

Long_Exposure_of_UV_Radiation_on_Erythema_and_Melanin_Index.pdf

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THE EFFECT OF LONG EXPOSURE OF UV RADIATION ON ERYTHEMA AND MELANIN INDEX

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Abstract:

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UV radiation are divided into 3, namely UV A (400 – 315 nm), UV B (315-280 nm), UV C (280 – 100 nm). UV C radiation have the greatest effect on skin damage compared to UV A and UV B. UV radiation can reach the earth's surface, that can cause burning of the skin with signs such as redness of the skin (erythema), pain, blistering and peeling of the skin. Until now there has been no research on the effect of long exposure of UV C radiation on the erythema and melanin index, so aim this research is know about the effect of long exposure of UV C radiation on the erythema and melanine index. This research was conducted by giving exposure to rat that had been shaved with variations in exposure time, namely 5 minutes, 10 minutes, 15 minutes and 20 minutes. The skin that has been exposed with UV radiation will be photographed for color analysis using a chromometer. The results showed that exposure of UV radiation for 10 minutes caused the greatest increase in the melanin and erythema index

Keywords: UV C rays, length of exposure, erythema index, melanin index, rat

Introduction

UV radiation are divided into three, namely UV A (400 – 315 nm), UV B (315-280 nm), UV C (280 – 100nm).^{1,2} Not all UV radiation from the sun reaches the earth. UV C rays which have the greatest energy cannot reach the earth's surface because they are absorbed in the ozone layer. Meanwhile, UV A and B radiation can penetrate the ozone layer.¹ Due to global warming so that the ozone hole is formed and causes UV C radiation to reach the earth's surface³.

UV radiation that reaches the earth's surface can cause burning of the skin with signs such as redness of the skin (erythema), pain, skin blisters and skin peeling¹. Erythema is redness of the skin caused by increased blood flow to the skin due to dilation. Superficial blood vessels in the dermis layer caused by exposure to UV radiation. This occurs through the interaction of reactive oxygen species (ROS) with mast cells in the dermis layer resulting in the release of inflammatory mediators such as histamine which causes vasodilation of blood vessels. In addition, exposure to UV radiation can cause hyperpigmentation to respond to the skin due to UV exposure. Exposure of UV radiation will encourage the production and proliferation of melanin which can cause skin discoloration.⁸ Melanin comprises the largest bioaggregate of different pigments formed from the oxidation and cyclization of the amino acid tyrosine.⁴

Until now there has been no research examining the effect of long exposure to UV radiation, especially UV C radiation on the erythema and melanin index

Research Method

Material

M. malabathricum L flower, UV C lamp cabinet with a length of 106 cm, width 34 cm and a height of 53 cm with 1 UV C lamp (PHILLIPS 30 Watt), 96% ethanol

Animals

Female wistar rats with body weight 200 – 250 g were housed in independent cage. Food and water provided without restriction. All procedures were accordance with our institutional guidelines and were approved by the ethics committee of faculty of medicine University of Lambung Mangkurat No. 591/KEPK-FK ULM/EC/IV/2021

Exposure UV Radiation

Rats was adapted for 7 days and on the 8th day, rats was shaved with an area of 15 cm² (3 x 5 cm). After being shaved, the skin of rats was photographed, then rats was put into a UV-C cabinet with a length of 106 cm, width 34 cm and height 53 cm which contained a UV-C lamp (PHILLIPS 30 Watt). The irradiation was carried out for 5 minutes, 10 minutes, 15 minutes and 20 minutes. After irradiation, skin of rats was photographed again and the values of the erythema and melanin index were measured.⁵

Melanin and Erythema Index Analysis

Melanin and erythema index were measured using a chromometer based on the L * a * b* color system with the formula⁴ :

Index Melanin = $1.06\Delta a * 1.44\Delta L *$.

Index Erythema = $1,68\Delta a * 0,60\Delta L *$.

