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Skin-attachable OLED electroceutical for chronic dermatitis treatment

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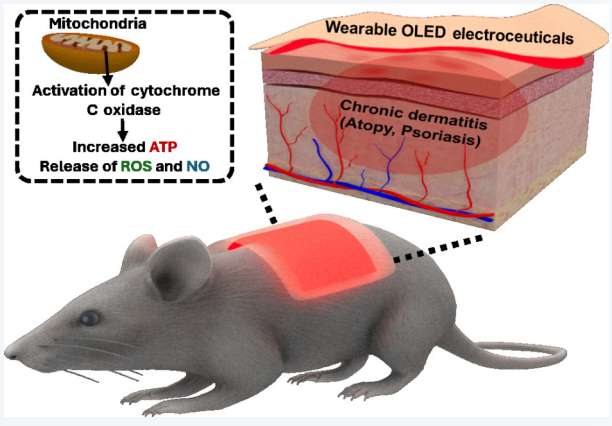
ABSTRACT: Wearable electroceuticals are emerging as a key platform for health management due to their safety and non-invasiveness, with therapeutic efficacy enhanced by close skin contact. In this study, we present a wearable organic light-emitting diode (OLED) patch that integrates flexible hardware and a flexible battery for self-operation and reusability. The OLED exhibits excellent mechanical flexibility, adhering intimately to the skin while maintaining a safe temperature range of 20 °C at 5 mW/cm², stable operation for over 1000 h, and high moisture resistance. Daily irradiation with 632 nm OLED light (9 J/cm²) in mouse models of psoriasis and atopic dermatitis (AD) led to significant recovery of epidermal thickness and transepidermal water loss (TEWL) to near-normal levels. The expression of inflammatory cytokines (IL-4, IL-17, and IL-22) also tended to be suppressed. These results suggest that wearable OLED-based phototherapy can complement the limitations of existing treatments and offer a self-administered treatment strategy.



KEYWORDS: organic light-emitting diode, electroceutical, atopic dermatitis, psoriasis, *in vivo*

1 Introduction

With the increasing prevalence of chronic diseases alongside an aging population, traditional treatment-focused medical systems are facing limitations, emphasizing the need for new approaches that enable preventive health management [1]. Effective management of chronic diseases requires long-term monitoring and continuous therapeutic intervention, leading to the emergence of electroceuticals as next-generation platforms that modulate physiological functions through electrical, electromagnetic, and optical stimulation [2–4]. Among these, photomedicine [5], including optogenetics [6], photodynamic therapy (PDT) [7], and photobiomodulation (PBM) [8] is being evaluated as safe and noninvasive method that offers a variety of treatment options. In particular, organic light-emitting diode (OLED)-based devices are well suited for wearable applications owing to their inherent mechanical flexibility, lightweight structure, and low heat generation [9]. These characteristics allow conformable contact with the skin and uniform surface emission, ensuring efficient light delivery for therapeutic use.

Previous studies have demonstrated the phototherapeutic potential of OLED-based devices. Flexible OLEDs operated at 6 mW/cm² for 3 h effectively inhibited *Staphylococcus aureus* (*S. aureus*) growth [10]. In a glioma mouse model, irradiation at 3 mW/cm² for 3.7 h suppressed tumor growth despite low power

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operation, confirming their suitability for PDT, which typically requires cumulative doses of 50–100 J/cm^2 [11, 12]. Beyond PDT, OLED-based PBM has been reported to promote keratinocyte proliferation and wound healing under 670 nm and 6 J/cm^2 conditions, as well as improve metabolic indices in diabetic rat using an implantable catheter-type OLED [13–15]. PBM exhibits a biphasic dose response in which insufficient stimulation yields limited biological activity, whereas excessive irradiance may diminish therapeutic efficacy [16]. For chronic diseases, gradual and controlled light exposure is considered more physiologically appropriate than brief high-power stimulation. In this regard, the unique advantages of OLED technology enable repeated, prolonged, low-intensity light irradiation.

Atopic dermatitis (AD) and psoriasis are representative chronic dermatitis characterized by inflammatory reactions, impaired skin barrier function, and severe itching [17]. While prompt treatment is necessary, many patients face difficulties in receiving ongoing hospital-based care due to time and financial constraints, which can delay treatment and easily lead to chronic progression [18]. Currently, widely used treatments include drug therapy such as topical steroids and immunomodulators, and ultraviolet (UV)-based phototherapy. While drug therapy is effective in suppressing inflammation in the short term, but there are concerns about side effects such as skin atrophy, pigmentation, and drug resistance when used repeatedly [19]. On the other hand, UV (UVA, UVB) LASER is considered a relatively noninvasive and stable treatment [20]. However, LASERs rely on fixed equipment in hospitals and require precise irradiation distance, time, and intensity settings that are not harmful to the human body, which can reduce treatment efficacy.

To overcome these limitations, various light source-based approaches have been explored. For example, 630 nm OLED-based PDT has been reported to improve psoriasis by applying a photosensitizer to the lesion and irradiating it to induce targeted tissue damage [21]. However, PDT requires high irradiance, and the associated thermal load may raise safety concerns when implemented on skin-attachable platforms. Another study reported nuclear factor kappa-B (NF-κB)-mediated anti-inflammatory effects in *in vitro* and *in vivo* lipopolysaccharide (LPS)-induced inflammation models using a 630 nm OLED based on PBM, suggesting potential therapeutic efficacy in AD [22]. Although not OLEDs, preliminary clinical study using a combination of 830 and 633 nm light-emitting diodes (LEDs) for refractory psoriasis further supports the broader therapeutic promise of light-based interventions in psoriasis and AD [23]. While near-infrared (NIR) wavelengths can penetrate deeper tissue layers and contribute to pruritus relief, this study aimed to evaluate the therapeutic feasibility of wearable OLED-based PBM for chronic inflammatory dermatitis. Since the pathological features of psoriasis and AD appear mainly in the epidermis and upper dermis, the red wavelength was selected to provide sufficient light stimulation while minimizing unnecessary deep tissue exposure and thermal burden.

