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HISTOLOGICAL IMPACTS OF ULTRAVIOLET RADIATION ON HUMAN SKIN: A MINI-REVIEW

IMPACTOS HISTOLÓGICOS DA RADIAÇÃO ULTRAVIOLETA NA PELE HUMANA: UMA MINI-REVISÃO

IMPACTOS HISTOLÓGICOS DE LA RADIACIÓN ULTRAVIOLETA EN LA PIEL HUMANA: UNA MINIREVISIÓN

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ABSTRACT

Introduction: Ultraviolet (UV) radiation is one of the main extrinsic factors involved in photoaging and the development of histological skin changes. Chronic exposure to UVA and UVB radiation is associated with direct DNA damage, oxidative stress, collagen and elastin degradation, and local immunosuppression, processes that favor the development of precancerous lesions and skin cancer. Objectives: To critically review the main histological changes induced by ultraviolet radiation in human skin, considering differences between skin phototypes and their relationship with photoaging. Methodology: This is a mini-literature review conducted in the PubMed, SciELO, and Scopus databases, using the descriptors 'ultraviolet radiation,' 'photoaging,' 'human skin,' 'histological changes,' and 'skin phototypes.' Articles published between 2020 and 2025, of an experimental or reviewal nature, were included. Duplicate studies, those not directly related to the topic, or those published before the defined period were excluded.

Keywords: Ultraviolet Radiation. Photoaging. Skin Cancer. Oxidative Stress. Photoprotection.

RESUMO

Introdução: A radiação ultravioleta (UV) é um dos principais fatores extrínsecos envolvidos no fotoenvelhecimento e no desenvolvimento de alterações histológicas cutâneas. A exposição crônica à radiação UVA e UVB está associada a danos diretos ao DNA, estresse oxidativo, degradação de colágeno e elastina, além de imunossupressão local, processos que favorecem o surgimento de lesões pré-cancerígenas e câncer de pele. Objetivos: Revisar criticamente as principais alterações histológicas induzidas pela radiação ultravioleta na pele humana, considerando diferenças entre fototipos cutâneos e a relação com o fotoenvelhecimento. Metodologia: Trata-se de uma mini revisão de literatura realizada nas bases de dados PubMed, SciELO e Scopus, utilizando os descritores 'radiação ultravioleta', 'foto envelhecimento', 'pele humana', 'alterações histológicas' e 'fototipos cutâneos'. Foram incluídos artigos publicados entre 2020 e 2025, de caráter experimental ou revisional. Foram

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excluídos trabalhos duplicados, não relacionados diretamente ao tema ou anteriores ao período delimitado.

Palavras-chave: Radiação Ultravioleta. Fotoenvelhecimento. Câncer de Pele. Estresse Oxidativo. Fotoproteção.

RESUMEN
Introducción: La radiación ultravioleta (UV) es uno de los principales factores extrínsecos implicados en el fotoenvejecimiento y el desarrollo de cambios histológicos en la piel. La exposición crónica a la radiación UVA y UVB se asocia con daño directo al ADN, estrés oxidativo, degradación de colágeno y elastina, e inmunosupresión local, procesos que favorecen el desarrollo de lesiones precancerosas y cáncer de piel. Objetivos: Revisar críticamente los principales cambios histológicos inducidos por la radiación ultravioleta en la piel humana, considerando las diferencias entre los fototipos de piel y su relación con el fotoenvejecimiento. Metodología: Se trata de una mini-revisión bibliográfica realizada en las bases de datos PubMed, SciELO y Scopus, utilizando los descriptores «radiación ultravioleta», «fotoenvejecimiento», «piel humana», «cambios histológicos» y «fototipos de piel». Se incluyeron artículos publicados entre 2020 y 2025, de carácter experimental o de revisión. Se excluyeron los estudios duplicados, aquellos no directamente relacionados con el tema o aquellos publicados antes del período definido.

Palabras clave: Radiación Ultravioleta. Fotoenvejecimiento. Cáncer de Piel. Estrés Oxidativo. Fotoprotección.

