HSR (LONDON),
HOUGHTON STREET REVIEW JOURNAL
ISSN: 2058-5519|Impact factor-15.9
http://houghtonstreetreview.com/index.php/hsr

EFFECT OF UV AND IF RAYS IN MODELING OXIDATIVE STRESS

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doi:https://doi.org/10.5281/zenodo.22725596

Abstract. Oxidative stress is a major biological mechanism underlying cellular injury caused by various environmental factors, including ultraviolet (UV) and infrared (IR) radiation. Excessive exposure to these types of radiation can disturb the balance between reactive oxygen species (ROS) production and antioxidant defense mechanisms. UV radiation, particularly UVA and UVB, induces oxidative stress through direct photochemical reactions and the activation of endogenous cellular chromophores. IR radiation, especially infrared-A (IR-A), can stimulate ROS production through mitochondrial and heat-associated mechanisms. The resulting oxidative imbalance may lead to lipid peroxidation, protein oxidation, DNA damage, mitochondrial dysfunction, inflammation, apoptosis, and premature tissue aging. This article examines the mechanisms by which UV and IR radiation contribute to oxidative stress and evaluates their potential application in experimental models. Particular attention is given to ROS generation, antioxidant defense systems, molecular signaling pathways, and biomarkers used to assess oxidative damage. Understanding the combined biological effects of UV and IR radiation is important for developing reliable experimental models of oxidative stress and for improving strategies aimed at preventing radiation-induced cellular damage.

Keywords: ultraviolet radiation, infrared radiation, oxidative stress, reactive oxygen species, antioxidants, lipid peroxidation, DNA damage, cellular stress.

Introduction. Oxidative stress is defined as a disruption of the physiological balance between the production of reactive oxygen species (ROS) and the ability of biological systems to neutralize these reactive molecules through antioxidant defense mechanisms. Under normal physiological conditions, ROS participate in intracellular signaling, immune responses, cellular proliferation, and metabolic regulation. However, excessive ROS accumulation can damage essential cellular components, including lipids, proteins, nucleic acids, and cellular membranes. Radiation exposure is one of the important environmental factors capable of disturbing cellular redox homeostasis. Among different forms of electromagnetic radiation, ultraviolet (UV) and infrared (IR) radiation have received considerable attention because of their effects on biological tissues, particularly the skin. UV radiation

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is traditionally divided into UVA (320–400 nm), UVB (280–320 nm), and UVC (100–280 nm). UVA represents the majority of solar UV radiation reaching the Earth's surface and has a strong association with oxidative mechanisms. UVB, in contrast, is more strongly absorbed by DNA and is particularly important in direct photochemical DNA damage. UV radiation can increase the intracellular concentration of superoxide anion, hydrogen peroxide, hydroxyl radicals, singlet oxygen, and reactive nitrogen species. These reactive molecules can initiate lipid peroxidation, oxidize proteins, and induce oxidative modifications of DNA. Research indicates that UVA is particularly effective at producing oxidative damage through photosensitization mechanisms involving endogenous chromophores and molecular oxygen [1, 2]. Infrared radiation has traditionally received less attention than UV radiation because it is associated primarily with thermal effects. However, evidence indicates that IR, particularly IR-A, can also influence cellular redox balance. Infrared-induced ROS formation may result from mitochondrial processes, intracellular chromophore activation, and radiation-associated increases in temperature. Some experimental studies have demonstrated that increased free-radical formation following IR exposure is associated with temperature elevation, suggesting an important interaction between radiation and heat stress [3, 4]. Therefore, studying the effects of UV and IR radiation provides an important experimental approach for modeling oxidative stress. Such models can be used to investigate the molecular mechanisms of cellular injury, evaluate antioxidant compounds, and develop protective strategies against radiation-associated biological damage.

Literature Review. Previous studies have demonstrated that UV-induced oxidative stress involves several interconnected molecular pathways. UVA radiation is weakly absorbed by DNA itself but interacts with endogenous photosensitizers such as flavins, porphyrins, melanin-related compounds, and other chromophores. Following photon absorption, these molecules can transfer energy or electrons to molecular oxygen, resulting in the formation of ROS [1, 5]. The major ROS generated during radiation exposure include superoxide anion ($O_2^{\bullet -}$), hydrogen peroxide (H_2O_2), hydroxyl radical ($^{\bullet}OH$), and singlet oxygen ($^1O_2$). Superoxide can be converted into hydrogen peroxide by superoxide dismutase (SOD). Hydrogen peroxide can subsequently participate in metal-catalyzed reactions leading to the formation of highly reactive hydroxyl radicals. These reactions can initiate oxidative damage to cellular macromolecules [6]. UVA-induced oxidative damage to DNA includes the formation of oxidized bases, particularly 8-oxo-7,8-dihydroguanine (8-oxoG), as well as DNA strand breaks. UVA-associated DNA oxidation is largely linked to singlet oxygen and, to a lesser extent, hydroxyl radicals. UVB can also contribute to oxidative DNA damage, although direct photochemical DNA lesions are generally more prominent following UVB exposure [1, 7]. The relationship between IR radiation and oxidative stress is more complex. IR-A can penetrate relatively deeply into skin tissue and may affect mitochondrial function and intracellular signaling. Studies indicate that solar IR can increase ROS production and activate signaling pathways associated with inflammation and tissue remodeling. The effects may be intensified when radiation-induced oxidative processes are accompanied by heat stress [4]. At the same time, the literature demonstrates that the