In this study, we developed a wearable OLED-based electroceutical for daily management of AD and psoriasis. The device integrates flexible circuitry, a compact battery, and an OLED that delivers uniform and stable light under mechanically and environmentally robust conditions. In chronic dermatitis models, periodic low-intensity irradiation (5 mW/cm^2) significantly reduced epidermal thickness, normalized transepidermal water loss (TEWL), and suppressed inflammatory cytokine expression, demonstrating the therapeutic efficacy of wearable OLED-based platforms.

2 Experimental

2.1 OLED performance

The optoelectrical performances were evaluated using a Current–Voltage–Luminance (*I*–*V*–*L*) test system (M6100, McScience Inc.). The power supply of the OLED was measured using a source meter (Keithley 2601A, Tektronix Inc.), and the luminescence characteristics were analyzed using a spectrometer (CS 2000, Konica Minolta Inc.). The operating temperature was measured using an infrared camera (HT-19, Dongguan Xintai Instruments Inc.), and the lifetime was measured using a lifetime test system (M6000, McScience Inc.).

2.2 Animal

Six-week-old male BALB/c mice were purchased from Orient Bio (Seongnam, Republic of Korea). All animal experiments were carried out according to the Gachon University Animal Research Guidelines (LCDI-2022-0101). The study was approved by Gachon University. Mice were acclimated for 1 week before immunization.

2.3 Induction of atopic dermatitis-like mouse model

The 6-week-old male BALB/c mice were divided into four groups, and each group consisted of five mice. Normal control was sensitized and challenged with phosphate-buffered saline (PBS). OXA group was treated with oxazolone (4-ethoxymethylene-2-phenyl-2-oxazolin-5-one; Sigma-Aldrich, St. Louis, MO, USA). Oxazolone (OXA) was dissolved in a 4:1 (v/v) vehicle mixture of acetone and olive oil. Briefly, the ears of BALB/c mice in both the oxazolone-treated group and the OLED-treated group were sensitized with 10 μL of 5% oxazolone on the first day. After the first challenge, 25 μL of 0.5% oxazolone was repeatedly applied to the dorsal skins for every two days (9 times). No substances were applied to the skin surface on the last day of the experiment. All OXA apply and other treatments were performed under light anesthesia with isoflurane. Every two days, characteristics of skin barrier function, including TEWL and skin thickness were evaluated. On the last day, mice were sacrificed, and skin and blood samples were collected for further analysis.

2.4 Imiquimod-induced psoriasis

The 6-week-old male BALB/c mice were divided into four groups, and each group consisted of six mice. For imiquimod (IMQ)-induced psoriasis, mice were shaved and completely unhaired with depilation cream on the back with 2 × 2 cm of size on day 1 prior to the first Aldara treatment. In the IMQ-treated group and the OLED-treated group, 83 mg Aldara cream (5% IMQ, iNova Pharmaceuticals Pty LTD., Chatswood, Australia) was applied on the shaved dorsal skin of mice for 11 consecutive days. In control of animals, vaseline was used and in the same amount. No substances were applied to the skin surface on the last day of the experiment. All IMQ applications and other treatments were performed under light anesthesia with isoflurane. Every two days, characteristics of skin barrier function, including TEWL and skin thickness were evaluated. After sacrificing the mice, skin and blood samples were collected for further analysis.

2.5 Light irradiation

After atopic dermatitis and psoriasis induction, the dorsal skin of mice was irradiated with 4.5 (15 min) and 9 (30 min) J/cm^2 at a wavelength of 632 nm using an OLED device for 10 and 8 days, respectively.

2.6 Measurement of TEWL

DERMALAB TEWL device (Cortex Technology, Hadsund, Denmark) was used to evaluate the dorsal skin surface of the hairless mice according to the manufacturer's instructions. TEWL was measured under standard conditions on every 2 days of experiment.

2.7 Determination of dorsal skin thickness

Dorsal skin thickness was measured by Vernier caliper (Mitutoyo Corp., Tokyo, Japan) on every 2 days of experiment. Mice were held by one operator to expose the dorsal skin, and another operator randomly picked 3 positions on the skin to perform measurement, double-layer thickness was recorded.

2.8 Serum IgE enzyme-linked immunosorbent assay (ELISA)

Blood serum samples collected from mice at the time of death were serially diluted. Serum levels of total IgE were measured by ELISA. Serum IgE concentrations were measured using an IgE ELISA kit (Biolegend, San Diego, CA, USA) according to the manufacturer's protocol.

2.9 Histological analysis

At the end of experiment, dorsal skin tissues were resected, formalin-fixed and embedded in paraffin. Sections were cut and stained with hematoxylin and eosin to observe histopathological changes such as dermal and epidermal hyperplasia, spongiosis, hyperkeratosis and immune cell infiltration. Stained tissue sections were observed using a light microscope at 200× magnification (Olympus, Tokyo, Japan).

3 Results and discussion

3.1 Overview of the chronic dermatitis treatment based on wearable OLED patch

As shown in Fig. 1(a), the wearable OLED patch was applied to the skin of a mouse model of chronic dermatitis to provide treatment. Red light induces the PBM, which is absorbed by cytochrome C oxidase in mitochondria within cells. This increases adenosine triphosphate (ATP) production and releases low concentrations of reactive oxygen species (ROS) and nitric oxide (NO), thereby alleviating inflammation and promoting cell regeneration [24].

