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Evaluation of Melanin Content and MITF Levels in UV-Exposed Mice Treated with Mulberry Extract and Hydroquinone

Yuvana Dewanti[1,2, 3,4], Suparmi Suparmi[5]*, Eko Setiawan[1,6,7]

[1]*Master's Study Program of Biomedical Science, Faculty of Medicine, Universitas Islam Sultan Agung, Semarang, Indonesia*
[2] *Community Health Centre, Purwokerto Selatan, Banyumas, Central Java, Indonesia*
[3]*Amanda Main Clinic, Banyumas, Central Java, Indonesia*
[4]*Amanda Pratama Clinic, Banyumas, Central Java, Indonesia*
[5]*Department of Biology, Faculty of Medicine, Universitas Islam Sultan Agung, Semarang, Indonesia*
[6]*Department of Surgery, Faculty of Medicine, Universitas Islam Sultan Agung, Semarang, Indonesia*
[7]*Department of Surgery, Sultan Agung Islamic Hospital, Semarang, Indonesia*

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ABSTRACT

Ultraviolet B (UVB) radiation induces skin hyperpigmentation by stimulating melanin synthesis via the upregulation of melanogenic pathways, particularly involving the microphthalmia-associated transcription factor (MITF). This study investigated the effects of mulberry extract serum at concentrations of 5% and 10% on UVB-induced hyperpigmentation in mice, comparing them with hydroquinone, a conventional depigmenting agent. Male C57BL/6 mice were exposed to UVB radiation for 14 days to induce hyperpigmentation, as confirmed via macroscopic observation and histopathological analysis of melanin deposition using Masson–Fontana staining. The impact of the treatments on melanin content was quantified biochemically, and the expression levels of MITF were evaluated using immunohistochemical methods. The findings indicated a significant increase in melanin accumulation and MITF expression following UVB exposure, corroborating the hyperpigmentation model. The treatment effects on total melanin in UVB-exposed mouse skin tissue showed values of 44.07 ± 0.57 for the control and 38.40 ± 0.47 and 34.80 ± 0.31 for the 5% and 10% mulberry extract serum, respectively. Treatment with hydroquinone resulted in an MITF level of 34.39 ± 1.32, whereas 5% mulberry extract serum effectively lowered melanin levels to 32.02 ± 4.4. The 10% mulberry serum led to a significant reduction in MITF expression to 28.70 ± 1.70. These results indicate that mulberry extract inhibits melanogenesis, potentially by downregulating MITF activity—a mechanism similar to that of hydroquinone. This dose-dependent depigmenting effect suggests mulberry's potential as a natural alternative for hyperpigmentation control. Overall, the findings from this study highlight the efficacy of mulberry extract in inhibiting UVB-induced melanin synthesis, supporting its use as a safe, plant-based alternative for skin lightening.

Keywords: Hyperpigmentation, mulberry extract, Microphthalmia-Associated Transcription Factor (MITF), melanin, Ultraviolet B

Introduction

Ultraviolet (UV) radiation, particularly UVB, plays a crucial role as an environmental factor that promotes skin hyperpigmentation and photoaging by stimulating melanogenesis, the process by which melanin is produced in melanocytes.[1] Excessive UV exposure activates multiple molecular pathways, leading to increased melanin production and resulting in dark spots, uneven skin tone and pigmentation issues. The process of melanogenesis is controlled by complex signalling cascades, with the microphthalmia-associated transcription factor (MITF) serving as a key regulator of melanocyte activity and melanin production.[2]

*Corresponding author. Email: suparmi@unissula.ac.id
Tel: (62) 821-3455-5381

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UVB radiation stimulates melanin production and its movement to the upper layers of the epidermis as a protective mechanism against UV damage. However, this process leads to visible hyperpigmentation.[3,4] Long-lasting pigmentation may result from UVB exposure, which hampers melanin removal in basal keratinocytes, leading to persistent hyperpigmentation.[5]

