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Ultraviolet light causes skin cell senescence

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Abstract
The skin serves as a strong barrier that keeps the body safe from harm brought on by the environment. Additionally, skin health has a big impact on outward appearance. Skincare and wellbeing are receiving more attention in this day and age of material and spiritual wealth. One of the most frequent external causes of diseases including sunburn, skin cancer, and skin ageing is ultraviolet radiation. Skin cell senescence, a kind of ageing marked by cell cycle halt and impaired cellular function, can be brought on by ultraviolet [UV] radiation, mainly UVA and UVB. DNA damage, oxidative stress, and inflammatory reactions are the main causes of this senescence, which eventually leads to skin ageing and may raise the risk of skin cancer. The main goal of prevention techniques is to reduce UV exposure by using sunscreen, protective clothes, and diets high in antioxidants. This article discusses a number of UV-induced skin cell senescence processes, each with unique traits and reciprocal effects, such as DNA damage, oxidative stress, inflammatory response, and mitochondrial failure. For example, oxidative stress and the activation of the NLRP3 inflammasome result from mitochondrial malfunction, which also causes electron evasion and the production of additional reactive oxygen species, which damages mitochondrial DNA [mt DNA]. According to the current methodology, effective preventative and therapeutic measures are suggested for sunscreen, nutrition, and experimental drugs, respectively, with the goal of delaying the ageing of skin cells and offering UV protection. Insights and recommendations for future research on the mechanism of skin cell senescence are provided, along with a summary of the effects of UV radiation on skin in the hopes of finding better preventative and therapeutic measures. Senescent cell ablation has so far only been demonstrated to have a positive impact on skin ageing in transgenic mouse models or after more general therapies. We conclude that there is strong but not yet absolute evidence that cellular senescence is a significant contributor to intrinsic skin ageing.

Keywords: UV light, skin ageing, skin structure, ageing mechanism, and preventive measure

Introduction

According to one definition, skin ageing is the degenerative process in which changes in the skin's structure and physiology affect its biological functions [1–3]. Depending on where the aging-causing stress comes from, it can be categorised as either intrinsic or extrinsic. Chronological ageing, another name for intrinsic skin ageing, is a result of time and is impacted by hormone levels and genetic background. On the other hand, extrinsic skin ageing, also known as photoaging, is brought on by outside stressors such pollution, UV light, and tobacco smoking [1]. UV light, which can be classified as either UVB (which reaches the epidermis and upper dermis) or UVA (which involves longer UV wavelength radiation that can penetrate the dermis), is the primary cause of photoaging [2]. DNA damage accumulation, genome instability, epigenetic dysregulation, extracellular matrix degradation, mitochondrial dysfunction, inflammation, loss of proteostasis, ER-stress, and autophagy dysfunction are some of the molecular alterations linked to the onset of skin ageing. Cellular senescence, one of the characteristics of skin ageing, is intimately linked to many of these changes. A condition known as cellular senescence occurs when cells undergo persistent cell cycle arrest and develop a range of phenotypic alterations pertaining to morphology, gene expression, and cellular activities [3]. This process usually starts after the body is exposed to small, long-term damages, like when the skin is exposed to UV light from the sun. Other known causes of cellular aging include telomeres getting shorter, genes that promote cancer becoming active, damage to the DNA, and problems with the mitochondria [4]. When DNA gets damaged, several pathways, including p53, p38-MAPK, NF, and mTOR, are turned on to help create and keep cells in a state of aging [5]. These pathways are also involved in stopping the cell from dividing and causing an inflammatory response, which is called the senescence-associated secretory phenotype (SASP). This is one of the main features of cell aging. The SASP creates an environment that makes it easier for the body to break down proteins in the skin and encourages aging in nearby cells, which can lead to tissue damage, aging, and the development of tumors. Senescent cells also have other characteristics that are used to detect aging in the lab and in the body. These include the cells looking bigger and flatter, a rise in the activity of a certain enzyme called senescence-associated β-galactosidase (SA-β-Gal), changes in how the DNA is organised, more sugar usage, proteins related to the immune system, pathways that help cells survive, DNA damage, loss of the body's ability to handle proteins properly, and problems with the mitochondria [6]. Out of all these, increased levels of SA-β-Gal activity is the most commonly used marker to identify aging cells. But since none of these signs are unique to aging, more than one marker is needed to be sure that a cell has aged [1-4]. The main job of the skin is to act as a protective barrier against harmful things like germs, chemicals, and physical stress and to help keep the body's internal balance. Most of the skin's barrier function comes from the keratinocytes in the outer layer, called the epidermis, with the stratum corneum playing a big role in keeping things safe [6]. The skin isn't completely waterproof; water can escape to the outside, a process called transepidermal water loss (TEWL), which is

one way to measure how well the skin's barrier is working. Other signs that show the skin's barrier is working well include how hydrated the outer layer is, the amount of oils (sebum) on the skin, and the skin's pH level [7]. There are also other techniques, like tape stripping, that help look at the makeup of the stratum corneum, either through mass spectroscopy or using methods like corneosurfametry, where samples are checked using staining and color measurements. These methods are useful for checking how treatments affect older skin and in 3D models that mimic real skin [8]. Skin aging is a process caused by both natural and external factors, leading to a gradual loss of structure and function over time.