Psoriasis is an autoimmune disease in which keratinocytes proliferate abnormally and rapidly due to excessive activation of immune cells, as depicted in Fig. 1(b) [25]. Environmental triggers such as physical trauma, infection, and stress can activate plasmacytoid dendritic cells to produce interferon-α (IFN-α) [26]. IFN-α stimulates myeloid dendritic cells to release interleukin (IL)-12 and IL-23, promoting the differentiation of T-Helper1 (Th1), Th17, and Th22 cells [27]. These T cells secrete pro-inflammatory cytokines, including IFN-γ, IL-17, IL-22, and tumor necrosis factor-α (TNF-α), which exacerbate keratinocyte hyperproliferation [28]. These inflammatory mediators also induce keratinocyte-derived chemokines such as CXCL8 (IL-8), which attract neutrophils and other immune cells to the skin, thereby maintaining and amplifying the chronic inflammatory characteristic of psoriatic lesions [29].

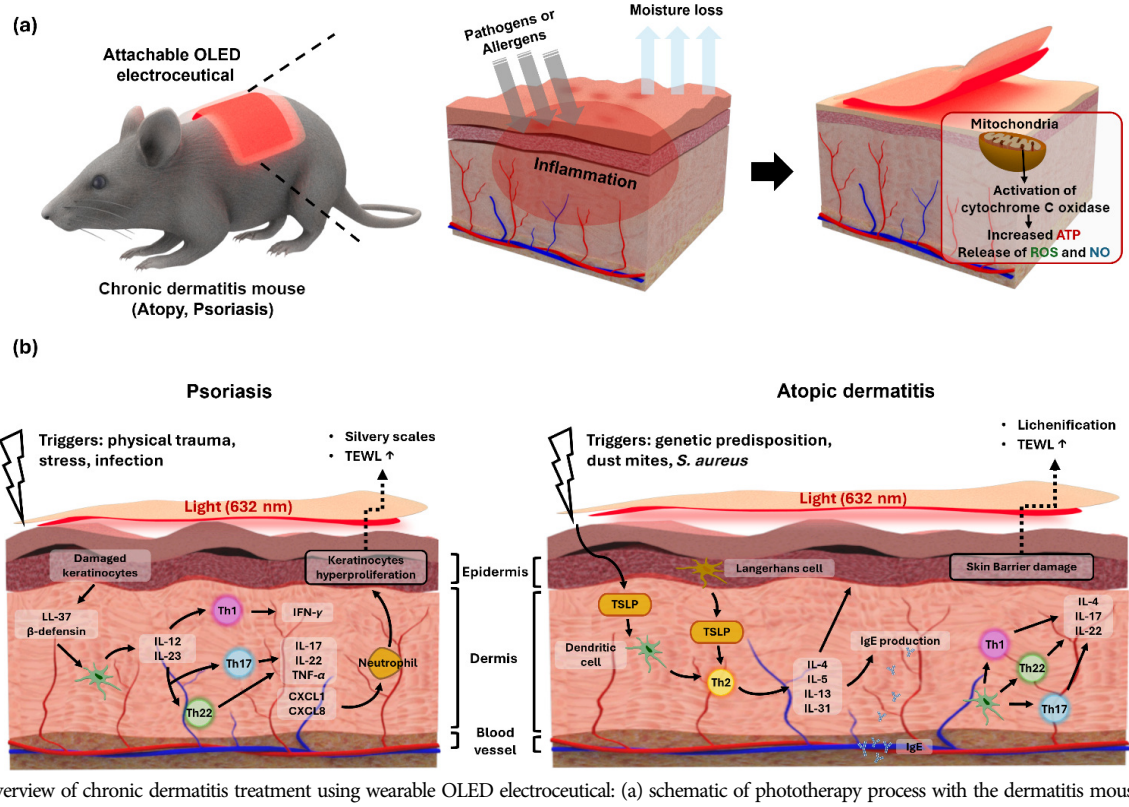

Figure 1 Overview of chronic dermatitis treatment using wearable OLED electroceutical: (a) schematic of phototherapy process with the dermatitis mouse model and (b) pathogenesis of psoriasis and atopic dermatitis.

When psoriatic skin is exposed to red light, the expression of inflammatory cytokines such as IL-17, IL-22, TNF-α, and IFN-γ is significantly reduced, along with CXCL8 levels. This immunomodulatory effect may contribute to the overall restoration of skin structure, normalizing abnormal keratinocyte differentiation, and promotion of epidermal tissue repair [30].

AD is a skin disease whose exact cause remains unclear, but several factors are implicated as potential contributors, including genetic mutations in filaggrin (a protein that forms the skin barrier) and acquired factors such as dust mite and *S. aureus* infections. Filaggrin disrupts the structural integrity and hydration capacity of the stratum corneum, promoting TEWL and increasing the penetration of allergens or pathogens [31]. Damage to the skin barrier caused by filaggrin deficiency induces the activation of Langerhans cells, which then secrete keratinocyte-derived thymic stromal lymphopoietin (TSLP) [32]. This TSLP stimulates dendritic cells to promote Th2 cell differentiation. Th2 cells secrete a series of cytokines, including IL-4, IL-5, and IL-13, which produce immunoglobulin E (IgE) and increase pruritus [33]. In the chronic phase, the response expands to Th1, Th17, and Th22 cells, further exacerbating epidermal hyperplasia, inflammation, and lichenification [34]. Stimulation with 630 nm red light, which has sufficient penetration depth into the dermis, suppresses the secretion of inflammatory cytokines derived from Th1, Th2, Th17, and Th22 cells while promoting the activation of regulatory T cells (Tregs), contributing to the restoration of immune homeostasis [35]. These findings suggest that red-light-based photomedicine can be utilized as a noninvasive and multifaceted treatment strategy to simultaneously modulate immune responses and tissue abnormalities in psoriasis and AD.