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biological response to IR is dependent on radiation intensity, exposure duration, wavelength, tissue temperature, and experimental conditions. Some investigations have reported little or no oxidative response when cells are maintained at physiological temperature, whereas increased free-radical formation has been observed at elevated temperatures [3]. These findings indicate that UV and IR radiation should not be considered identical oxidative stressors. UV radiation acts predominantly through photochemical mechanisms, whereas IR-induced oxidative stress may involve both photobiological and thermal mechanisms.

Materials and Methods. The present study was designed as a conceptual experimental framework for modeling oxidative stress induced by UV and IR radiation. The proposed methodology is based on mechanisms reported in the scientific literature.

Experimental model. An in vitro cellular model can be used to investigate the effects of radiation. Human keratinocytes, fibroblasts, or another appropriate mammalian cell line may be selected depending on the research objective.

Cells should be divided into several experimental groups:

1. Control group – cells without radiation exposure.
2. UV group – cells exposed to controlled UV radiation.
3. IR group – cells exposed to controlled infrared radiation.
4. Combined UV + IR group – cells exposed sequentially or simultaneously to UV and IR radiation.
5. Positive oxidative-stress control – cells treated with an established oxidative stress inducer.
6. Protective-treatment group – irradiated cells treated with a selected antioxidant or protective compound.

Radiation exposure. The UV experimental condition should preferably distinguish between UVA and UVB because their mechanisms of biological action differ. UVA primarily induces oxidative processes through photosensitization, whereas UVB causes substantial direct photochemical DNA damage and can additionally stimulate ROS generation [1, 2]. For IR experiments, the radiation source should be characterized according to wavelength, intensity, exposure duration, and temperature. Infrared-A is particularly relevant for studying skin-associated oxidative stress because of its deeper tissue penetration.

Oxidative stress biomarkers. The oxidative response can be evaluated using several biochemical and molecular indicators:

- intracellular ROS concentration;
- hydrogen peroxide concentration;
- malondialdehyde (MDA);
- protein carbonyl content;
- 8-oxo-7,8-dihydroguanine;
- reduced glutathione (GSH);

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- oxidized glutathione (GSSG);
- SOD activity;
- catalase (CAT) activity;
- glutathione peroxidase (GPx) activity;
- inflammatory signaling markers;
- mitochondrial membrane potential.

The ratio between reduced and oxidized glutathione can provide additional information about changes in the cellular redox state.

Statistical analysis. Experimental measurements should be performed using biological and technical replicates. Data may be presented as mean ± standard deviation. Differences among experimental groups can be analyzed using one-way analysis of variance (ANOVA), followed by an appropriate post-hoc test. A probability level of $\mathbf{p < 0.05}$ can be considered statistically significant.

Results and Discussion. The expected outcome of UV exposure is an increase in intracellular ROS. UVA radiation is particularly relevant because it can interact with endogenous photosensitizers and generate singlet oxygen, superoxide, hydrogen peroxide, and other reactive species. These processes can initiate oxidative damage in cellular membranes, proteins, and DNA [1, 5]. UV-induced ROS production may exceed the capacity of antioxidant systems. Under normal conditions, SOD converts superoxide into hydrogen peroxide, while catalase and glutathione peroxidase contribute to hydrogen peroxide removal. When ROS generation exceeds these antioxidant capacities, oxidative stress develops.

A simplified representation of the process is:

UV radiation → chromophore activation → ROS generation → antioxidant depletion → oxidative stress → cellular damage

The increase in ROS may subsequently cause lipid peroxidation. Polyunsaturated fatty acids in cellular membranes are particularly susceptible to oxidative attack. Lipid peroxidation produces secondary reactive aldehydes, including malondialdehyde, which can further interact with proteins and nucleic acids.

UV-induced DNA damage. DNA is an important target of UV-associated oxidative stress. UVB can directly interact with DNA bases and produce photoproducts such as cyclobutane pyrimidine dimers. UVA, on the other hand, produces substantial oxidative DNA damage through ROS-mediated mechanisms. Oxidized guanine derivatives such as 8-oxoG are commonly used as indicators of oxidative DNA damage [1, 7]. Persistent oxidative DNA damage may activate DNA repair mechanisms and cellular stress pathways. If the damage is excessive or repair mechanisms are overwhelmed, cells may undergo apoptosis or develop genomic instability.

IR-induced oxidative stress. Infrared radiation demonstrates a different pattern of oxidative effects. IR-A can penetrate deeper into biological tissues than UVB and can influence cellular metabolism. Research suggests that solar IR-associated biological effects involve ROS generation and

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activation of signaling pathways. Importantly, IR-induced oxidative stress may be strongly influenced by thermal effects [4]. In experimental systems, an increase in tissue or cellular temperature may enhance mitochondrial ROS production. Therefore, temperature should be carefully monitored during IR experiments. Otherwise, it may be difficult to distinguish direct radiation effects from secondary heat-stress effects. This issue is particularly important for experimental modeling. Some studies have reported that IR exposure did not induce substantial free-radical production when cells were maintained at approximately physiological temperature, while oxidative responses became evident at higher temperatures [3].

