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 ORIGINAL ARTICLE

Thiamidol: A Breakthrough Innovation in the Treatment of Hyperpigmentation

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ABSTRACT

Cutaneous hyperpigmentation, including melasma, solar lentigines, and post-inflammatory hyperpigmentation (PIH), results in a significant impact on patients' quality of life. Unfortunately, many currently available over-the-counter (OTC) options have been limited by efficacy, safety, and tolerability concerns. Additionally, limited patient awareness and education on disease manifestation and root causes of hyperpigmentation often leave patients undiagnosed and untreated. Melanogenesis is driven by a complex pathway resulting in the ultimate production and deposition of melanin in the skin. The major rate-limiting step of melanogenesis centers on the conversion of L-Dopa to the final melanin product mediated by a cellular tyrosinase, causing the overproduction of melanin clinically resulting in hyperpigmentation. Recently, isobutylamido thiazolyl resorcinol (Thiamidol) has been identified as the most effective inhibitor of human tyrosinase out of 50,000 compounds screened, and thus, a novel ingredient for inclusion in OTC products to address hyperpigmentation. We describe here the current pre-clinical and clinical safety and efficacy data of Thiamidol formulations aimed at educating the dermatology community on a safe and effective OTC option for use as part of the overall management of hyperpigmentation in patients.

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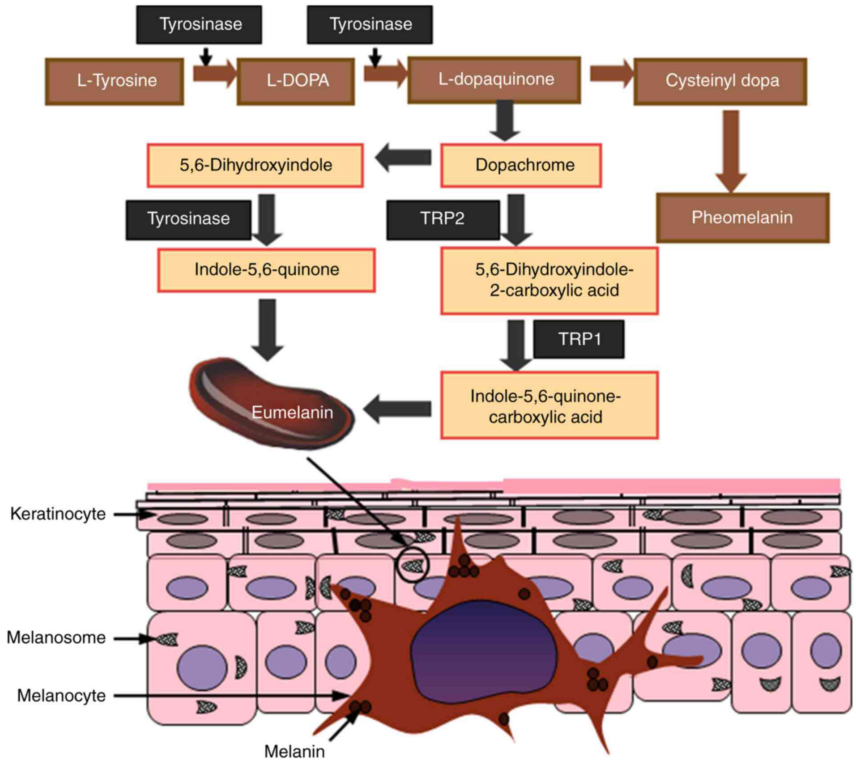
INTRODUCTION

Hyperpigmentation disorders, including melasma, post-inflammatory hyperpigmentation (PIH), dermal macular hyperpigmentation, seborrheic melanosis, hyperpigmentation associated with acanthosis nigricans, and solar lentigines, represent a set of pigmentary disorders that are usually characterized by overstimulation of the melanin production pathway, resulting in the deposition of excess melanin in the skin.[1-3] The stimuli of melanin production can include hormonal causes, ultraviolet (UV) radiation, endogenous factors (eg, inflammatory conditions), or exogenous factors (eg, mechanical trauma) in PIH.[1,3]

Pigmentary disorders, including hyperpigmentation, represent one of the most common dermatologic diagnoses in individuals with skin of color (SOC), particularly individuals with African, Asian, or Hispanic heritage.[4,5] In a recent survey of 48,000 individuals in 34 countries from December 2022-2023 responding to an online auto-administered questionnaire, 15% (n=7,126) of responders (56% women, 30% Fitzpatrick Skin Types (FST) IV-VI, mean age = 39 years) reported suffering from PIH.[6] For melasma, the prevalence in the general population is approximately 1% but has been reported as high as 50% in high-risk populations and accounts for between 4 to 10% of dermatology diagnoses in Central and South America.[3,7] The incidence of solar lentigines increases with age, affecting more than 90% of individuals with FSTs I-II older than 50 years.[8]

Effective treatment options for hyperpigmentation include agents that reduce melanin synthesis or enhance the dispersion and removal of melanin once formed.[9] Prior to the passing of the CARES Act on September 20, 2023, 2% hydroquinone

FIGURE 1. Melanogenesis pathway and melanin transport.