3.2 Fabrication of wearable OLED patch

This OLED patch (TENTECH Inc., Seoul, Republic of Korea) includes a dedicated hardware platform for immediate operation during daily life. It was fabricated in the form of a flexible printed circuit board (FPCB) to ensure stable operation in a wearable condition. The overall architecture of this system is illustrated in Fig. 2(a), while Fig. 2(b) presents the actual platform along with its components. The core of the system is a microcontroller unit (MCU) PSOC4124 (Cypress, USA) for command configuration and processing. The power supply consists of a 3.7 V, 110 mAh rechargeable battery integrated with a regulation structure to provide constant voltage operation of the OLED.

Additionally, an LED indicator connected to a charging integrated circuit (IC) intuitively indicates the charging and discharging status to the user, and the battery can be recharged by connecting it to an external power source, making the OLED patch reusable. The developed FPCB operated at a constant voltage of 3.3 V, and the power density of the OLED, a key component for phototherapy, was controlled through the OLED driver. Specifically, to maintain the power density suitable for the therapeutic purposes, the driving protocol was designed to continuously supply a constant power density of approximately 5 mW/cm^2. Furthermore, the circuit structure shown in Fig. 2(c) was applied to control thermal changes that occur during OLED operation. To assess heat generation in actual driving conditions, the OLED temperature was measured according to power density, confirming that the temperature remained constant during operation. A detailed circuit diagram of the entire hardware system is provided in Fig. S1 in the Electronic Supplementary Material (ESM). The FPCB was positioned between the flexible battery and the OLED, supplying power to the OLED while enabling recharging through the flexible battery (Fig. 2(d)). It was flexible enough to be attached to the skin and supported reliable and stable operation in wearable photomedicine.

3.3 Characteristics of wearable OLED patch for chronic dermatitis treatment

The OLED used in this work was placed on the bottom layer of the patch so that it can deliver light directly to the skin. As summarized

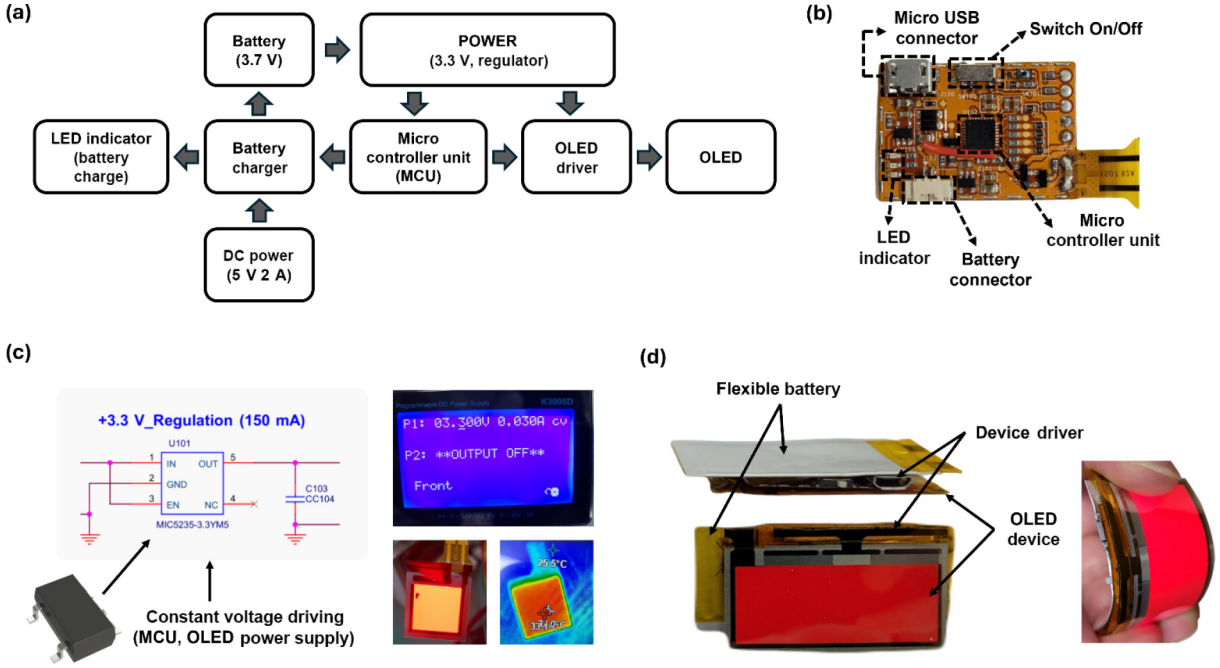

Figure 2 Flexible hardware system of wearable OLED patch. (a) Hardware design architecture for operating OLED. (b) An image of the actual hardware platform and components. (c) A circuit diagram of the hardware system and the OLED driving photograph. (d) A image of the actual wearable OLED patch.

in Table S1 in the ESM, conventional LASERs and LEDs, as point light sources, often result in localized heating and uneven irradiation. In contrast, OLEDs are inherently surface-emitting devices, allowing for thermally stable operation at low power densities, making them ideal for continuous low-intensity light exposure. The device was turned on at 2.1 V and achieved an irradiance of 30 mW/cm^2 and a current efficiency of 613 mW/A at a maximum voltage of 4 V (Fig. 3(a) and Fig. S2 in the ESM). The emission spectrum exhibited a peak at 632 nm with a full width at half maximum (FWHM) of 50 nm, indicating that the OLED could operate stable at a target irradiance of 5 mW/cm^2, which is within the effective range of PBM therapy (Fig. 3(b)).

