milligram; **USA:** United States of America.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

REFERENCES

1. Manley A, Sansom J. Phototoxic reaction to ciprofloxacin. BMJ 2019;365: l2413.
2. Karisma VW, Wu W, Lei M, Liu H, Nisar MF, Lloyd MD, *et al.* L.UVA-Triggered Drug Release and Photo-Protection of Skin. 2021 https://doi.org/10.3389/fcell.2021.598717. Front Cell Dev Biol.
3. Patel K, Goldman JL. Safety concerns surrounding quinolone use in children. J Clin Pharmacol. 2016;56(9):1060-75. doi: 10.1002/jcph.715, PMID 26865283.
4. Adam J. MRCPCH, and Andrew B. MD. If you can't stand the rash, get out of the kitchen: An unusual adverse reaction to ciprofloxacin. Pediatr Pulmonol. 1999;28(6):449-50.
5. Donmez F, Dogan A. Investigation of the effects of three different generations of fluoroquinolone derivatives on antioxidant and immunotoxic enzyme levels in different rat tissues. Drug Chem Toxicol. 2022:t 4; 1-13;45(6):2686-98. doi: 10.1080/01480545.2021.1982624, PMID 34601990.
6. Sabharwal SS, Schumacker PT. Mitochondrial ROS in cancer: initiators, amplifiers or an Achilles' heel? Nat Rev Cancer. 2014;14(11):709-21. doi: 10.1038/nrc3803, PMID 25342630.
7. Ali D, Ray RS, Hans RK. UVA-induced cyototoxicity and DNA damaging potential of Benz (e) acephenanthrylene. Toxicol Lett. 2010;199(2):193-200. doi: 10.1016/j.toxlet.2010.08.023, PMID 20832460.
8. Cohen G, Heikkila RE. The generation of hydrogen peroxide, superoxide radical, and hydroxyl radical by 6-hydroxydopamine, dialuric acid, and related cytotoxic agents. J Biol Chem. 1974;249(8):2447-52. doi: 10.1016/S0021-9258(19)42751-8, PMID 4362682.
9. Nash T. The colorimetric estimation of formaldehyde by means of the Hantzsch reaction. Biochem J. 1953;55(3):4161953:416-21. doi: 10.1042/bj0550416, PMID 13105648.
10. Borenfreund E, Puerner JA. Toxicity determined *in vitro* by morphological alterations and neutral red absorption. Toxicol Lett. 1985;24(2-3):119-24. doi: 10.1016/0378-4274(85)90046-3, PMID 3983963.
11. Ali D, Verma A, Mujtaba F, Dwivedi A, Hans RK, Ray RS. UVB-induced apoptosis and DNA damaging potential of chrysene via reactive oxygen species in human keratinocytes. Toxicol Lett. 2011;204(2-3):199-207. doi: 10.1016/j.toxlet.2011.04.033, PMID 21571051.
12. Bradford MM. A rapid and sensitive method for the quantitation of microgram quantities of protein utilizing the principle of protein dye binding. Anal Biochem. 1976;72:248-54. doi: 10.1016/0003-2697(76)90527-3, PMID 942051.
13. Ellman GL. Tissue sulfhydryl groups. Arch Biochem Biophys. 1959;82(1):701959:70-7. doi: 10.1016/0003-9861(59)90090-6.
14. Alarifi S, Ali D, Alkahtani S. Oxidative stress-induced DNA damage by manganese dioxide nanoparticles in human neuronal cells. BioMed Res Int. 2017; 2017:5478790. doi: 10.1155/2017/5478790, PMID 28596964.
15. Oliveira CS, Turchiello R, Kowaltowski AJ, Indig GL, Baptista MS. Major determinants of photoinduced cell death: subcellular localization versus photosensitization efficiency. Free Radic Biol Med. 2011;51(4):824-33. doi: 10.1016/j.freeradbiomed.2011.05.023, PMID 21664269.
16. Ye JS, Liu J, Ou HS, Wang LL. Degradation of ciprofloxacin by 280 nm ultraviolet-activated persulfate: degradation pathway and intermediate impact on proteome of Escherichia coli. Chemosphere. 2016;165:311-9. doi: 10.1016/j.chemosphere.2016.09.031, PMID 27664520.

Cite this article: Ali D, Alarifi S, Almutairi B, Almarzoug MH, Alakhtani S. Mechanism of Reactive Oxygen Species in the Photo-Cytotoxicity and Photo-Apoptotic Effects of Ciproquin on L929 Cells. Indian J of Pharmaceutical Education and Research. 2026;60(1s):s350-s357.