Cellular Senescence

Replicative senescence was first discovered in 1961 by Hayflick and Moorhead. It refers to the loss of a cell's ability to divide after a certain number of times it has doubled in culture. Later, it was found that telomeres, which are the protective ends of chromosomes, get shorter with each cell division. This shortening was linked to replicative senescence. Telomerase, an enzyme, can prevent telomeres from getting too short, which helps stop replicative senescence. When telomeres get too short, the ends of chromosomes look like breaks in DNA, which triggers a DNA damage response. If this response continues, it leads to senescence. Because telomeres are hard for the DNA repair system to reach, they cause ongoing DNA damage that can lead to senescence or cell death. In the body, replicative exhaustion is just one of many factors that cause senescence.

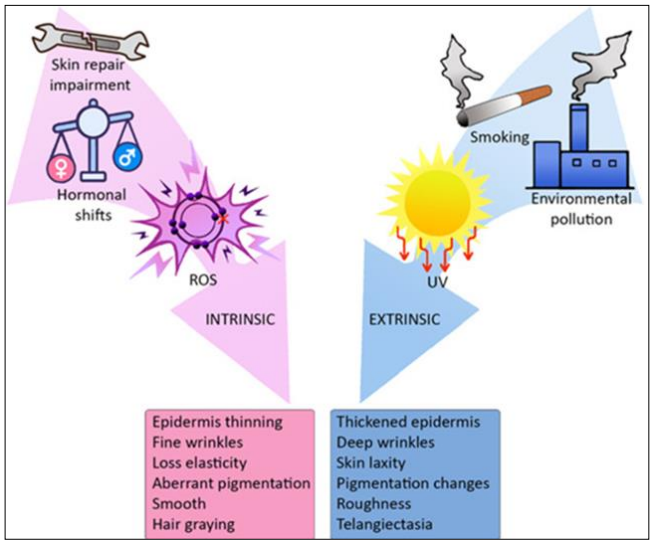

Fig 1: Both extrinsic (such as smoking, UV exposure, and environmental pollution) and intrinsic (such as ROS, hormones, and impairment in skin repair) factors contribute to skin ageing

Other factors, like DNA-damaging substances, radiation, or certain genes, can also lead to senescence. Even though telomere uncapping is a common feature of senescent cells, there are many different ways senescence can happen. This variety comes from the different way's cells are triggered into senescence. To detect senescent cells, scientists use several markers. One well-known marker is the activity of an enzyme called beta-galactosidase, which becomes more active in older cells. Other markers include proteins that stop cells from dividing, like p16, p21, p53, and others, and a decrease in proteins that promote cell growth. Other signs include changes in parts of the cell's structure, such as the loss of LaminB1 or the movement of a protein called HMGB1 from the nucleus. Checking for DNA damage foci along with telomeres can also indicate a long-lasting DNA damage response that leads to senescence. However, no single marker is enough to confirm a cell is senescent. This is because many of these markers can also be found in stressed cells that do not become senescent, and senescence can look different in different cell types or under different conditions. Also, the methods used to measure these markers aren't always accurate enough to give a complete picture. Therefore, using multiple markers together is now the standard way to detect senescent cells. Even though senescent cells can't divide, they are still active in other ways. This activity helps them develop various changes, or 'building blocks,' that help them stay in a senescent state. One important building block is mitochondrial dysfunction, which leads to more of a harmful substance called ROS. This helps maintain senescence by keeping the DNA damage response going. Mitochondria play a key role in the process of cell senescence. Another important building block is the Senescence-Associated Secretory Phenotype (SASP).

Senescent cells release many active molecules, such as inflammatory chemicals, growth factors, and enzymes. They also release other substances like small particles and harmful materials. The make-up of the SASP varies depending on the cell type, how the senescence started, and the timing of the process. For example, some cells release anti-inflammatory signals first, but later they might release pro-inflammatory ones. The SASP can attract immune cells that help remove senescent cells, but it can also keep the cells in a senescent state and even cause other nearby cells to become senescent. Some components of the SASP, like TGF-β, can affect the ability of stem cells to differentiate or grow. These factors can lead to chronic inflammation and tissue changes. Because of this, recent research describes senescence as a stress response that involves stopping cell division and a range of other changes. This means even cells that don't divide, such as neurons or muscle cells, can undergo this type of stress. In fact, these cells can develop senescence-like changes as they age.