In addition, as shown in Fig. 3(c), the OLED has a large emission area of 100 mm^2, which should allow for consistent skin contact coverage with the same output power across the entire emitting surface. Excellent spatial uniformity was observed, with a light intensity deviation of less than ± 6% across nine measurement points. This uniform large-area emission ensures homogeneous dose delivery to irregularly shaped lesions, improving consistency of treatment outcomes. Most importantly, as a result of a 1000 cycles of repeated bending with radius of 30 and 25 mm, the initial irradiance (23.3 mW/cm^2) showed negligible changes to 23.1 and 22.8 mW/cm^2, respectively (Fig. 3(d)). When bent to a 20 mm radius, the irradiance decreased to 15.9 mW/cm^2 due to increased mechanical stress. Nevertheless, at the therapeutic operating level of 5 mW/cm^2, the irradiance remained comparable to the pre-bending condition. Considering that AD and psoriasis are treated not only at joint regions but also across broader body surfaces, a bending radius of 20 mm is a reasonable requirement for both experimental validation and practical application. Further optimization of the OLED patch will enable stable operation even in more highly contoured regions.

As shown in Figs. 3(e) and 3(f), the operating stability was evaluated at different output levels. At the therapeutic power of 5 mW/cm^2, the temperature remained at approximately 20 °C after 1 h of operation. Even at the higher output of 15 mW/cm^2, the temperature rose to 31 °C after 1 h, remaining within a safe range that does not cause skin irritation or low-temperature burns. Furthermore, the operating lifespan maintained 95% and 85% of the initial luminance, respectively, over a continuous operation period of 1530 h under 5 and 15 mW/cm^2 conditions. To simulate actual usage conditions, moisture resistance was assessed by immersing the device in deionized (DI) water for 30 min, following the same protocol used during the *in vivo* experiments. The irradiance measured before and after immersion was 25.1 and 24.6 mW/cm^2, respectively, showing little difference (Fig. S3 in the ESM).

These results indicate that the device can maintain stable performance over extended periods, even when exposed to external factors such as sweat, moisture, and exudates. In addition, the low thermal load allows safe operation in close contact with the skin. Collectively, these strengths of the OLED patch facilitate low-intensity and repetitive light, which is particularly important in PBM therapies where cumulative biological responses are important [36, 37].

3.4 *In vivo* experiment for atopic dermatitis treatment

The potential of AD treatment was assessed through *in vivo* experiments based on the performance of OLED patch. As shown in Video S1 in the ESM, a complete wearable platform integrating the FPCB, flexible battery, and OLED was attached to the mouse model. AD was induced by applying OXA to the ears and backs of BALB/c mice (Fig. 4(a)). The treatment was performed with a 632 nm wavelength OLED at a power density of 5 mW/cm^2 for 15 min (4.5 J/cm^2) and 30 min (9 J/cm^2) daily for a total of 10 days (Fig. 4(b)). These irradiation conditions were selected to remain within the established therapeutic window of PBM, which follows a biphasic dose response. Excessive irradiance may attenuate

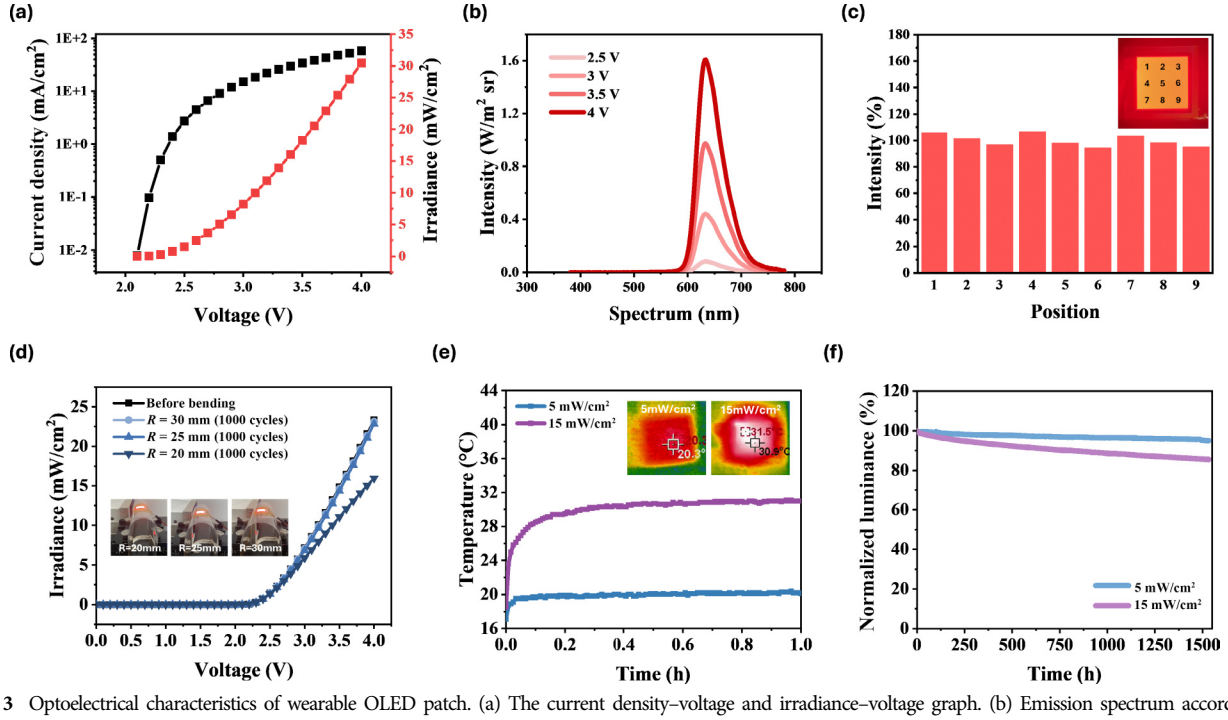

Figure 3 Optoelectrical characteristics of wearable OLED patch. (a) The current density–voltage and irradiance–voltage graph. (b) Emission spectrum according to voltage. (c) Spatial uniformity of OLED light source. (d) Irradiance variation according to repeated bending. (e) Heat generation according to output power. (f) Operating lifetime according to output power.