Conventional skin-lightening agents, such as hydroquinone, have been widely regarded as the gold standard for treating hyperpigmentation disorders, including melasma, post-inflammatory hyperpigmentation and solar lentigines. Its primary mechanism involves inhibiting tyrosinase, an enzyme essential for melanin production, thereby reducing pigmentation.[6,7] However, researchers and clinicians have raised concerns about their safety and long-term effects. Hydroquinone may cause skin irritation and contact dermatitis, potentially leading to post-inflammatory hyperpigmentation, especially in individuals with darker skin.[6,8] Extended use can result in exogenous ochronosis, a condition characterised by blue–black pigmentation, especially in darker skin types.[6,9] Hydroquinone is cytotoxic to melanocytes and mutagenic to mammalian cells, raising concerns about its long-term safety.[6] Evidence suggests that hydroquinone could cause cancer in animals, but its significance for humans remains unclear.[10] Consequently, interest in investigating natural alternatives with fewer side effects is growing.

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Mulberry (*Morus alba* L.) extract is a botanical compound known for its antioxidant and skin-protective properties. Mulberroside A and oxyresveratrol have been shown to decrease the expression of MITF, a key transcription factor that regulates the production of melanogenic enzymes, including tyrosinase, TRP1 and TRP2.[11–13] This downregulation occurs via the activation of the ERK signalling pathway, which leads to the degradation of MITF.[13]

Previous research suggests that natural extracts can influence melanogenesis by reducing MITF levels and decreasing melanin production, although their effectiveness depends on concentration and formulation.[1] While in vitro studies have reported the potential of mulberry extract to inhibit tyrosinase and offer sun protection,[14] in vivo assessments in animal models exposed to UV radiation are limited. Existing studies emphasise the protective and anti-ageing effects of mulberry extract against UV-related skin damage, but a direct comparison with hydroquinone, a common skin-lightening agent, is lacking.

This study compared the effects of 2% hydroquinone and mulberry extract serum at concentrations of 5% and 10% on key melanogenesis markers, including MITF levels and total melanin content, in UVB-irradiated C57BL/6 mice. The aim was to assess whether mulberry extract could serve as a natural alternative for hyperpigmentation treatment and provide a foundation for future targeted comparative research. Understanding how these agents affect molecular markers, such as MITF and melanin content, in UV-exposed skin can aid in the development of safer and more effective skin-lightening therapies.

Materials and Methods

Ethical Approval

All experimental procedures followed the guidelines for laboratory animal care and use, as outlined in the Helsinki Declaration and the National Ethical Criteria for Health Research from the Indonesian Ministry of Health. Ethical approval was granted by the Ethics Committee of the Faculty of Medicine at Universitas Islam Sultan Agung, Indonesia (document number 298/VI/2025/Komisi Bioetik).

Preparation of Mulberry Extract Serum

All chemicals used were of analytical grade. Mulberry (*M. alba* L.) extract in powder form was purchased from PT. INBI Nusantara Sejahtera, Bali, Indonesia. Serum containing 5% and 10% mulberry extract was sourced from PT. Derma Elok Farma in Tangerang, Indonesia. Hydroquinone 2% was acquired from PT. Surya Dermato Medica Laboratories (SDM), Jakarta, Indonesia.

Experimental Animals and Design

This study involved 19 male C57BL/6 mice weighing 20–25 g. It was conducted atthe Integrated Biomolecular Laboratory of the Faculty of Medicine, Universitas Islam Sultan Agung, Semarang, Indonesia. The mice were housed in standard polypropylene cages measuring 70 × 50 × 30 cm, with a rigid base, and maintained at 23°C ± 2°C, 60% humidity, and a 12-h light/dark cycle. Throughout the study, they had free access to filtered water and a normal rodent diet. The mice were acclimated to the laboratory environment for 7 days prior to the start of the experiment.