Solar Ultraviolet Spectra and Effects on the Skin

The type of ultraviolet radiation (UVR) that reaches the Earth's surface is mostly long wavelength ultraviolet A (UVA), which ranges from 320 to 400 nm. Only a small part, about 5%, is the shorter wavelength ultraviolet B (UVB), which ranges from 280 to 320 nm. The even shorter wavelength ultraviolet C (UVC), which is between 200 and 280 nm, doesn't reach the Earth's surface because it is blocked by oxygen in the atmosphere and absorbed by the ozone layer. However, as the ozone layer gets thinner, more UVB radiation gets through, which increases the risk of skin cancer. Studies show that a 1% drop in ozone levels can lead to a 1% to 2% increase in melanoma deaths. Solar simulated radiation (SSR) is a type of light created by a device that mimics the sun's radiation. This kind of light is about 50% visible light, 40% infrared light, and 9% UV light, with about 0. 4% of that being UVB. It's important to remember that the amount of sunlight reaching the Earth changes depending on where you are, the time of year, and the time of day. UVR causes both short-term and long-term

effects on the skin. Short-term effects include skin damage, redness, changes in skin cells, weakened immune response, vitamin D production, and tanning. Long-term effects include skin aging and skin cancer, which are thought to be caused by changes in skin cells and a weakened immune system.

DNA Photo Damage

One of the most important things that happens when people are exposed to ultraviolet radiation (UVR) is damage to their DNA. UVA and UVB rays affect the skin in different ways. UVB is more harmful to skin cells and can cause mutations in DNA. Studies show that UVB is much more effective than UVA at causing DNA damage, redness, tanning, and skin cancer in mice, depending on the wavelength of the light [7, 8, 9]. UVA, on the other hand, can pass through window glass and goes deeper into the skin, reaching the layer beneath the surface. It's been found that about half of the UVA exposure happens even when someone is in the shade [10]. UVB is directly absorbed by DNA and causes direct damage to the structure of DNA bases. UVA doesn't directly damage DNA but instead causes indirect damage by creating harmful chemicals called reactive oxygen species (ROS), such as superoxide, hydrogen peroxide, and singlet oxygen [11]. These ROS can cause breaks in DNA strands and link DNA to proteins [2, 3]. The type of UVR between 245 and 290 nm is particularly absorbed by DNA [4], which suggests UVB is a main cause of DNA mutations [5]. UVB causes two main types of DNA damage called cyclobutane pyrimidine dimers (CPD) and pyrimidine (6-4) pyrimidone photoproducts (64PP), with a ratio of 4:1 to 10:1 [6, 7]. These types of damage are linked to the development of skin cancer, especially in animals. CPD and 64PP can cause specific mutations, such as changing pairs of bases from CC to TT or changing a single base from C to T, which are known as UVB fingerprint mutations [9]. More than half of basal cell carcinomas (BCC) and squamous cell carcinomas (SCC) have these UVB fingerprint mutations [9, 10].

Even small amounts of UVB, less than what causes sunburn, can quickly cause CPD and 64PP in human skin [7]. It seems that CPD formation isn't limited to UVB. UVA, which isn't absorbed much by DNA, can also cause CPD. In fact, UVA causes more T=T CPDs than UVB does [2-4]. The reason for this is not fully understood, but it involves a process that uses a photoexcited chromophore, which is still unknown. After exposure, 64PP is repaired quickly, but CPD takes longer to fix, with many lesions still present after a day [6, 7]. There's also evidence that some DNA damage, like those with cytosine, is repaired faster than others, like those with thymidine [6, 8]. DNA damage from UV radiation can lead to gene mutations in important genes, such as p53. This is believed to be the first step in causing non-melanoma skin cancer [11].

Sunburn

The ability of UV light to cause skin redness (erythema) gets weaker as the wavelength gets longer. To get the same redness effect, you need about 1000 times more UVA than UVB [8]. Redness from UVB appears about 4 hours after exposure, gets worst between 8 to 24 hours, and goes away in a day or so. In people with fair skin or older individuals, this redness can last for weeks. The time it takes for UVA to cause redness and tan is in two parts. Redness often shows up right after exposure and fades in a few hours, then comes back later, starting around 6 hours and peaking at 24 hours. Redness is linked to many changes in skin cells and molecules, especially the presence of dead skin cells (sunburn cells) [2, 3]. The pattern of UV causing tanning and redness is almost the same, but UVA is better at causing tanning while UVB is better at causing redness. The fact that the pattern for redness is very similar to that for DNA damage suggests that DNA damage plays a big role in causing redness [7].