TRP1, tyrosinase related protein-1; TRP2, tyrosinase related protein-2; L-DOPA, L-3, 4-dihydroxyphenylalanine. (Adapted from Qian et al).[11]

was widely utilized by consumers as an over-the-counter (OTC) option for treatment of hyperpigmentation; however, its removal from the market created a major gap in OTC options, particularly for patients with SOC and other underrepresented minorities who struggle with access to care.[9]

Recently, isobutylamido thiazolyl resorcinol (Thiamidol) was identified as the most effective inhibitor of human tyrosinase out of 50,000 compounds screened,[10] and thus, a novel ingredient for inclusion in OTC hyperpigmentation products. We review here the current pre-clinical and clinical studies on Thiamidol formulations aimed at educating the dermatology community on a safe and effective OTC option for use as part of the overall management of hyperpigmentation in patients.

MATERIALS AND METHODS

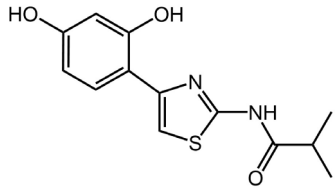
A literature search was conducted in February 2025 on PubMed and Google Scholar databases using the search terms Thiamidol or isobutylamido thiazolyl resorcinol. This yielded a total of 21 articles which were evaluated for relative information on the discovery and chemistry of Thiamidol, including in vitro analysis, as well as clinical studies investigating Thiamidol as a treatment or prophylaxis for hyperpigmentation.

Melanogenesis and Role of Tyrosinase Activity in Hyperpigmentation

Melanin is primarily synthesized in melanocytes, where it is packaged in specialized organelles called melanosomes.[11] The production of melanin is a complex process with multiple stages, which when altered may lead to pigmentation defects including both hypo- and hyperpigmentation, with or without a change in the number of melanocytes.[12-14] At the center of melanin synthesis is the enzyme tyrosinase which is involved in multiple steps during melanin biosynthesis, including the rate-limiting step of conversion of tyrosine to L-DOPA (Figure 1), eventually leading to the production of 2 types of melanin: eumelanin and pheomelanin.[11] Eumelanin is a soluble polymer that is brown-black in color, while pheomelanin is light red-yellow in color.[15]

Since tyrosinase plays an essential role in melanogenesis, its inhibition is one of the most targeted approaches for addressing hyperpigmentation.[11,16] Tyrosinase inhibitors include true inhibitors, specific tyrosinase inactivators, and alternative tyrosinase substrates, and can be derived from natural sources such as fungi, bacteria, and plants, or from semi-synthetic and fully synthetic sources.[17] One of the main challenges with tyrosinase

FIGURE 2. Chemical structure of Thiamidol.



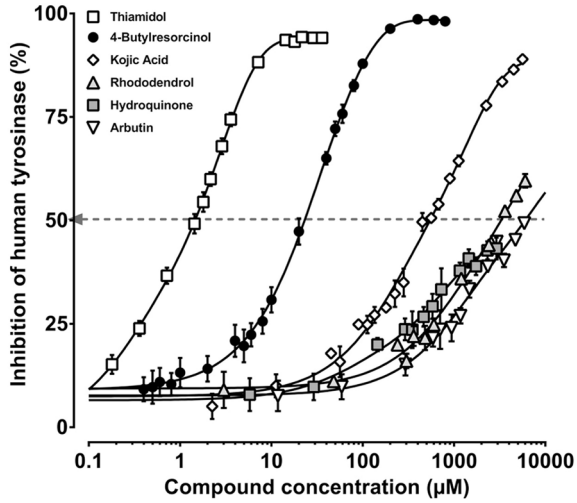
N-(4(2,4-dihydroxyphenyl)thiazol-2-yl)isobutyramide; Isobutylamido-Thiazolyl-Resorcinol.

inhibitors is the concern with safety and instability when used for extended periods and high concentration.[18] Anti-pigmentation ingredients including arbutin, kojic acid, deoxyarbutin, and hydroquinone have also been shown to be toxic to melanocytes.[17] To date, hydroquinone remains the most widely used agent for the treatment of hyperpigmentation.[19] However, concerns with side effects including irritant dermatitis, exogenous ochronosis, and in rare cases permanent leukoderma,[17,20] have resulted in the use of hydroquinone for short durations and only by prescription in the United States,[21] and a complete ban from use in OTC products as previously described. These safety concerns have left a significant gap in OTC treatment options for patients with limited access to dermatology health professionals or limited resources.