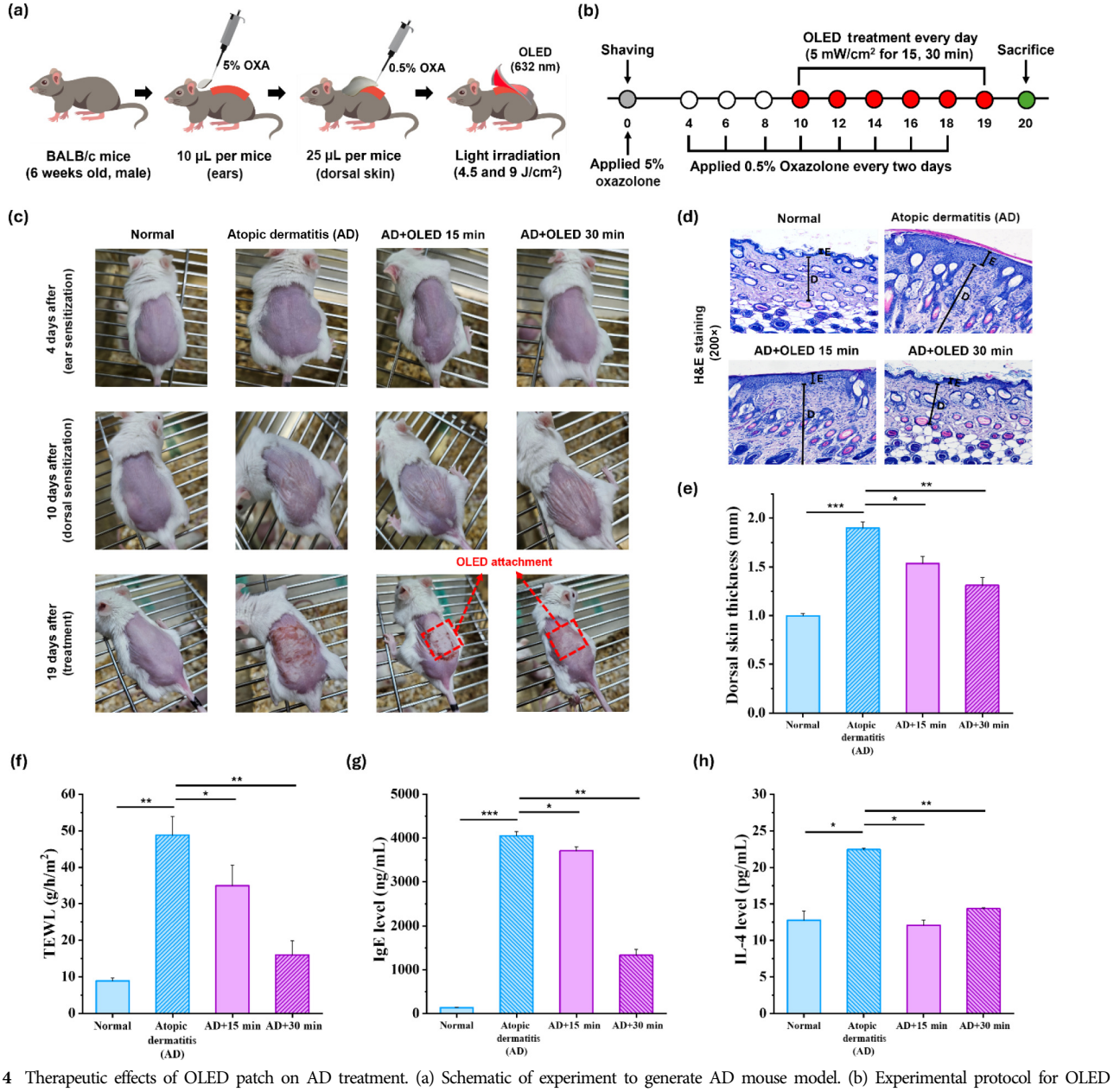

Figure 4 Therapeutic effects of OLED patch on AD treatment. (a) Schematic of experiment to generate AD mouse model. (b) Experimental protocol for OLED stimulation in AD mouse. (c) Images of skin lesions in the indicated model. (d) H&E stained sections in the indicated model (200× magnification). (e) Dorsal skin thickness on day 20; $n = 5$. (f) TEWL values on day 20; $n = 3$ per group. (g) IgE level; $n = 3$ per group. (h) IL-4 expression level; $n = 2$ per group. Data are expressed as mean ± standard deviation; statistical significance was determined using one-way ANOVA; $^*P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$.

biological effects, whereas controlled low-intensity exposure enables sustained mitochondrial activation and immunomodulatory signaling. Accordingly, a repetitive low-dose protocol was employed to achieve gradual regulation of inflammatory responses in the chronic dermatitis model.

Changes in body weight, skin thickness, and TEWL were monitored in normal mice, AD mice, and OLED-treated AD mice to assess disease progression and treatment response. Although the OLED-treated group showed a slight reduction in body weight, this change was attributed to transient stress associated with the device attachment (Fig. S4 in the ESM). As shown in Fig. 4(c), mice were observed on days 4 (ear sensitization), 10 (back sensitization), and 19 (last PBM treatment) of the experiment. The AD-induced mice exhibited progressive worsening of symptoms, including erythema, dryness, and skin roughness. However, both the 15 and 30 min-treated groups showed visible improvement in lesion appearance, with the 30 min mice displaying skin conditions comparable to those of normal mice. In addition, dorsal skin thickness was measured every two days. AD mice showed twofold thicker skin than normal controls, whereas significant attenuation of skin thickening was observed in the 30 min light-treated mice ($P < 0.01$, Fig. 4(e)). Consistently, hematoxylin and eosin (H&E) staining revealed epidermal hyperplasia in the AD model, while the 30 min treated model showed a normalization of epidermal thickness, with a significant decrease in hyperkeratosis and immune cell infiltration (Fig. 4(d) and Fig. S5 in the ESM). Chronic inflammation in AD not only causes epidermal thickening but also compromises the skin's ability to retain moisture. Accordingly, TEWL increased from

8.9 g/h/m² in normal mice to 48.7 g/h/m² in AD-induced mice. In the 30 min OLED treatment, this value significantly decreased to 16 g/h/m², indicating substantial restoration of skin barrier function ($P < 0.01$, Fig. 4(f)).