To validate UVB-induced hyperpigmentation, four mice were categorised into two groups: one group was exposed to UVB at 180 mJ/cm², 302 nm wavelength, 10 min/day for 14 days, and the other served as a control. The remaining 15 mice were segregated into three experimental groups, each comprising five mice. The hydroquinone group was exposed to UVB and subsequently treated with hydroquinone at a concentration of 2%; the mulberry 5% group was exposed to UVB, followed by treatment with serum containing 5% mulberry extract; and the mulberry 10% group was exposed to UVB and then treated with serum containing 10% mulberry extract. The treatments were administered topically on the UVB-exposed skin at a dose of 0.5 g/day for 5 days.

Sampling

For the validation study, the skin was excised after 14 days of UVB exposure. In the treatment group, it was collected after 19 days of UVB exposure and treatment. The dorsal skin samples exposed to UVB were carefully cleaned with sterile saline to remove debris and then gently dried. The mice were anaesthetised with chloroform. The exposed skin was carefully isolated and immediately preserved in 10% buffered formalin for approximately 16 h. After fixation, the tissue was embedded in paraffin, sectioned into thin slices and placed on glass slides for analysis.[15]

Assessment of UVB Effect on Hyperpigmentation in Mice

To confirm hyperpigmentation caused by UV exposure, skin images were acquired using a polarised dermoscope (DermLite DL4, 3Gen LLC, USA) and a mobile camera. Assessments were performed at baseline (prior to UV exposure), immediately after UV irradiation and at specific time points following treatment. The dermoscope was held perpendicularly on the skin to maintain consistent contact and image clarity. All images were obtained under uniform lighting and magnification (10×) to facilitate standardised comparisons.[15]

Histopathological Analysis of Melanin Content Using Masson–Fontana Staining

To evaluate melanin levels in skin tissue, 4-µm-thick sections were prepared and mounted on slides. Masson–Fontana staining was performed according to standard procedures to specifically visualise melanin deposits. Briefly, tissue sections were deparaffinised in xylene, rehydrated via a graded series of alcohols and treated with silver nitrate (Masson–Fontana reagent) under light exposure to develop pigmentation. The slides were then counterstained with nuclear fast red, dehydrated, cleared and mounted. For melanin measurement, three random fields in the epidermal area were selected at 400× magnification per slide. Images were captured using a digital camera on a light microscope under consistent lighting conditions. Brown-coloured melanin granules were distinguished from other tissue structures. Melanin content was quantified by calculating the average percentage area within each field using ImageJ software. The mean of the three regions was used to determine a representative melanin level for each sample. These data were statistically analysed to assess treatment effects on melanin levels.[16]

Assessment of Hydroquinone and Mulberry Extract–Containing Serum on the Level of MITF in UVB-Induced Mice

Following the manufacturer's protocol, MITF levels were assessed in skin tissue samples via an enzyme-linked immunosorbent assay (ELISA). Tissue samples were homogenised with radioimmunoprecipitation assay buffer, supplemented with a proteinase inhibitor, then centrifuged to remove debris and stored at −80°C until analysis. A commercial MITF ELISA kit, which included precoated 96-well plates, standards and detection reagents, was employed. Standards and samples were run in duplicate and incubated with the detection antibody and substrate solution. The reaction was terminated using a stop solution, and the absorbance was read at 450 nm with a STAT FAX® 4700 ELISA Microstrip Reader, USA. A standard curve was generated to quantify the levels of MITF in the samples.[15]

Statistical Analyses

All graphs were drawn using GraphPad Prism software (version 10.1.0, GraphPad Software, San Diego, CA, USA) for Mac. Data were presented as mean ± standard errors. Group means for total melanin content before and after UVB exposure were analysed using a paired t-test. By contrast, group means for MITF and total melanin content in the treatment groups were compared using a one-way analysis of variance (ANOVA), followed by the least significant difference test as a post-hoc analysis. A p-value of <0.05 was considered statistically significant. Statistical analysis was performed using IBM SPSS Statistics version 31.0.0.0 for Mac.