Discovery of Isobutylamido-Thiazolyl-Resorcinol, Thiamidol

A key aspect associated with the lack of efficacy of many previously identified tyrosinase inhibitors may be due to the use of tyrosinase isolated from the mushroom *Agaricus bisporus* as the target,[22,23] which has been shown to be significantly different structurally compared to the human tyrosinase enzyme.[10,24,25] A recent screen of 50,000 compounds yielded derivatives of thiazolyl-resorcinol, which were optimized for compatibility with topical formation resulting in the generation of Thiamidol

FIGURE 3. Inhibition of human tyrosinase by Thiamidol, 4-butylresorcinol, kojic acid, rhododendrol, hydroquinone, and arbutin.

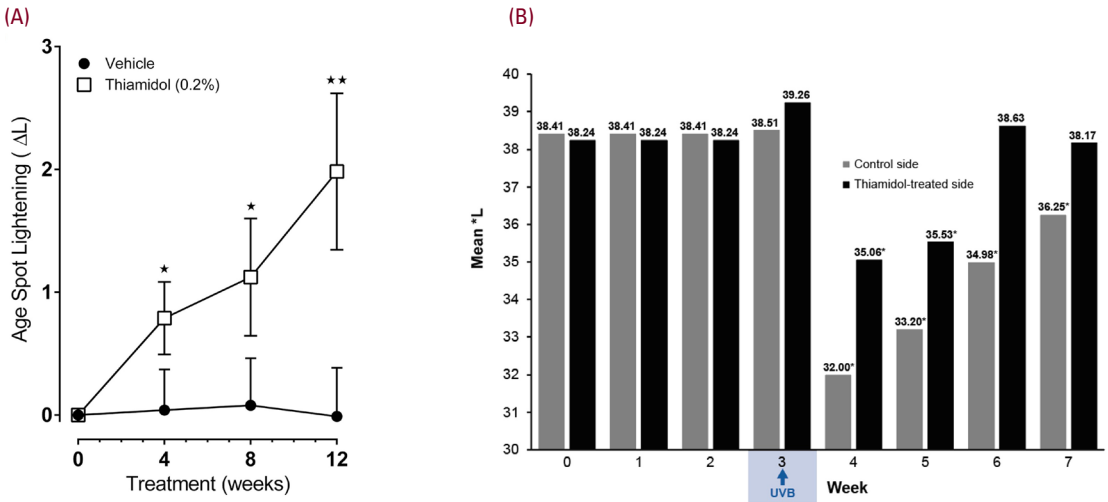


In vitro assays using purified hTyr in 50 mmol/L sodium phosphate buffer, pH 7.0, at a substrate (L-dopa) concentration of 1 mmol/L and various concentrations of inhibitors as noted. Data represent the mean ± standard deviation of three independent experiments. Kinetics of inhibition of hTyr by Thiamidol (Beiersdorf AG, Hamburg, Germany) at the concentrations noted. The experiment was performed in triplicate at pH 7.0. The data are plotted according to Lineweaver-Burk (with permission from Mann et al, 2018).[10]

(Figure 2), the most potent derivative, with a half-maximal inhibitor concentration (IC_{50}) of 1.1 mmol/L, and near 100% inhibition of human tyrosinase at 10 mmol/L (Figure 3). The IC_{50} of Thiamidol is approximately 20X lower than 4-butylresorcinol (IC_{50} = 21 mmol/L) and approximately 4000X lower than hydroquinone (IC50 = >4000 mmol/L).[10]

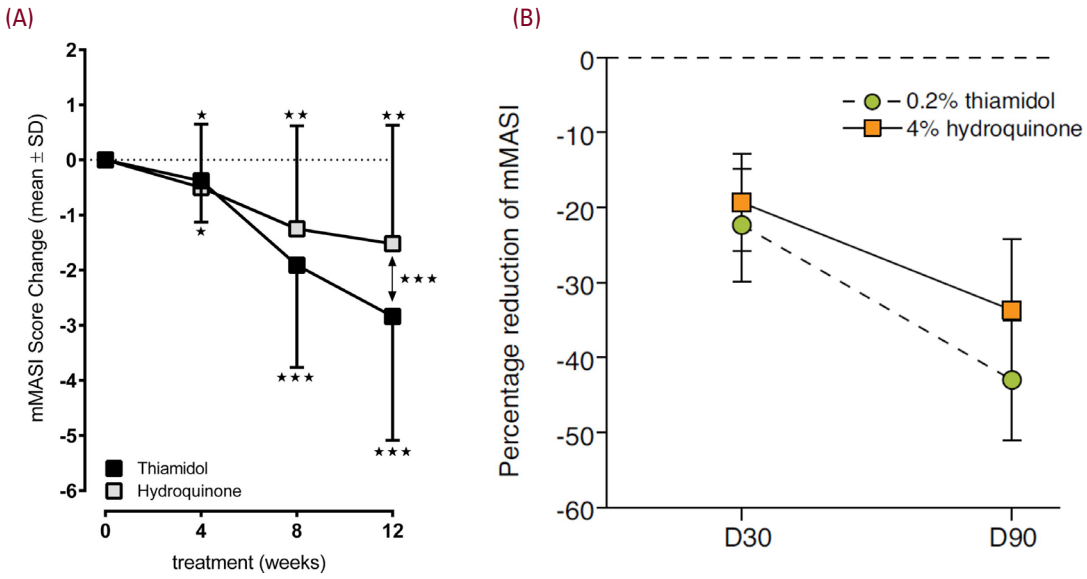
Interestingly, Thiamidol had limited efficacy for inhibiting mushroom tyrosinase, clearly demonstrating the significant difference in structural components between the human and mushroom tyrosinase.[10]

FIGURE 4. Impact of Thiamidol on UVB-induced hyperpigmentation.