To evaluate the modulatory effect on the immune response, serum IgE levels were measured (Fig. 4(g)). AD mice exhibited marked IgE overexpression, reflecting enhanced Th2-mediated immune activation. OLED irradiation led to a dose-dependent reduction, with decrease of 10% after 15 min and 70% after 30 min treatment ($P < 0.01$). Given that allergen–IgE interactions are known to trigger inflammatory responses mediated through various cytokines, particularly IL-4, which plays a pivotal role in the AD exacerbation. Following OLED treatment, IL-4 levels were reduced to values comparable to those of normal controls, indicating effective attenuation of Th2-dominant inflammation (Fig. 4(h)). In contrast, IL-17 and IL-22 cytokines, more closely associated with the chronic immune phase of AD, did not show significant changes throughout the experimental period (Fig. S6 in the ESM). This suggests that the relatively short treatment period primarily affected acute Th2-mediated responses, rather than fully established chronic immune reconstitution. Nevertheless, the improvement in epidermal morphology and barrier function, along with the suppression of IgE and IL-4, indicating that repeated low-intensity OLED irradiation modulates key immune pathways involved in the pathogenesis of AD.

3.5 *In vivo* experiments for psoriasis treatment

Following the positive results obtained in the AD model, the effects of OLED irradiation were further evaluated in psoriasis model. Psoriasis was induced in BALB/c mice by administering IMQ to the dorsal skin (Fig. 5(a)). Mice received daily irradiation at 632 nm and 5 mW/cm² for 15 or 30 min over 8 consecutive days (Fig. 5(b)). As in the AD model, minor body weight reduction was observed and attributed to mechanical stress from patch attachment (Fig. S7 in the ESM).

Macroscopic examination revealed typical erythema and silvery scales in psoriasis induced mice, whereas the 30 min treated group (total energy dose of 9 J/cm²) exhibited marked visual improvement, approaching the appearance of normal skin (Fig. 5(c)). Consistent with these observations, H&E staining demonstrated a reduction of epidermal hyperplasia in the 30 min group (Fig. 5(d) and Fig. S8 in the ESM). Quantitative measurements further confirmed significant reductions in dorsal skin thickness ($P < 0.001$, Fig. 5(e)). Likewise, TEWL, which was elevated in psoriatic mice due to barrier disruption, decreased toward near-normal levels following 30 min irradiation, suggesting restoration of epidermal integrity (Fig. 5(f)).

Because IgE and IL-4 are not central to the pathogenesis of psoriasis and thus were not suitable markers for evaluating therapeutic outcomes. Instead, IL-17 and IL-22, representative cytokines of Th17 and Th22 immune responses, were analyzed. As