1 INTRODUCTION

Ultraviolet (UV) radiation is a form of electromagnetic radiation that comes from the sun, with wavelengths between 100 and 400 nanometers (nm). It is subdivided into three categories: UVA (320–400 nm), UVB (290–320 nm), and UVC (100–290 nm). Most UVC radiation is absorbed by the ozone layer and does not reach the earth's surface. Exposure to UV radiation is a determining factor in human skin health, and can cause a range of biological effects, from cellular changes to the development of skin cancer (Saha B, et al., 2024).

These effects vary according to the intensity, duration of exposure and the type of radiation involved. UVB radiation is known to cause direct DNA damage, resulting in the formation of pyrimidine dimers, which can lead to mutations and eventually skin cancer. On the other hand, UVA radiation penetrates deeper into the skin, reaching the dermis, and is associated with photoaging and the formation of reactive oxygen species (ROS), which can induce oxidative stress and inflammation (Gromkowska-Kępka KJ, et al., 2021). In addition to direct DNA damage, exposure to UV radiation can also affect the skin's immune system. UV radiation induces local immunosuppression, decreasing the skin's ability to respond to pathogens and increasing the risk of developing malignant lesions.

In the clinical context, the effects of UV radiation are observed in a variety of dermatological conditions, including sunburn, photoaging, actinic keratoses, and non-melanoma skin cancers. These conditions result from UV-induced histological and molecular changes, which affect both the epidermis and the dermis (Ke Y, et al., 2021). In view of the high incidence of skin diseases related to sun exposure, especially in tropical countries such as Brazil, it is essential to understand the biological and histological mechanisms involved in the damage caused by UV radiation.

2 MAJOR HISTOLOGICAL CHANGES

Ultraviolet (UV) radiation, present in sunlight, has profound effects on human skin. Initially, UVB rays (280–320 nm) concentrate their action on the epidermis, where they induce direct injury to the DNA of keratinocytes, such as the formation of thymine dimers (Vechtomova YL, et al., 2021).

These changes can lead to cell death or mutations capable of triggering tumor processes. UVA rays (320–400 nm), on the other hand, because they have greater penetrating power, reach the dermis and promote changes in the skin's support matrix, including the degradation of collagen and elastin fibers. This process is directly associated

with premature aging, known as photoaging, one of the main chronic impacts of solar radiation (De Araújo; De Souza, 2022). In addition to direct genetic damage, UV radiation stimulates the formation of free radicals, triggering oxidative stress in skin cells. This redox imbalance compromises essential structures such as lipids, proteins, and DNA, resulting in histological alterations, such as thinning of the dermis, loss of elasticity, and weakening of the skin barrier (Kiyoi T., 2024)

In experimental models with hairless mice, thickening of the epidermis and dermis was also observed, as well as loss of affinity with collagen-specific dyes, evidencing irreversible damage to the tissue (Mayangsari E, et al., 2024). As a response to these aggressions, the skin triggers local inflammatory processes, characterized by the dilation of blood vessels, infiltration of defense cells, and release of mediators such as interleukins (IL-1, IL-6) and tumor necrosis factor alpha (TNF-α). Although acute inflammation favors cell renewal, its persistence can cause tissue degeneration. In the Brazilian scenario, reviews indicate that cumulative sun exposure is associated with a higher incidence of skin cancer in fair-skinned populations, especially in the South and Southeast regions, where intense and prolonged UV radiation is added to lower pigment protection (Vind AC, et al., 2024).

Finally, UV radiation also compromises skin immunity, reducing the activity of Langerhans cells responsible for defending against aggressive agents and altering the production of immunoregulatory substances. This condition of local immunosuppression favors the persistence of damaged cells and the progression of pre-existing lesions, reducing the ability to recognize and eliminate potentially malignant cellular alterations. Together, these mechanisms direct DNA damage, oxidative stress, chronic inflammation, and immunosuppression interact and potentiate each other, explaining the strong association between sun exposure and the development of skin diseases, including skin cancer (Ortner D, et al., 2024).