Results and Discussion

Effect of UVB Exposure on Hyperpigmentation: Validation Study

Macroscopically, camera observations revealed that the skin of normal mice prior to UVB exposure was smooth, pinkish and free of excessive pigmentation or noticeable textural changes (Figure 1a). However, after 14 days of UVB exposure, hyperpigmentation appeared as increased

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yellow–brown colouration, skin folds and surface thickening (Figure 1b). Dermoscopy exhibited clear differences from the original macroscopic appearance of mouse skin before (Figure 1c) and after 14 days of UVB exposure (Figure 1d). These findings demonstrate that 14 days of UVB exposure induces hyperpigmentation in C57BL/6 mouse skin.

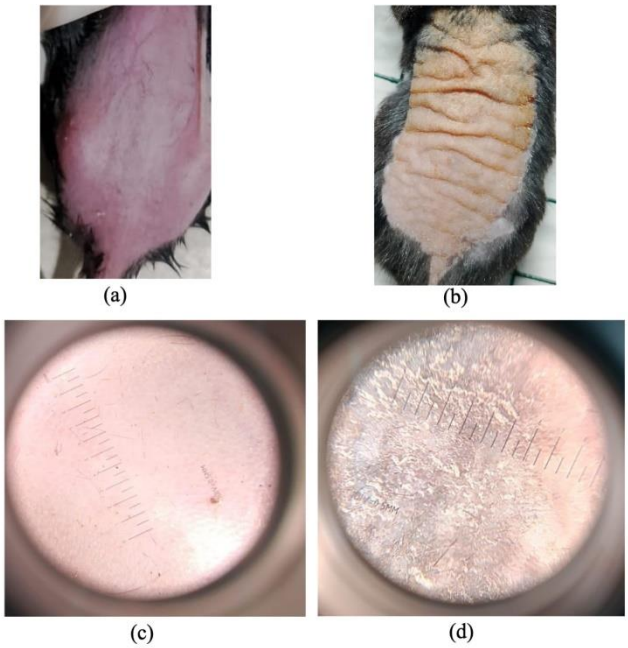

Figure 1: Macroscopic images of mouse skin: (a) skin condition before UVB exposure, (b) skin condition after 14 days of UVB exposure, showing signs of hyperpigmentation, (c) skin dermoscopic image before UVB exposure, (d) after 14 days of UVB exposure

Figure 2 presents the histopathological analysis of mouse skin tissue stained with Masson–Fontana, observed before and after 14 days of UVB exposure (Figure 2A). Initially, the basal layer of the epidermis demonstrated minimal and sparsely distributed melanin granules, indicating a low density. Nonetheless, following 14 days of UVB exposure, the number of melanin granules increased significantly, with dense and intensely stained granules extending into the suprabasal layer (Figure 2B). This increase in melanin density suggests enhanced melanin production due to UVB exposure.

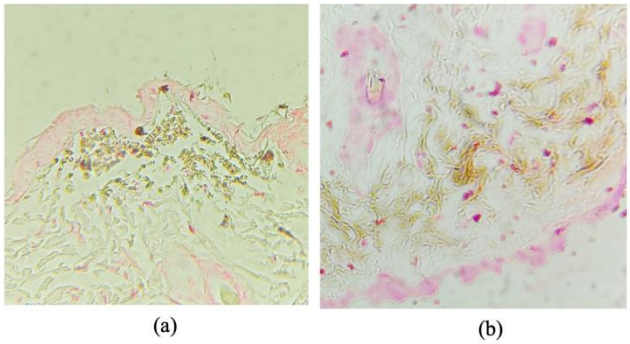

Figure 2: Histopathological examination of mouse skin tissue prepared with Masson–Fontana staining: (a) before UVB exposure and (b) after 14 days of UVB exposure