A) Solar lentigines on the volar forearms of each subject were treated twice daily for 12 weeks with 0.2% Thiamidol (Beiersdorf AG, Hamburg, Germany) or with the vehicle only as a control using a spot applicator. Efficacy was evaluated after 4, 8, and 12 weeks. Data represent the mean ± standard error of the mean of 17 subjects. * $P<0.05$, ** $P<0.01$; statistically significant versus the control (with permission from Mann et al, 2018). (B) Mean lightness index (L*) of Thiamidol treated side and control side compared to baseline at each visit. *Significant difference compared with baseline, $P<.05$ (adapted from Vachiramon et al, 2021).[27]

FIGURE 5. Mean mMASI scores before and after treatment with Thiamidol or hydroquinone.



(A) Time course of mMASI score changes compared to the baseline. Scores were assessed after 4, 8, and 12 weeks of treatment. Data are reported as means ± SD. Significant differences as marked in comparison to baseline and hydroquinone (* P<0.05, ** P<0.01, *** P<0.001). mMASI, modified Melasma Area and Severity Index; SD, standard deviation. (B) Mean (CI 95%) percentage reduction of modified Melasma Area and Severity Index (mMASI) scores at D30 and D90 for the 0.2% Thiamidol and 4% hydroquinone groups. (with permission from Arrowitz, and Lima)[28,30]

Clinical Evaluation of Thiamidol for Treatment of Hyperpigmentation

Ultraviolet-Induced Hyperpigmentation

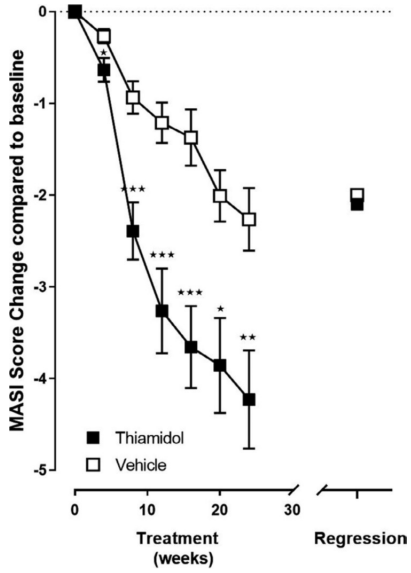
Exposure to UV radiation results in the proliferation of melanocytes and the accumulation of melanin in keratinocytes leading to solar lentigines.[26] As previously mentioned, solar lentigines are quite common over the age of 50, especially in individuals with skin phototypes I-III.[8] The utilization of Thiamidol in both treatment and prevention of UVB-induced solar lentigines and hyperpigmentation has been reported in two studies.[10,27] Mann et al, reported on the treatment of lentigines on the volar forearms of subjects (n = 19, no ethnicities nor FSTs reported), treated twice daily for 12 weeks with a 0.2% Thiamidol formulation vs vehicle only, demonstrating improvements in lentigines as early as week 4 with continued improvement up to the end of the study, week 12 (Figure 4A).[10] Vachiramon et al, demonstrated that Thiamidol followed by UVB irradiation resulted in hyperpigmentation with a significantly lower mean lightness index (skin lightness on a gray scale from 0-100, where 0 = total black and 100 = total white) as measured by colorimeter compared to vehicle control in healthy volunteers (n = 30, FSTs II-IV, no ethnicities reported). Pre-treated sites also resumed normal skin color earlier than control sites and clinical evaluation by blinded physician evaluators and subjects favored the Thiamidol-treated sites over control (P<0.05; Figure 4B).[27]

Melasma

Thiamidol treatment for melasma has been evaluated in several clinical studies, including two head-to-head studies vs hydroquinone (2% and 4%).[28-30] In an exploratory double-blind, randomized, split-face study, 0.2% Thiamidol was compared to 2% hydroquinone (n = 28; Asian 35.7%, Caucasian, 60.7%, multiracial, 3.6%; no FSTs reported) in subjects with mild-to-moderate melasma who applied the test compounds to either side of the face twice daily. 78.6% of subjects demonstrated improvements in modified Melasma Area and Severity Index (mMASI) at 12 weeks after using Thiamidol, compared to 60.7% of subjects demonstrating improvement in mMASI at 12 weeks after using 2% hydroquinone. 10.7% of hydroquinone-treated subjects showed worsening of melasma while none in the Thiamidol group experienced worsening.[28] Additionally, the mMASI scores on the Thiamidol-treated side of the face were significantly reduced by -2.84 + 1.87 (P<0.001) vs a decrease of -1.52 + 2.15 (P=0.002) for the hydroquinone side, with statistical superiority in favor of the Thiamidol side vs hydroquinone (P<0.001; Figure 5A).[28]