2.1 EPIDERMAL HYPERPLASIA AND HYPERKERATOSIS

Ultraviolet (UV) radiation, especially in the UVB spectrum, is one of the main physical agents capable of inducing adaptive responses in the human epidermis. Among the most common alterations, epidermal hyperplasia stands out, characterized by the accelerated proliferation of keratinocytes in response to damage to DNA and cellular proteins. This physiological mechanism seeks to restore the integrity of the skin barrier and compensate for radiation-induced cell loss (Lu W, et al., 2023). As reported by Silva, Souza, and Labre (Silva,

et al, 2022), the increased production of new cells represents a temporary defense, which confers greater thickness to the epidermis and greater resistance to subsequent exposures.

Chronic exposure to the sun favors hyperkeratosis, characterized by thickening of the layer as an adaptive response. National studies point to a higher prevalence of this alteration in workers exposed to radiation, such as farmers and fishermen, especially in the face, arms, and hands (De Oliveira, 2020). Although the accumulation of keratin acts as a barrier against UV radiation, its persistence can evolve into pathological changes. Histologically, hyperplasia and hyperkeratosis alter the epidermal architecture, promoting cellular disorganization and increasing the risk of precancerous lesions, such as actinic keratosis, and non-melanocytic skin tumors. Long-term exposure to UVB, therefore, encourages disordered proliferation and increases the likelihood of genetic mutations related to cutaneous carcinogenesis (Hu et al., 2025).

In summary, although initially protective, epidermal hyperplasia and hyperkeratosis have a paradoxical character. If, on the one hand, they contribute to reducing the penetration of UV radiation and temporarily protecting the skin, on the other hand, its chronic maintenance is directly associated with premature photoaging and the development of serious skin diseases. In this sense, as they point out, such adaptations reveal the skin's ability to respond to environmental stress, but also highlight the importance of continuous photoprotection as an essential strategy to prevent progression to irreversible conditions (Wang T, et al., 2020).

2.2 INFLAMMATION AND NECROSIS IN RESPONSE TO IRRADIATION DOSE AND TIMING

Ultraviolet (UV) radiation triggers inflammatory responses in the skin, the intensity of which varies according to the dose and duration of exposure. At moderate levels, it manifests as solar erythema, characterized by redness and local warmth (Salminen et al., 2022). Prolonged exposure, on the other hand, can cause cell necrosis, tissue disorganization, and immunological changes. UV radiation modulates cytokines and T lymphocytes, favoring the escape of abnormal immune system cells and increasing the risk of infections and skin cancer (Lopes et al., 2022).

UV-induced cell necrosis results from the formation of reactive oxygen species (ROS), which cause damage to cellular DNA, leading to programmed cell death or apoptosis. Studies indicate that the efficiency of DNA repair mechanisms can vary between individuals,

influencing susceptibility to damage caused by UV radiation and, consequently, the risk of developing skin cancer. In addition, UV radiation can alter the tumor microenvironment, promoting inflammatory processes, angiogenesis, and evasion of the immune response, factors that contribute to the development of skin neoplasms (Papaccio F, et al., 2022).

The inflammatory response induced by UV radiation is closely linked to photoaging, a cumulative process that is more frequent in the elderly and in regions with high solar incidence (Lee et al., 2021). This phenomenon involves the generation of ROS, which degrade collagen and elastic fibers, compromising skin integrity. Prolonged exposure also promotes structural changes in the epidermis, dermal-epidermal junction and dermis, such as thickening of the layer, disorganization of the extracellular matrix and increased vascular permeability, factors that favor precancerous lesions and skin cancer (Peres; Miot, 2020).

2.3 COLLAGEN DEGRADATION AND ACTINIC ELASTOSIS

Chronic exposure to ultraviolet (UV) radiation is one of the main environmental factors responsible for the degradation of collagen fibers and the development of actinic elastosis in human skin. These structural changes are characteristic of photoaging, a process that results in loss of skin firmness, elasticity, and integrity (Liu H, et al., 2024). Histological studies reveal that UV radiation induces the expression of matrix metalloproteinases (MMPs), such as MMP-1, which cleaves collagen fibers, and MMP-3, which amplifies the degradation of existing collagen. In addition, UV radiation reduces the synthesis of new collagen, contributing to the weakening of the extracellular matrix (MARTIN, et al, 2023).