Combined effects of UV and IR. Combined UV and IR exposure represents a more realistic model of natural solar exposure. UV radiation may initiate strong photochemical oxidative reactions, while IR can contribute to oxidative and thermal stress. The interaction between these processes may increase the overall cellular burden.

The proposed model can therefore be represented as:

UV + IR exposure → ROS/RNS accumulation + thermal stress → antioxidant imbalance → lipid/protein/DNA damage → inflammation and apoptosis

However, the combined effect should not automatically be assumed to be purely additive. Depending on exposure sequence, intensity, wavelength, cellular state, and temperature, IR may modify the cellular response to UV in either a protective or damaging direction. Previous experimental studies have reported that certain IR exposures can reduce some UV-associated cellular damage, emphasizing the complexity of radiation interactions [3].

Antioxidant defense mechanisms. The cellular antioxidant system is a central determinant of radiation-induced oxidative damage. Important enzymatic antioxidants include SOD, catalase, and glutathione peroxidase. Non-enzymatic antioxidants include glutathione, vitamin C, vitamin E, and other low-molecular-weight antioxidant molecules. When radiation exposure increases ROS production, antioxidant systems may initially compensate for the increased oxidative burden. However, prolonged or intense exposure can decrease antioxidant capacity and result in accumulation of oxidative damage.

A useful conceptual relationship is:

Oxidative stress = ROS production > antioxidant defense capacity

Thus, experimental assessment should include both oxidative damage markers and antioxidant defense markers. Measuring ROS alone may not adequately describe the cellular redox state.

Biological consequences. Radiation-induced oxidative stress can activate multiple cellular signaling pathways. Oxidative modifications may influence transcription factors, inflammatory mediators, mitochondrial function, and cell-death pathways.

Long-term consequences may include:

- cellular senescence;
- premature skin aging;

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- chronic inflammation;
- mitochondrial dysfunction;
- DNA mutations;
- apoptosis;
- impaired tissue regeneration;
- extracellular matrix remodeling.

UVA-associated oxidative stress has been linked to lipid peroxidation, protein carbonylation, DNA lesions, inflammatory responses, photoaging, and photocarcinogenesis [5].

Table 1. Proposed biomarkers for radiation-induced oxidative stress

Biological parameter	Main significance	Expected response after excessive exposure
ROS	General oxidative status	Increase
H_2O_2	Reactive oxygen intermediate	Increase
MDA	Lipid peroxidation	Increase
8-oxoG	Oxidative DNA damage	Increase
Protein carbonyls	Protein oxidation	Increase
GSH	Cellular antioxidant reserve	Decrease
SOD	Superoxide defense	May decrease or become dysregulated
Catalase	H_2O_2 detoxification	May decrease or become dysregulated
GPx	Peroxide detoxification	May decrease or become dysregulated
GSH/GSSG ratio	Redox balance	Decrease
Mitochondrial membrane potential	Mitochondrial integrity	Decrease

Implications for Experimental Modeling. A reliable oxidative-stress model requires careful control of radiation dose, exposure time, wavelength, temperature, cellular type, and post-exposure observation period. UV and IR should therefore be investigated separately before their combined effects are assessed. For UV exposure, UVA and UVB should preferably be analyzed independently because their dominant mechanisms differ. UVA is strongly associated with photosensitized ROS production, whereas UVB has a greater direct photochemical effect on DNA [1, 2]. For IR exposure, temperature must be measured continuously or at sufficiently frequent intervals. This is essential because heat itself can influence mitochondrial activity and ROS production. Failure to control temperature can lead to an overestimation of the direct oxidative effect of IR radiation.

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Conclusion. UV and IR radiation can contribute significantly to the development of oxidative stress, although their mechanisms of action are not identical. UV radiation, particularly UVA, induces ROS predominantly through photosensitization and photochemical mechanisms, while UVB additionally produces direct DNA photodamage. IR radiation, especially IR-A, can influence redox homeostasis through mitochondrial and heat-associated mechanisms. The excessive production of ROS and reactive nitrogen species can overwhelm antioxidant defenses and cause lipid peroxidation, protein oxidation, DNA damage, mitochondrial dysfunction, inflammation, apoptosis, and tissue aging. Therefore, UV and IR exposure can serve as useful experimental tools for modeling oxidative stress when radiation parameters and environmental conditions are carefully controlled. A comprehensive model should measure both oxidative damage and antioxidant defense. The simultaneous assessment of ROS, MDA, 8-oxoG, GSH/GSSG, SOD, catalase, GPx, and mitochondrial function can provide a more reliable characterization of radiation-induced oxidative stress. Further experimental studies should focus on the dose-dependent and time-dependent interactions between UV and IR radiation and on the effectiveness of antioxidant and photoprotective interventions.

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