Lima et al, compared the efficacy of 0.2% Thiamidol (applied twice daily) vs 4% hydroquinone (applied at bedtime) for 90 days in patients (n=50; FSTs II-V; no ethnicities reported) with clinically diagnosed facial melasma, with both groups demonstrating a reduction in mMASI, MELASQoL, and color contrast scores (P<0.01).[30] The mean reduction of mMASI was 43% and 33% for the Thiamidol and hydroquinone group, respectively, with no statistical difference between the two groups (Figure 5B).[30] Lastly, Roggenkamp, evaluated the therapeutic benefits of a Thiamidol-based treatment regimen (Thiamidol serum with hyaluronic acid and licochalcone A, twice daily, Thiamidol day

FIGURE 6. Changes of Melasma Area and Severity Index (MASI) score (mean ± standard error of the mean) versus baseline at every time point measured after treatment with Thiamidol or vehicle.



Significant improvement in comparison to baseline for Thiamidol and vehicle at all points in time. Significant differences between Thiamidol and vehicle as indicated (*P<0.05, **P<0.01, ***P<0.001). After 13 to 20 weeks of cessation of all treatment (regression), including sunscreen use, there was a significant difference for both treatments compared to baseline (P<0.005) (with permission from Roggenkamp)[29]

cream with SPF and licochalcone A once daily, and Thiamidol night cream once daily) in subjects (n = 48; Caucasian 1.9%, multiracial, 67.9%, Indian, 30.2%; FSTs II-V) with moderate-to-severe melasma for 24 weeks with a subsequent regression phase.[29] The decrease in MASI score was observed as early as week 4 with continued improvement through week 14, with statistical significance between the Thiamidol regimen group and baseline and the vehicle control (same skin care routine without Thiamidol) at all timepoints (Figure 6).[29] Following the regression phase (13-20 weeks post-end of treatment period), there remained a statistically significant reduction in MASI score compared to baseline (Figure 6).[29]

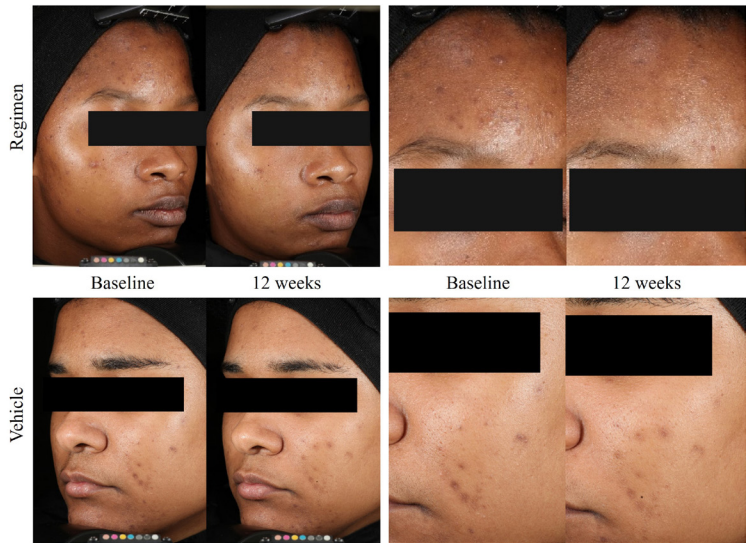
Post-inflammatory Hyperpigmentation

Acne-Vulgaris Induced PIH

Acne-vulgaris is a leading cause of PIH, especially in patients with SOC (FST III-VI).[31] The pigmentation alteration can be long lasting and significantly affect patients' quality of life (QoL).[31] Efforts to treat acne-induced PIH with Thiamidol have been clinically evaluated in a single-blinded, vehicle control, single-center study.[32] Subjects with FST V, with a history of acne and PIH (n = 64, no ethnicities reported) were treated with either a thiamidol formulation (Thiamidol formulated in an alcoholic gel without SPF, no concentration reported) or a vehicle control twice daily for 12 weeks. At week 12, visible changes in PIH were observed (Figure 7A) and patients reported a significant improvement in their hyperpigmentation after Thiamidol use.[32]

In a separate, cosmetic clinical observational study conducted on 32 subjects with FST V-VI (self-reported of African ancestry) Roggenkamp et al, evaluated the efficacy of a Thiamidol-based regimen (Thiamidol serum twice daily, Thiamidol day cream with SPF once daily, and Thiamidol night cream once daily) for treatment of acne-related PIH. At week 12, the Thiamidol-based regimen led to a significant improvement in the melanin index score, with no surrounding lesional hypopigmentation, often seen with the use of hydroquinone (Figure 8).[32]

FIGURE 7. Treatment of acne-induced PIH in dark-skinned individuals.