Actinic elastosis is characterized by the abnormal accumulation of elastin in the dermis, replacing damaged collagen fibers. This elastic material has a thick and tangled appearance, with impaired functionality, compromising the elasticity of the skin. The formation of elastosis is associated with prolonged exposure to UV radiation, which induces the production of reactive oxygen species (ROS), leading to the formation of free radicals that damage elastic and collagen fibers (Md Jaffri J., 2023).

The process of collagen degradation and the formation of actinic elastosis are more intense in fair-skinned individuals, due to the lower amount of melanin and, consequently, greater susceptibility to damage from UV radiation. Advanced age and a history of unprotected sun exposure also increase the risk of these changes, often observed in exposed regions, such as the face, neck, and back of the hands (Shi S, et al., 2024). Prevention involves continuous photoprotection, with regular use of broad-spectrum sunscreens,

appropriate clothing, and reduced exposure at peak times. Topical antioxidants, such as vitamins C and E, also aid in the neutralization of free radicals, preserving the extracellular matrix and slowing photoaging (Lyons AB, et al., 2024).

2.4 OXIDATIVE STRESS AND TISSUE INJURY

Ultraviolet (UV) radiation stimulates the production of reactive oxygen species (ROS), promoting oxidative stress that damages lipids, proteins, and DNA, leading to inflammation, apoptosis, and necrosis of keratinocytes and fibroblasts (Shih et al., 2020). This process compromises the synthesis of collagen and elastin, activates matrix metalloproteinases and accelerates photoaging. As a consequence, chronic sun exposure reduces the skin's regenerative capacity and increases the risk of premalignant lesions.

In addition to the structural effects, oxidative stress affects the cutaneous microcirculation, promoting vasodilation, increased vascular permeability, and infiltration of inflammatory cells. According to Oliveira and Andrade (Oliveira, et al, 2020), these changes contribute to edema and exacerbation of inflammatory processes, amplifying necrosis and tissue degeneration. Cumulative damage from sun exposure not only compromises the physical integrity of the skin, but also interferes with local immune function, making the tissue more vulnerable to infections and malignant transformations (Guan LL, et al, 2021).

The prevention of oxidative stress in the skin involves photoprotection strategies and the use of topical or oral antioxidants, which neutralize free radicals and reduce cell damage. Studies indicate that compounds such as vitamin C, vitamin E, and polyphenols have a significant protective effect against lipid peroxidation and collagen degradation, slowing down the photoaging process and minimizing UV-induced histological changes (Barbosa, et al., 2021).

2.5 VARIATION WITH SKIN TYPE

The cutaneous response to ultraviolet (UV) radiation depends on the phototype, determined by the amount of melanin. Fair-skinned individuals, with less pigment protection, are more susceptible to erythema, inflammation, and cell necrosis. Melanin acts as a natural filter, absorbing part of the UV radiation and protecting DNA, which reduces the occurrence of premalignant lesions in darker skin (Silva; Souza, 2022; Gęgotek et al., 2022). However, this protection is not absolute: even tall phototypes can develop histological changes, such as epidermal hyperplasia and actinic elastosis, when chronically exposed. Thus, the

accumulation of UV radiation over the years promotes dermal and epidermal degeneration in all phototypes, although the severity of the damage is inversely proportional to the amount of melanin (Passeron et al., 2021).

Therefore, the distribution of melanin in the skin influences the inflammatory response. Brazilian studies have shown that fair-skinned individuals have more intense inflammation and greater sensitivity to erythema formation, while darker-skinned individuals have more moderate but not always less severe responses show that, despite the lower initial susceptibility, chronic unprotected exposure to dark phototypes can also result in cumulative tissue damage, increased oxidative stress and predisposition to structural changes, such as collagen degradation (Aloe Vera, et al, 2021).