Figure 3 depicts the impact of 14 days of UVB exposure on total melanin content in the skin. The results showed that UVB exposure in the group after this period can cause hyperpigmentation in mouse skin, indicated by an increase in melanin content. This finding aligns with previous research, which suggests that UVB can stimulate melanin synthesis via signalling pathways that involve the increased production of reactive oxygen species, leading to oxidative stress and damage to proteins that regulate melanogenesis.[17,18] UVB promotes the increased expression of MITF, a crucial regulator of melanogenesis that controls genes such as tyrosinase and TYRP1, which are essential for melanin synthesis.[19] This study supports findings that UVB exposure increases MITF activity, leading to skin hyperpigmentation, a process involved in melanin production in the epidermis.[19–21]

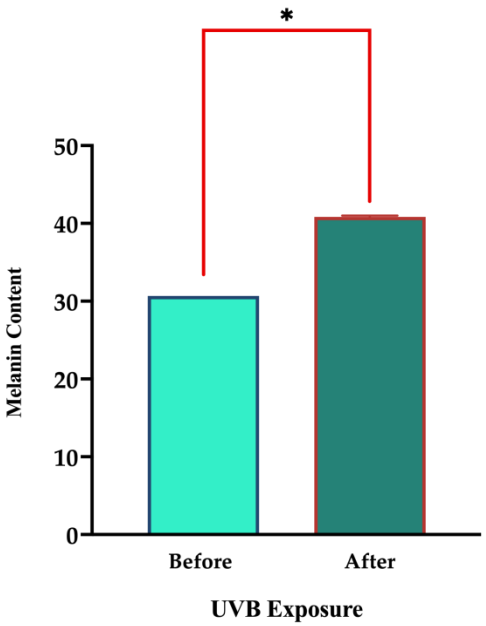

Figure 3: The red line and the asterisk above the bars denote that a paired t-test was performed, and the results were statistically significant. * Indicate statistically significant differences ($p < 0.05$)

Effect of Hydroquinone and Mulberry Extract-Containing Serum on Melanin Content in UVB-Induced Mice

Figure 4 illustrates the effects of hydroquinone and the serum containing mulberry extract on melanin levels in UVB-exposed mice. The results showed that both hydroquinone and the mulberry extract serum efficiently decreased UVB-induced melanin production, with hydroquinone displaying a more significant depigmenting effect—consistent with its mechanism of action.[12] The significant differences confirmed via ANOVA analysis ($p < 0.05$) indicate that mulberry extracts can induce hyperpigmentation. This observation supports previous research findings suggesting the potential therapeutic use of natural extracts,[22,23] such as mulberry, as safer alternatives to conventional depigmenting agents, including hydroquinone. These results imply that mulberry extract may be a natural and effective option for treating hyperpigmentation caused by UV exposure.

The skin tissue in the hydroquinone group exhibited a dense accumulation of melanin granules that extended into the suprabasal layer. The group treated with 5% mulberry extract serum showed fewer melanin granules, which remained somewhat dense and were present in multiple areas. In the 10% mulberry extract serum group, there was a clear reduction in staining intensity, with fewer granules dispersed more sparsely and displaying a paler colour.

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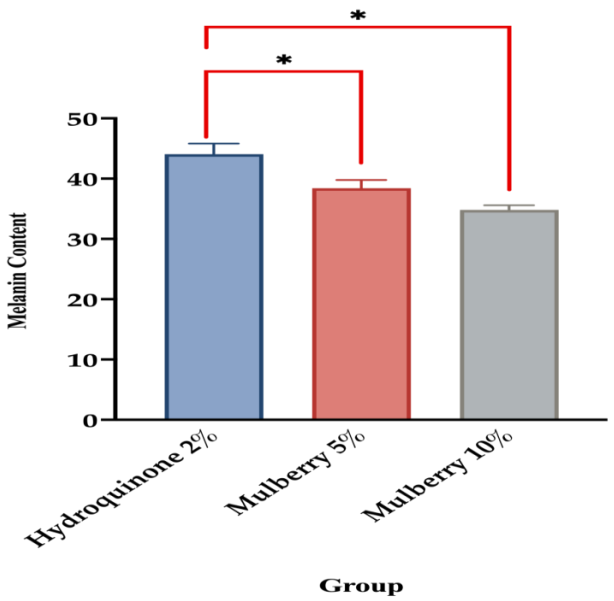

Figure 4: Effect of hydroquinone and mulberry extract serum on melanin content in UVB-exposed mice. The graph shows a significant reduction in melanin levels in the treatment groups compared with the UVB-only group.