Representative images of subjects with acne-induced PIH at baseline and after 12 weeks of treatment with the Thiamidol-containing regimen or the vehicle. (with permission from Roggenkamp et al, 2020).[32]

FIGURE 8. Treatment with a skin care regimen of three Thiamidol-containing products.

(A) Representative images of a subject at baseline and after 4, 8, and 12 weeks of treatment with the Thiamidol-containing skin care regimen (day cream, night cream, and dual serum). (B) Melanin index scores of lesional and perilesional skin after 4, 8, and 12 weeks of treatment. Data are depicted as mean ± SD. Significant differences are marked in comparison to the baseline (***P<0.001) (with permission from Roggenkamp)[32]

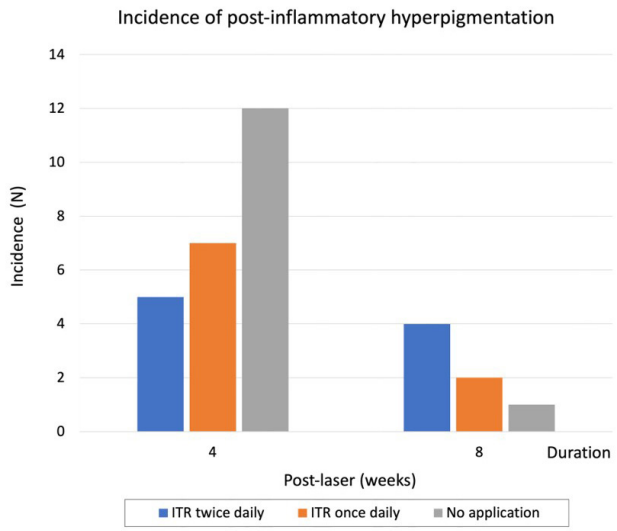
Laser-Induced Post-Inflammatory Hyperpigmentation

Laser treatments are often used as a therapeutic modality for hyperpigmentation; however, the possibility of post-laser PIH or further exacerbation of these conditions is of concern, especially in patients with SOC.[33,34] Recently, a randomized, evaluator-blinded study in patients with solar lentigines (n=24, no ethnicities nor FSTs reported) was conducted to evaluate the efficacy of a Thiamidol for preventing laser-induced PIH.[33] Subjects applied a Thiamidol-based spot corrector formulation once daily, twice daily, or no application for 2 weeks. After 2 weeks of pre-treatment, lesions were treated with a single session of 532-nm Q-switched Nd:YAG laser and then assessed by digital photographs, Antera 3D, Colorimeter, and PIH self-grading (10 cm, VAS) on days 28, 42, and 70. The incidence of PIH at week 4 after laser treatment was statistically lower in the Thiamidol twice daily group compared to the no-application group (20.83% vs 50%, *P*=0.028; Figure 9).[33]

Adjunctive Use of Thiamidol for Treatment of Hyperpigmentation

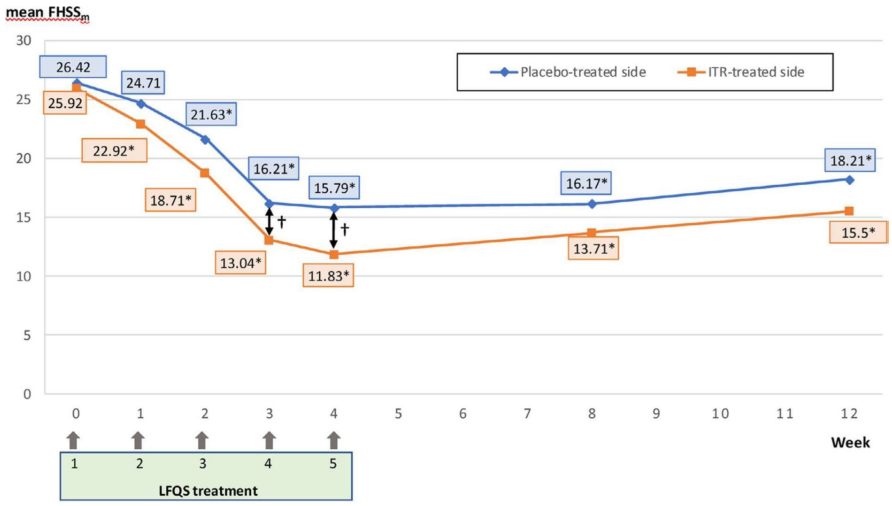
Therapeutic treatment of hyperpigmentation involves a multi-modal approach that includes photoprotection, topical and systemic therapies, and in some instances procedural approaches including chemical peels, micro-needling, and laser/light-based therapies; and requires personalized regimen on a case-by-case basis.[35] It's recognized that Thiamidol when utilized, will often be part of a multi-arm therapeutic plan. Efforts to address its compatibility have been evaluated in two separate studies. The first involves the adjunctive use of Thiamidol with low-fluence Q-switched Nd:YAG laser (LFQS) treatment and the second evaluated the replacement of 5% hydroquinone with Thiamidol in Kligman's trio formulation.[36,37] Vachiramon et al, demonstrated the superiority of Thiamidol combined with LFQS vs LFQS alone based on the Mean Facial Hyperpigmentation Severity Score of the malar area ($FHSS_m$) (Figure 10) in patients with symmetrical facial hyperpigmentation on both malar areas (n=25, no ethnicities nor FSTs reported),[36] while