Finally, the variation with skin type also impacts photoprotection and preventive strategies. Fair-skinned individuals should adopt strict measures, such as the use of broad-spectrum sunscreens, protective clothing, and strategic sun exposure times, while darker-skinned individuals, although less susceptible, are not fully protected and should maintain continuous preventive care. Silva points out that educational policies and awareness about the risks of UV radiation are essential for all phototypes, considering the cumulative effect of the sun on the skin (Silva, Miot, 2024).

2.6 SUBCELLULAR CHANGES AND IMMUNOSUPPRESSION

Ultraviolet (UV) radiation not only affects the macroscopic architecture of the skin, but also causes significant changes at the subcellular level, affecting organelles, nucleus, and cell signaling pathways. Brazilian studies indicate that UV induces direct DNA damage, causing the formation of thymine dimers and changes in hydrogen bonds, compromising the genomic integrity of epidermal cells. These modifications can result in apoptosis or, when repair mechanisms fail, in mutations that favor cutaneous carcinogenesis (Agrez M, et al, 2023).

However, UV radiation also causes mitochondrial dysfunction, increasing the production of reactive oxygen species (ROS) and reducing cellular antioxidant capacity. These changes compromise energy metabolism, structural protein synthesis, and tissue regeneration, accelerating photoaging and favoring programmed cell death (Tsuchida et al., 2023). Another critical effect is local and systemic immunosuppression, marked by reduced activity of Langerhans cells, T lymphocytes, and other mediators of cutaneous immunity, which allows the persistence of damaged cells and increases the risk of malignant lesions.

This phenomenon, more evident in chronic exposures, increases the vulnerability of the skin to infections, cancer, and other pathologies associated with a drop in the immune response (Yardman-Frank et al., 2021; Silva; Miot, 2020).

In addition to the direct effects on organelles and immunity, UV radiation causes changes in cell signaling, activating inflammatory and apoptotic pathways that amplify tissue damage. Studies indicate that the combination of oxidative stress, DNA degradation, and immunosuppression creates an environment conducive to chronic histological changes, including hyperplasia, actinic elastosis, and extracellular matrix disorganization (Cai CS, et al, 2023). These subcellular mechanisms highlight the complexity of the cutaneous response to UV radiation and reinforce the importance of photoprotection and the use of antioxidants as essential preventive measures.

3 COMPARISON: ACUTE VERSUS CHRONIC EXPOSURES

Ultraviolet (UV) radiation causes different effects on the skin depending on the duration and frequency of exposure. Acute exposures, characterized by short, high-intensity episodes such as sunburn, trigger immediate responses, including erythema, edema, and pain. Silva and Souza (Silva & Souza, 2022) highlight that these changes are primarily inflammatory, with rapid activation of chemical mediators and recruitment of immune cells to repair tissue damage. Although temporary, repeated exposures can add cumulative effects, increasing the probability of permanent histological changes. Ultraviolet radiation causes different effects on the skin depending on the duration and frequency of exposure.

Table 1 summarizes the differences between acute and chronic exposures, followed by the references used.

Table 1

ASPECT	ACUTE EXPOSURE	CHRONICLE EXHIBITION	RECENT REFERENCES
Duration	Short, intense, episodic (e.g., sunburn)	Long, repetitive, months or years	Shih et al., 2020; Wong & Chew, 2021
Main effects	Erythema, oedema, pain, focal necrosis	Epidermal hyperplasia, collagen degradation, actinic elastosis	Ansary et al., 2021; Liu et al., 2024
Predominant mechanisms	Acute inflammation, cytokine release	Cumulative oxidative stress, reorganization of the extracellular matrix	Salminen et al., 2022; Cai et al., 2023
Reversibility	Usually reversible after a few days	Progressive and irreversible changes	Yeh & Schwartz, 2022; Shin et al., 2023
Oncological risk	Low on isolated exposures	High risk of precancerous lesions and skin cancer	Ortner et al., 2024; Kumar et al., 2024
Clinical example	Sunburn in young people	Deep wrinkles, basal cell carcinoma	Tsai & Chien, 2022; Vind et al., 2024

Source: Authors.