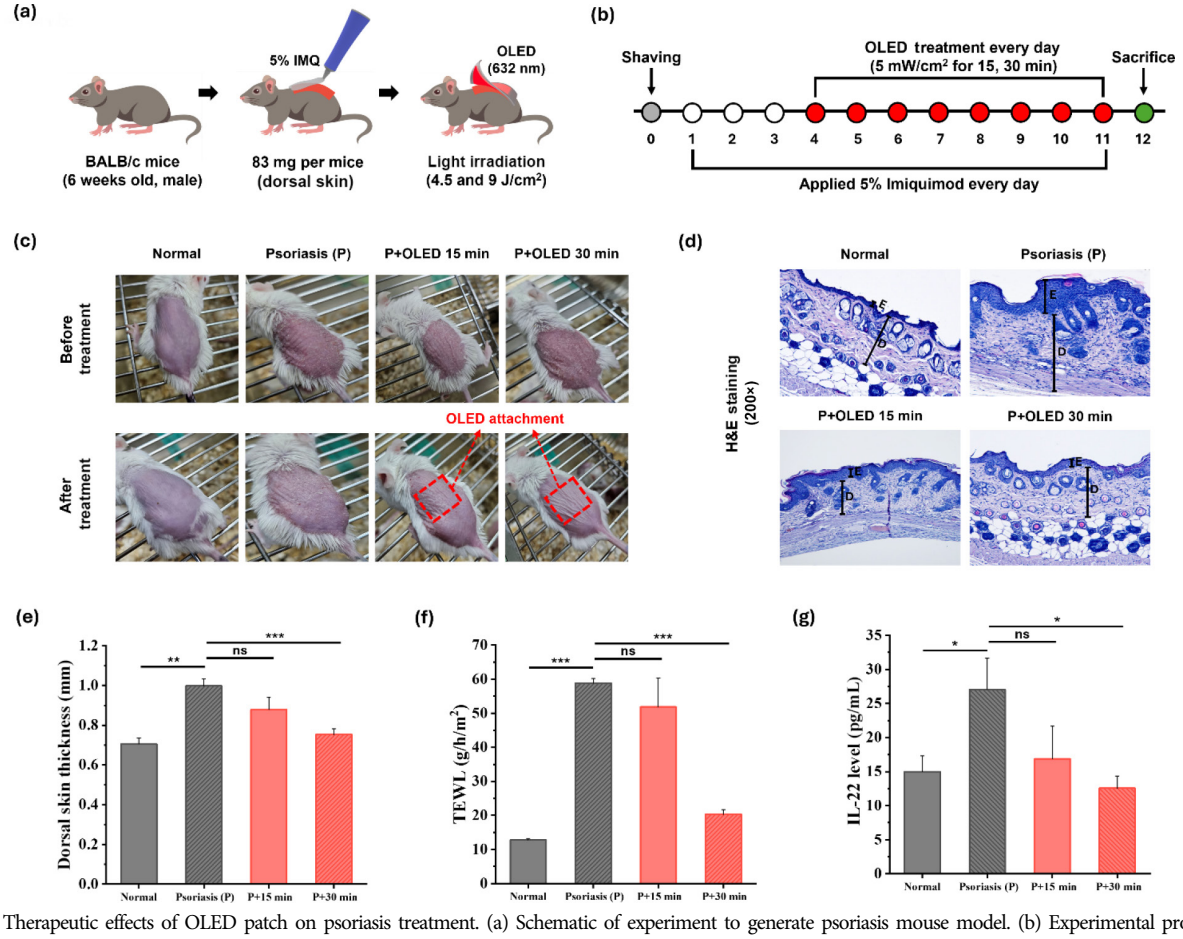

Figure 5 Therapeutic effects of OLED patch on psoriasis treatment. (a) Schematic of experiment to generate psoriasis mouse model. (b) Experimental protocol for OLED stimulation in psoriasis mouse. (c) Images of skin lesions in the indicated model. (d) H&E stained sections in the indicated model (200× magnification). (e) Dorsal skin thickness; $n = 4$ per group. (f) TEWL values; $n = 4$ per group. (g) IL-22 expression level; $n = 3$ per group. Data are expressed as mean ± standard deviation; statistical significance was determined using one-way ANOVA; $^*P < 0.05$; $^{**}P < 0.01$; $^{***}P < 0.001$; ns, not significant.

shown in Fig. 5(g), IL-22 levels were increased in psoriasis mice compared to the normal mice but were significantly reduced in the 30 min treated mice ($P < 0.05$). Similarly, IL-17 expressions decreased to levels comparable to that of the normal mice (Fig. S9 in the ESM). These results demonstrate that OLED treatment effectively suppressed Th17 and Th22-related cytokines, which are responsible for neutrophil infiltration and abnormal keratinocyte differentiation, thereby contributing to the suppression of inflammatory signaling pathways.

Taken together, periodic low-intensity irradiation (9 J/cm^2) using wearable OLED patch induced both physiological and immunological improvements in AD and psoriasis. In the AD model, regulation of Th2-related responses (IgE and IL-4) led to normalization of epidermal thickness and barrier function, along with visible reduction of erythema and skin roughness. In the psoriasis model, suppression of Th 17 and Th 22 cytokines resulted in decreased epidermal hyperplasia, TEWL recovery, and marked alleviation of erythema and scaling. These effects were achieved through sustained phototherapy within the biphasic nature of PBM, enabling stable immunomodulation.

4 Conclusions

In this study, we systematically verified the potential of wearable OLED electroceuticals for AD and psoriasis. Daily irradiation with 632 nm red light at 5 mW/cm^2 for 30 min resulted in consistent improvements in visible lesion severity, recovery of skin barrier function, and modulation of disease-relevant immune pathways. These structural and immunological changes suggest effective attenuation of inflammatory activity in a preclinical setting. From a clinical perspective, parameters improved in this work are closely related to existing severity indices used in AD and psoriasis. Although complete remission cannot be determined within the timeframe of this study, the observed improvements reflect meaningful symptom alleviation, an important therapeutic objective in chronic dermatitis management.

Beyond biological efficacy, the OLED patch is characterized by high flexibility, excellent skin adhesion, and low thermal operation enables stable application to actual skin. Compared with hospital-based high-intensity LASER systems or long-term topical pharmacotherapy, the OLED patch offers a reusable, non-invasive, and wearability suitable for sustained management. Therefore, these findings provide both technological validation and biological evidence supporting the future clinical translation of wearable OLED-based treatment as a patient-adaptable strategy for long-term control of chronic inflammatory dermatitis.

Electronic Supplementary Material: Supplementary material (including additional device characteristics and analysis of AD and psoriasis therapeutic effects (Figs. S1–S9 and Video S1): circuit diagram of the entire hardware system (Fig. S1); voltage-current efficiency of OLED (Fig. S2); irradiance of OLED before and after DI water dipping (Fig. S3); weight change in AD model (Fig. S4); H&E stained images of AD model (Fig. S5); IL-17 and IL-22 expression levels in AD model (Fig. S6); weight change in psoriasis model (Fig. S7); H&E stained images of psoriasis model (Fig. S8); IL-17 expression level in psoriasis model (Fig. S9); table of characteristics of LASER, LED, and OLED (Table S1); and video of OLED patch attached in mouse model (Video S1)) is available in the online version of this article at https://doi.org/10.26599/NR.2026.94908607.

Data availability

All data needed to support the conclusions in the paper are presented in the manuscript and the Electronic Supplementary Material. Additional data related to this paper may be requested from the corresponding author upon request.

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Declaration of competing interest

All the contributing authors report no conflict of interest in this work.

Author contribution statement

Y. J. S.: Conceptualization, data curation, formal analysis, writing – original draft, writing – review & editing, visualization. K. A. K.: Conceptualization, data curation, formal analysis, investigation, writing – review & editing. H. J. K.: Data curation, investigation. J. O. B.: Data curation, investigation. Y. S. C.: Data curation, investigation. D. B: Conceptualization, formal analysis, investigation. K. K.: Formal analysis, investigation. S. J. K.: Resources, project administration. E.-S. C.: Resources, funding acquisition, project administration, supervision. S. T. K.: Conceptualization, project administration, validation, supervision, writing – review & editing. Y. J.: Conceptualization, project administration, validation, supervision, writing – review & editing. All the authors have approved the final manuscript.

Informed consent

Not applicable.

Ethics statement

The animal experiments were carried out following the guidelines of the Gachon University Animal Research Guidelines and were approved by the Gachon University (Ethical code: LCDI-2022-0101).

Use of AI statement

None.

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