Effect of Hydroquinone and Mulberry Extract–Containing Serum on the Level of MITF in UVB-Induced Mice

As presented in Table 1, a notable decline in MITF levels was evident after treatment with mulberry extract serum, especially at the 10% dosage. This reduction suggests that mulberry extract effectively inhibits the activity of MITF, a key transcription factor that regulates melanogenesis.[11–13] The inhibition of MITF resulted in decreased melanin production, as evidenced by histopathological findings that revealed a reduction in both the quantity and pigmentation of melanin granules in the treated groups compared with the negative control.

These results agree with earlier research reporting that natural extracts rich in flavonoids and phenolic compounds, such as those found in mulberry, can suppress MITF expression and reduce tyrosinase activity, thereby lowering melanin synthesis. The dose-dependent decrease suggests that higher concentrations of mulberry extract have a more substantial effect, which is promising for developing natural skin-lightening agents with fewer side effects compared with conventional options such as hydroquinone.[24–26] This study confirms the effectiveness of mulberry extract as a depigmenting agent by regulating melanogenic pathways.

Compounds derived from mulberry, such as maclurin and oxyresveratrol, considerably decrease melanin accumulation in skin cells exposed to UVB and in human skin models. This reduction primarily results from their ability to inhibit tyrosinase, a crucial enzyme for melanin production, and their strong antioxidant properties.[27]

Table 1: Effects of treatments on MITF levels in UVB-exposed mouse skin tissue

Group	MITF Level
Hydroquinone 2%	34,39±1.32
Mulberry extract containing serum at 5% consentration	32,02±4.4
Mulberry extract containing serum at 10% consentration	28,70±1.70

Nb: Different letters within the same column indicate statistically significant differences ($p < 0.05$).

Mulberry extracts are rich in antioxidants and have demonstrated protective effects against UV-induced skin cell damage, including reduced cell death and modulation of key signalling pathways involved in inflammation and survival, such as the mitogen-activated protein kinase, mechanistic target of rapamycin and nuclear factor kappa-light-chain-enhancer of activated B cells.[28] In vivo studies have shown that mulberry fruit extracts can improve skin moisture, reduce advanced glycation end-products and mitigate oxidative stress in ageing mice, which are relevant to skin health and pigmentation. The antioxidant properties of mulberry are believed to be the basis for its protective effects against UV-induced oxidative stress and inflammation, both of which contribute to the development of hyperpigmentation.[28,29] Compounds derived from mulberry, such as maclurin and oxyresveratrol, notably decrease melanin build-up in skin cells exposed to UVB and in human skin models. These effects are primarily due to the inhibition of tyrosinase, a vital enzyme in melanin production, and their strong antioxidant activity.[27]

Conclusion

This study demonstrated that exposing mice to UVB radiation for 14 days effectively induces hyperpigmentation, as indicated by increased melanin levels and enhanced MITF expression. Treatment with hydroquinone and mulberry extract serum resulted in a significant reduction of melanin accumulation and a decrease in MITF levels. Notably, a 10% mulberry extract concentration displayed a dose-dependent depigmenting effect similar to that of hydroquinone, highlighting its promise as a natural, safer alternative for UV-related hyperpigmentation. These results support the potential of mulberry extract to inhibit melanogenesis and serve as a new skin-lightening agent. However, further studies are needed to elucidate the molecular mechanisms and to assess the long-term safety and efficacy of mulberry-based products.

Conflict of Interest

The authors declare no conflict of interest.

Authors' Declaration

The authors hereby declare that the work presented in this article is original and that any liability for claims relating to the content of this article will be borne by them.

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