FIGURE 9. Comparison in the incidence of post-inflammatory hyperpigmentation between prophylactic Thiamidol-treatment twice daily, Thiamidol-treatment once daily, and no application at 4- and 8-weeks post-laser.



(With permission from Vachiramon et al, 2024).[33]

FIGURE 10. Mean Facial Hyperpigmentation Severity Score of the malar area (FHSSm).



LFQS, low-fluence Q-switched Nd:YAG 1,064-nm laser; ITR, Isobutylamido thiazolyl resorcinol, Thiamidol. *significantly different from baseline; †significantly different compared between groups (Adapted from Vachiramon et al, 2021).[36]

Bertold et al, demonstrated non-inferiority in modified MASI (mMASI) between a modified Kligman's trio (5% hydroquinone, 0.1% retinoic acid, and 0.1% dexamethasone acetate) vs Thiamidol trio (0.1% Thiamidol, 0.1% retinoic acid, and 0.1% dexamethasone acetate; Figure 11A), with statistically significant improvement at week 12 in the MelasQoL for the Thiamidol Trio group compared with the Kligman's trio group in subjects with clinically diagnosed melasma (n=36; Caucasian, 59%, African, 38.4%, Asian, 2.6%; FSTs II-V; Figure 11B).[37]

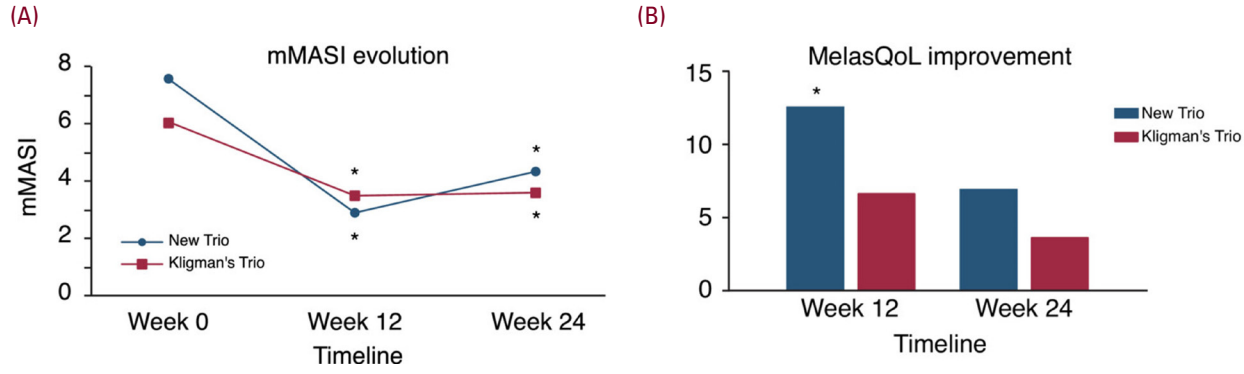
Thiamidol Safety and Tolerability

While none of the clinical studies evaluated focused specifically on safety and tolerability, most studies reported no significant adverse events associated with Thiamidol formulations, regimens, or use as adjunctive therapy. In the head-to-head study of Thiamidol vs 4% hydroquinone, Lima et al, reported that 2 Thiamidol-treated patients (8%) developed allergic contact dermatitis at days 60 and 75 of follow up.[30]

Study Limitations

This review is limited by studies conducted primarily on female participants, without full demographic information regarding race, ethnicity, or FSTs for several studies, and with limited information on efficacy, safety, and tolerability in men. Additionally, many of the studies were conducted and/or sponsored by Beiersdorf AG, the developers of Thiamidol; thus, independent clinical studies would be beneficial.

FIGURE 11. Clinical evaluation of Thiamidol trio vs Kligman's trio in melasma.