Results

Results of the UV radiation test, the highest erythema and melanin index occurred in 10 minutes of radiation (Fig 1 and 2)

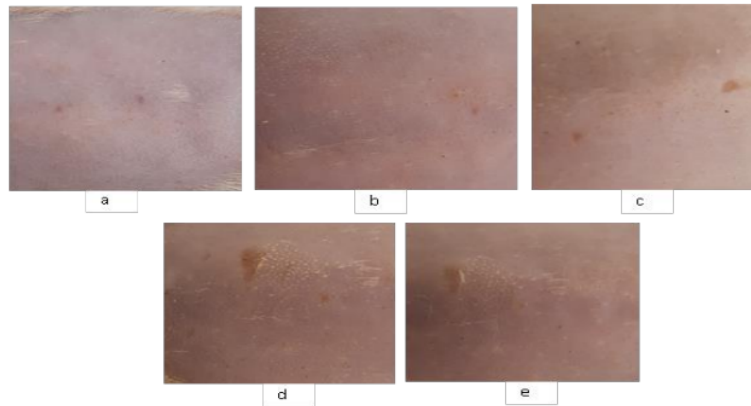


Figure 1 Skin after exposure to UV lamp (a) 0 minutes, (b) 5 minutes, (c) 10 minutes, (d) 15 minutes, (e) 20 minutes

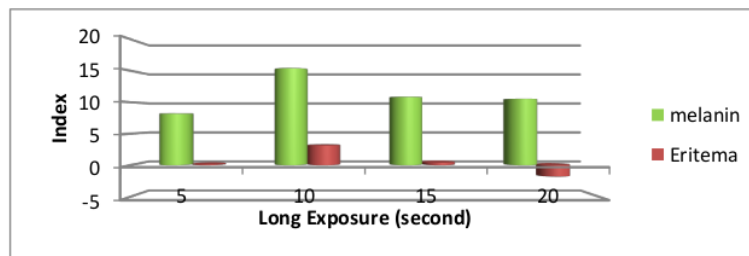


Figure 2 Effect of long exposure to UV light on melanin and erythema index

Discussion

UV radiation have many physiological effects on the skin, both acutely and delayed. One of the acute effects of UV radiation is the induction of inflammation. UV radiation induces a cascade of cytokines, vasoactive and neuroactive mediators in the skin which together produce an inflammatory response and cause "sunburn". One of the effects of UV radiation is the darkening of the skin.⁶

UV-mediated skin darkening is actually biphasic, with the initial darkening occurring from redistribution and/or molecular changes in the existing epidermal melanin pigment.

Whereas the delayed increase in skin darkening, mediated by the actual up-regulation in melanin synthesis and transfer to keratinocytes, begins hours to days after UV exposure. Adaptive melanization is probably a complex physiological response.⁶

Melanin is a large bio-aggregate consisting of subunits of different pigment species formed by the oxidation and cyclization of the amino acid tyrosine.⁹ The amount and type of melanin are the main factors that determine skin color and UV sensitivity.⁸ There are 2 main chemical forms of melanin, namely (1) eumelanin, a dark

pigment that is widely expressed in the skin of highly pigmented individuals, and (2) pheomelanin, a light-colored sulfate pigment that results from the incorporation of cysteine into the melanin precursor.^{6,7}

Melanin synthesis is triggered by the presence of tyrosine, which is then converted into dopaquinone which is the initial precursor for the formation of eumelanin and pheomelanin. The presence of one of the compounds such as cysteine, glutathione, tripeptide components of cysteine, glutamate, and glycine causes the formation of pheomelanin. When intramelanosomal cysteine is depleted, dopaquinone spontaneously cyclizes to form leukodopachrome (cyclodopa). The redox reaction between leukodopachrome and dopaquinone is unchanged further giving rise to orange dopachrome. Dopachrome spontaneously decarboxylates to 5,6-dihydroxyindole (DHI), which is rapidly oxidized and polymerized to form insoluble brown-black eumelanin. However, if dopachrome tautomerase (TYRP2, DCT) is available, dopachrome will tautomerize without losing its carboxylic acid to form DHI-2-carboxylic acid (DHICA). Tyrosinase-associated protein 1 (TYRP1, GP75) catalyzes the conversion of DHICA to soluble light brown eumelanin.^{7,8}

Melanin has a shielding effect against UV exposure by dispersing or absorbing radiation to prevent penetration through the epidermis. Melanin is estimated to absorb up to 50% to 75% of UV in contact with the skin and convert energy into heat through internal conversion.⁷ Of the two types of melanin, eumelanin has far more photoprotective properties than pheomelanin. Melanosomes in darker skin, having a high eumelanin content, have been shown to be intact in the epidermis due to their resistance to lysosomal degradation.^{7,8}

Eumelanin is much more efficient at blocking UV radiation than pheomelanin, so more eumelanin in the skin, less UV permeable to epidermis of skin. White people who are almost sensitive to UV and have a high risk of skin cancer have less epidermal eumelanin. Epidermal eumelanin levels that determine skin color, UV sensitivity and cancer risk. Data show that pheomelanin can enhance oxidative DNA injury and melanomagenesis by generating free radicals in melanocytes even in the absence of UV.^{6,7,8}