In contrast, chronic exposure involves continuous or frequent radiation over months and years, promoting profound structural and functional changes in the skin. Chronic exposure induces epidermal hyperplasia, collagen degradation, actinic elastosis, and local immunosuppression, typical features of photoaging. These modifications are progressive and cumulative, making the skin more susceptible to precancerous lesions and non-melanocytic skin cancer (Wong Qya, et al, 2021). From a histological point of view, the differences between acute and chronic exposures are also evident. While acute exposures produce focal necrosis and temporary inflammation, chronic exposure leads to permanent reorganization of the extracellular matrix, decreased fibroblast density, and changes in cutaneous microcirculation. Oliveira and Andrade reinforce that the radiation accumulated over time favors the degradation of collagen and elastin fibers, with a consequent loss of elasticity and firmness of the skin (Oliveira & Andrade, 2020).

In turn, the immune system's response varies greatly depending on the type of sun exposure. When exposure is rapid and short-lived, it causes a temporary drop in immunity. Continuous and prolonged exposure, on the other hand, leads to longer-lasting immune suppression, which can facilitate the growth of altered cells and increase the risk of skin cancer. Barbosa and colleagues point out that these changes that last longer, if not avoided, can evolve into irreversible lesions and impair skin function. Therefore, sun protection measures must take into account not only the intensity of radiation, but also how often and for how long we are exposed to the sun (Barbosa, et al, 2021).

4 CLINICAL IMPLICATIONS AND POSSIBLE INTERVENTIONS

Exposure to ultraviolet (UV) radiation generates clinical implications ranging from transient effects, such as erythema and edema, to chronic conditions, such as photoaging and skin cancer. Cell damage depends on the intensity and duration of exposure, involving inflammation, necrosis, collagen degradation, and actinic elastosis. In addition to the aesthetic impact, these changes compromise the skin barrier, favoring infections and premalignant lesions (Yeh et al., 2022; Silva; Souza, 2022). Photoprotection is the most effective clinical measure, including the regular use of broad-spectrum sunscreens, appropriate clothing, hats, and glasses. Brazilian studies highlight that reapplying sunscreen every two hours is essential to reduce the cumulative effects of solar radiation (Tsai et al., 2022).

In this context, health education policies to raise awareness about the risks of the sun contribute to the prevention of long-term damage. Another clinical approach involves the use of topical and oral antioxidants, which neutralize free radicals and reduce UV-induced oxidative stress. Peres and Miot point out that the combination of vitamin C, vitamin E, and polyphenols acts to protect cellular DNA, preserve collagen, and reduce inflammation, slowing down the photoaging process and reducing the risk of serious skin lesions. In patients with a history of chronic exposure, regular dermatological follow-up is indicated to monitor early changes and prevent malignant complications (Kumar V, et al, 2024).

Finally, clinical interventions also include regenerative treatments, such as the use of retinoids and topical therapies with growth factors, which stimulate the regeneration of the epidermis and dermis. Oliveira and Andrade believe that these approaches can improve skin texture and elasticity, reduce the appearance of wrinkles and actinic lesions, and promote partial recovery of the damaged extracellular matrix. However, the effectiveness of these interventions depends on adherence to ongoing preventive measures, highlighting the importance of combining sun protection, antioxidants, and specialized dermatological care (Shin SH, et al, 2023).

5 FINAL CONSIDERATIONS

Ultraviolet (UV) radiation causes complex changes in the skin, including epidermal hyperplasia, inflammation, oxidative stress, collagen degradation, and immunosuppression, the cumulative effects of which contribute to photoaging and increased risk of skin cancer. Acute exposures cause temporary damage, such as erythema and edema, while chronic exposures promote permanent changes in the extracellular matrix and reduction in fibroblast density, compromising skin function in a sustained manner. Thus, the continuous adoption of preventive strategies, such as photoprotection, use of antioxidants, and dermatological follow-up, associated with regenerative therapies when necessary, is essential to preserve the integrity, elasticity, and immunity of the skin, indicating the need for future research aimed at improving more effective preventive and therapeutic measures.

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