(A) Evolution of the modified Melasma Area Severity Index (mMASI) score. Ordinate represents the mean mMASI score of the subjects of each treatment arm in points. mMASI values range from 0 to 24. (B) Improvement of the melasQoL score. Ordinate represents the mean melasQoL score improvement of the subjects of each treatment arm in points. melasQoL values range from 0 to 70. *$P<0.05$. (With permission from Bertold et al)[37]

CONCLUSION

Skin hyperpigmentation, including melasma, solar lentigines, and post-inflammatory hyperpigmentation, results in a significant impact on patients' quality of life. It has been shown that the major rate-limiting step of melanogenesis centers on tyrosinase enzyme activity within the melanocyte that results in the overproduction of melanin. Currently available over-the-counter (OTC) treatments are limited by efficacy, safety, and tolerability concerns. Recently, isobutylamido thiazolyl resorcinol has been identified as an effective inhibitor of human tyrosinase and melanin production, with an IC50 = 1.1 mmol/L as compared to hydroquinone's IC50 > 4000 mmol/L. In clinical studies,Thiamidol has been shown to reduce hyperpigmentation in solar lentigines, mild-to-severe melasma, and acne-, and laser-induced PIH and prevent and enhance the resolution of UV-induced pigmentation. Additionally, Thiamidol has been shown to work as well as the Kligman formula when used in combination with tretinoin and dexamethasone acetate and to be suitable adjunctive therapy with commonly used lasers. From these results,Thiamidol appears to be a safe and effective ingredient that should be considered when recommending an OTC option as part of the overall treatment regimen for patients with hyperpigmentation.

DISCLOSURES

CF is an investigator affiliated with Novartis. Additionally, she serves as a consultant or advisor for Galderma, Sun Pharma, Avita, Procter & Gamble, Regeneron, Aerolase, Benev, L'Oréal, La Mer, Avène, Kenvue, Bristol Myers Squibb, Nutrafol, Acclaro, Beiersdorf, Primus, Johnson & Johnson, Crown Aesthetics, and AbbVie. PG has served as an investigator and/or consultant for Allergan, BOD Clinuvel, Clinuvel, Incyte, Johnson & Johnson, L'Oréal, Merck, Pfizer, SkinBetterScience, and Versicolor Technologies. VDC has served as an investigator for Allergan Aesthetics, Avava, Eirion Therapeutics, Incyte, Janssen, Lilly, Pfizer, Prollenium, Regeneron, Symatese, and Teoxane; as a consultant for Almirall, Beiersdorf, Estée Lauder, Juenes Aesthetics, L'Oréal, Ortho Derm, Oruka Therapeutics, and Skinceuticals; and as a speaker for Aerolase, Arcutis, L'Oréal, and Pfizer. She has also received royalties from UpToDate. AA has served as an investigator from AbbVie, Amgen, Arcutis, Castle, Dermavant, Galderma, Incyte, and Leo; as a consultant for AbbVie, Allergan, Almirall, Alphyn, Amgen, Apogee, Arcutis, Bausch Health, Beiersdorf, BMS, Boehringer Ingelheim, Canfield, Castle, Dermavant, Dermsquared, Galderma, Genentech, Genzyme, Incyte, Janssen, L'Oreal, Leo, Lilly, Ortho, Pfizer, Sanofi-Regeneron, Swiss American, Symrise, UCB, and VisualDx; and as a speaker for Aerolase, Janssen, L'Oréal, Regeneron, Sanofi-Genzyme, and Scientis. He has also received equipment from Aerolase and royalties from Elsevier, Springer, Wiley-Blackwell, and Wolters Kluwer Health. HB has served as a consultant and/or speaker for Avene, Beiersdorf, Johnson and Johnson, Kenvue, L'Oréal, and La Roche-Posay. NE has served as an investigator for Pfizer; and as a speaker and/or advisory board member, for AbbVie, Allergan, Avita, Beiersdorf, Canfield, Dior, Galderma, Incyte, Janssen, L'Oréal, La Roche-Posay, Lilly, McGraw Hill, Medscape, Pfizer, Sanofi, Takeda, and VisualDx. She has also received royalties from McGraw-Hill and has stock options in VisualDx. PF serves as an advisor, consultant or speaker for Advanced Biomedical, Avene AlumierMD, Beiersdorf, Bubble, Cerave, Estee Lauder, Kenvue, La Roche Posay, L'Oreal, Nutraceutical Wellness, RATIONALE, Skin Better Science, Skinceuticals and Skinmedica.ST has served as an investigator for Allergan Aesthetics, Concert Pharmaceuticals, Croma-Pharm GmbH, Lilly, and Pfizer; as a consultant for Arcutis, Armis, Avita Medical, Beiersdorf, Biorez Inc, BMS, Cara Therapeutics, Dior, EPI Health, Estee Lauder, Evolus Inc, Galderma, GloGetter, Hugel America, Incyte, Johnson & Johnson, L'Oréal, Lilly, Medscape, Pfizer, Piction Health, Sanofi, Scientis US, UCB, Vichy Laboratories; as a speaker for Beiersdorf, Catalyst Medical Education, CME Outfitters, DermSquared, HMP Global, LearnSkin, Medscape, and MJH Lifesciences; and as an editorial board member for *Archives in Dermatologic Research*, *Cutis*, and *Practical Dermatology*. She has also received royalties from McGraw-Hill. SRD has served as an investigator and/or consultant for multiple entities in the past, including Beiersdorf. He has also held multiple leadership positions in organized medicine.

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