Factors that influence the formation of melanin are genetic factors such as age, ethnicity, extrinsic factors including UV radiation and certain chemical compounds, while intrinsic factors include molecules secreted by surrounding keratinocytes, fibroblasts, inflammatory cells, nerves or endocrine which are affected by conditions such as pregnancy and diabetes.⁸

The increase in skin pigmentation in response to UV exposure takes place in several different steps—the first step is a transient phenomenon, called direct pigment darkening (IPD), which occurs within minutes of UV exposure. It appears as a grayish tint that gradually fades to a brown color over a few minutes to days, depending on the UV dose and individual skin tone. IPD is not based on the synthesis of new melanin, but rather the result of photooxidation of pre-existing melanin and redistribution of existing melanosomes from the perinuclear to peripheral dendritic sites. IPD is followed by a second phase called persistent pigment darkening (PPD). PPD, which is brown to brown in color, is thought to be the result of melanin oxidation (similar to IPD), occurring within hours of UV exposure and persisting for at least 3-5 days. The last stage of skin tanning, delayed tanning response (DT). DT is different from PPD and will become apparent 2-3 days after UV exposure. DT results from the

stimulation of melanin synthesis and involves an increase in the number and activity of functional melanocytes, an increase in dendricity, an increase in the synthesis and transfer and a change in the packaging of melanosomes. According to the tanning reaction, there is an increase in the activity of tyrosinase, a rate-limiting enzyme in the melanogenic pathway. Maximum DT from 10 days to 3-4 weeks, depending on UV dose and individual skin tone. It may take weeks or months for the skin to return to its constitutive base color. UVA-induced DT is 2-3 times less efficient per unit dose than UVB and has an earlier onset, often immediately after IPD. Moreover, it has a distinct oxygen – dependent pathophysiology unlike UVB.⁹

IPD/PPD is caused by oxidation of melanin (a precursor) and redistribution of melanin without melanin synthesis. But recently, it was found that pigment cells (melanocytes) express low levels of rhodopsin photoreceptors of the same family as in the retina of the eye, but are "tuned" differently. After UVA radiation, these photoreceptors trigger early melanin synthesis in melanocytes, with peak efficiency at UVA, not UVB. IPD/PPD and UVA photoreceptor-induced melanin synthesis most likely contribute to the efficient skin pigmentation induced by UVA-enriched tanning lamps.¹⁰

In addition to changes in melanin, the effect of UV light is the occurrence of erythema. The effectiveness of UV light in inducing erythema depends on the wavelength of UV light. To produce the same erythema response, approximately 1000 times more doses of UVA than UVB are needed. UVB-induced erythema occurs about 4 hours after exposure, peaks at about 8-24 hours, and fades over about a day; in fair-skinned and older people, UVB erythema may be persistent, sometimes lasting for weeks. UVA-induced erythema is biphasic. Erythema

is often evident immediately at the end of the radiation period and will fade within a few hours, followed by delayed erythema starting at 6 hours and peaking at 24 hours. Erythema is associated with a wide variety of changes at the cellular and molecular level, but especially with the appearance of apoptotic keratinocytes (sunburned cells). The spectrum of action for UV-induced tanning and erythema was almost identical, but UVA was more efficient in inducing tanning whereas UVB was more efficient in inducing erythema. The observation that the spectrum of action for erythema is very similar to that of CPD induction suggests that DNA damage is an important trigger for erythema.⁹

In this study, the detected skin discoloration probably occurred in the IPD phase due to photooxidation of pre-existing melanin and redistribution of existing melanosomes from perinuclear to peripheral dendritic sites. This can be seen by increasing the length of exposure causing a decrease in the melanin index. The decrease in the melanin index is due to the amount of existing epidermal melanin that has been used up/reduced, so that when the length of exposure is increased, it causes a decrease in melanin.

Further research is needed on the mechanism of changes in the erythema and melanin indices that occur and the time required to reach the IPD, PPD and DT phases on exposure to UV C light.

Conclusions

UV radiation cause an increase in the erythema and melanin index. The greatest increase in the erythema and melanin indices occurred after 10 minutes of UV exposure

Further research is needed on the mechanism of changes in the erythema and melanin indices that occur and the time

required to reach the IPD, PPD and DT phases on exposure to UV C light.

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