UV and Immunosuppression

Besides causing harmful effects like skin damage and redness, UVR also weakens the body's immune system both locally and throughout the body [4]. It can lower the body's ability to detect and fight against cancer or virus-related substances [5]. When the skin is exposed to UV light, the immune cells called Langerhans cells change in function and appearance, leading to their reduced presence in the skin. This UV-induced immunosuppression might be a way to stop the body from attacking harmful materials produced due to UV damage, like damaged DNA [e. g. UV-damaged DNA]. Most of the immune system weakening caused by UV exposure is linked to UVB rays. However, recent research shows that UVA rays can be even more harmful to the immune system than UVB [6].

Studies on mice show that this immune weakening from UV exposure might increase the risk of developing skin cancer [5]. In an experiment with mice, skin tumors that formed in adult mice after long-term exposure to high levels of UVB light were not able to grow when placed in healthy mice, but they did grow when placed in mice that had been exposed to UVB before [7]. In humans, UVR can also stop the body from developing a reaction called contact hypersensitivity, which is used as a model to understand immune responses connected to skin cancer [8, 9]. Interestingly, this reaction is easier to block in people with lighter skin types [types I/II] than in those with darker skin types [types III/IV] [10]. Also, there is evidence that people with different skin types respond differently to oxidative stress [11], which is believed to be involved in cancer development [3].

Discussion

Cellular senescence and photoaging have been widely studied in recent research. In fact, the presence and buildup of senescent cells in the skin are considered part of the aging process of this tissue. The appearance of senescent melanocytes in the skin is seen as a way to stop the growth of cancer cells. However, there is still not enough understanding about how UVB light harms melanocytes and how this process connects with skin aging and changes in skin color. The goal of this study was to create a model that shows how UVB causes melanocytes to become senescent, so we could look into the same pathways that have been studied in other skin cells, like fibroblasts [12]. We also looked at how fibroblasts and melanocytes interact, and how factors from senescent fibroblasts might affect melanocyte behavior. Skin tanning is a protective response to sunlight. When skin is exposed to UV light, it gets darker quickly and stays that way for a few days. This happens because existing melanin oxidizes and becomes more compact, and melanosomes move around in the skin. This process is mainly caused by UVA light. UVB causes a different kind of response, called delayed tanning, which happens a few

days after sun exposure and involves making new melanin through a process called neomelanogenesis, which activates the pathway that makes melanin. Most methods for measuring melanin use absorption spectroscopy, which looks at melanin in cells that have been broken apart, but ignores the build-up of melanosomes and the new melanin being made [13]. The Fontana-Masson (FM) staining technique is commonly used to find argentaffin granules in skin tissue, but it's not often used to detect melanosomes in melanocytes grown in a monolayer. In our UVB-induced senescence model, we found that UVB exposure led to a big increase in melanin inside the cells, as measured by a color-based test. We also modified the FM method to work with cells growing on coverslips. This helped show that the increase in melanin after UVB exposure is due to more melanosomes being stored [12, 13]. This confirmed that FM staining can effectively show how melanin and melanosomes change under different conditions. We also confirmed that repeated low doses of UVB can lead to real melanin production. The skin is an important part of how we look, and changes in its color, like pigmentation issues, can be very upsetting and affect how people feel and behave. Still, the reasons behind pigmentation changes during aging are not fully understood. Recent studies have shown that pigmentation problems like melasma, vitiligo, and senile lentigos are linked to chemicals from senescent fibroblasts. Using a system where fibroblasts and melanocytes are grown together, we looked into how factors from senescent fibroblasts might affect melanocyte functions, especially those that change when melanocytes become senescent from UVB. In this system, melanocytes that were grown with senescent fibroblasts had higher melanin levels and more melanosomes per cell, showing how interactions between fibroblasts and melanocytes help with UVB-related skin color changes. This also shows that factors from senescent fibroblasts can control the production of melanin [14].

Conclusions

A significant health danger, photo damage is linked to obvious and unattractive skin effects including wrinkles and hypo or hyperpigmentation, as well as a higher chance of skin cancer and a loss of insulation and defence against external pathogens. Increased ROS and DNA damage levels are important indicators of photo aging's negative impacts, as they can cause most skin cell types to prematurely undergo senescence. Senescent cells can also cause skin ageing through non-cell-autonomous processes such promoting aberrant remodelling and chronic inflammation, as well as cell-autonomous mechanisms like decreasing regenerative ability. Public education about photoprotective measures should be maintained in order to reduce the harmful effects of UVR. A deeper comprehension of melanin, its photoprotective qualities, and the roles that melanocytes play in cancer would be ideal, despite the fact that there are many different substances with photoprotective qualities, such as antioxidants, plant extracts, and DNA repair enzymes. This might open up new possibilities for safely adjusting pigmentation when there is no sunlight, both to prevent skin cancer and to enhance pigmentation for aesthetic purposes. However, in order to prevent interfering with some senescent cells' capacity to repair tissue, senolytic interventions must be thoroughly described and optimised.

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