

## ORIGINAL ARTICLE

# NB-UVB irradiation attenuates inflammatory response in psoriasis

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## Abstract

Psoriasis is a chronic inflammatory disease characterized by immunological imbalance and vasodilation. Many triggering factors for psoriasis initiate inflammation via the activation of NF- $\kappa$ B. Narrow-band ultraviolet B (NB-UVB) irradiation can be used as a general treatment for psoriasis, although the molecular mechanism has not yet been determined. The aim of this study was to elucidate the potential molecular mechanism of NB-UVB irradiation therapy on psoriasis. We collected serum samples from patients with psoriasis and healthy control, and detected the expression of inflammatory factors by ELISA. In addition, we established mouse model of psoriasis. After different doses of NB-UVB irradiation, the proportion of CD4<sup>+</sup>, CD8<sup>+</sup>, and CD11c<sup>+</sup> cells in mouse spleen was detected by flow cytometry. Meanwhile, the expression of inflammatory factors in the damaged skin of mice was detected by RT-PCR and Western blot analysis, and mouse serum levels of inflammatory factors were detected by ELISA. Our results showed that NB-UVB irradiation regulated the expression of inflammatory factors in psoriasis patients. In mice, high-dose NB-UVB irradiation effectively eliminated IMQ-induced psoriasis-like dermatitis and inhibited the expression of pro-inflammatory factors. In conclusion, our results indicate that NB-UVB irradiation could regulate the expression of inflammatory factors and attenuate psoriasis plaques.

## KEYWORDS

inflammatory factors, NB-UVB irradiation, psoriasis

## 1 | INTRODUCTION

Psoriasis is a common chronic inflammatory skin disease characterized by T-cell-mediated keratinocyte proliferation, affecting 2% to 3% of global population.<sup>1,2</sup> At present, the etiology of psoriasis is still unclear, but genetic background, environmental factors, and immune system are related to the risk of psoriasis.<sup>3</sup> The activation of Nuclear

Factor Kappa B (NF- $\kappa$ B) leads to the recruitment of inflammatory cells and the production of pro-inflammatory mediators, such as interleukin 1 (IL-1), IL-6, IL-8, and tumor necrosis factor  $\alpha$  (TNF- $\alpha$ ) in psoriasis.<sup>4</sup> The CD4<sup>+</sup> T helper cell also called Th17 cell is important in the pathogenesis of many diseases,<sup>5</sup> including psoriasis.<sup>6</sup> New anti-T lymphocyte immunotherapy and some traditional anti-pain drugs, such as methotrexate, steroids, and cyclosporine, confirmed the important

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role of the immune system in psoriasis.<sup>7</sup> IL-17 is detected in skin lesions of psoriasis patients.<sup>8</sup> Moreover, interferon- $\gamma$  (IFN- $\gamma$ ) enhanced the pro-inflammatory effect of IL-17, resulting in increased secretion of IL-6 and IL-8.<sup>9</sup> IFN- $\gamma$  also activated granulocytes and monocytes to regulate inflammation by releasing various cytokines, including IL-1B, TNF- $\alpha$ , granulocyte colony-stimulating factor (G-CSF), and granulocyte-macrophage colony stimulating factor (GM-CSF).<sup>10</sup> Piskin et al. showed that the expression of IL-12, IL-18, and IL-23 in the skin of psoriasis patients decreased after narrow-band ultraviolet B (NB-UVB) treatment.<sup>11</sup> Therefore, early treatment interventions in psoriasis patients may be beneficial to prevent systemic inflammatory response.<sup>12</sup>

NB-UVB irradiation can be used as a general treatment for psoriasis.<sup>13</sup> However, molecular mechanisms underlying the efficacy of NB-UVB are incompletely understood.<sup>14</sup> Keratinocyte (KC) absorbs nearly 90% of NB-UVB irradiation and is considered to be the main target for regulating plaque mitigation in chronic psoriasis.<sup>15</sup> In addition, NB-UVB irradiation alleviates inflammation by reducing the expression of pro-inflammatory cytokines, such as IL-1 $\alpha$ , IL-5, and IL-6,<sup>16</sup> and enhances the synthesis of anti-inflammatory cytokines.<sup>17</sup> The direct effect of NB-UVB irradiation is mainly confined to the upper part of epidermis and papillary dermis, and it reduces the number of epidermal Langerhans cells (LC) and dermal dendritic cells (DCs), inhibits TH1 and TH17 signaling pathways, and normalizes the expression of genes associated with epidermal proliferation and differentiation.<sup>18,19</sup> NB-UVB irradiation also downregulates pro-inflammatory cytokines and TH1 and TH17 signaling pathways in the skin.<sup>20</sup> However, the effect of NB-UVB irradiation on systemic inflammation in psoriasis patients and the possible mechanism have not been fully investigated.

In this study, we aimed to elucidate potential molecular mechanism of NB-UVB irradiation therapy on psoriasis.

## 2 | MATERIALS AND METHODS

### 2.1 | Patients

This study was approved by Ethics Committee of The First Affiliated Hospital of Yunnan University of Traditional Chinese Medicine. All participants signed written informed consent. Thirty-one psoriasis patients (20 male and 11 females; mean age 50-80 years old) and 29 healthy controls (17 male and 12 females; mean age 50-80 years old) were recruited from The First Affiliated Hospital of Yunnan University of Traditional Chinese Medicine. Patients were treated with NB-UVB using UVB apparatus (Sigma, Germany) with fluorescent lamps (311 ~ 313 nm) three times a week. Standard dose ranges were 0.55 to 3.13 J/cm<sup>2</sup>.

### 2.2 | Animals

SKH-1 hairless mice, 8 to 10 weeks old, were purchased from Hangzhou Normal University. Next, psoriasis-like dermatitis in mice was induced by Aldara imiquimod cream (62.5 mg/day). Briefly, mice were randomly assigned into five groups: the imiquimod (IMQ) group (administered IMQ alone), the high-dose group (IMQ + NB-UVB,

cumulative dose of 2000 mJ/cm<sup>2</sup>), the medium dose group (IMQ + NB-UVB, cumulative dose of 1000 mJ/cm<sup>2</sup>), the low-dose group (IMQ + NB-UVB, cumulative dose of 500 mJ/cm<sup>2</sup>), and the control group. The mice in the high dose group were irradiated every other day from the first day of the experiment. From the initial dose of 300 mJ/cm<sup>2</sup>, 50 mJ/cm<sup>2</sup> was added to each continuous dose to reach the cumulative dose of 2000 mJ/cm<sup>2</sup>. The mice in the medium dose group were irradiated with 80 mJ/cm<sup>2</sup> every other day to reach the cumulative dose of 1000 mJ/cm<sup>2</sup>. The mice in the low dose group were irradiated with 100 mJ/cm<sup>2</sup> every other day until the cumulative dose of 500 mJ/cm<sup>2</sup> was reached. Animal experiments were approved by the Institutional Animal Use Committee.

### 2.3 | ELISA assay

The contents of TGF- $\beta$ 1, IL-17, IL-10, IL-6, and IFN- $\gamma$  in serum of patients and healthy people were detected using ELISA Kit (Elabscience, E-EL-M0046c) according to the manufacturer's instructions. Similarly, the contents of GM-CSF, IFN- $\gamma$ , IL-9, IL-10, and IL-17 in serum of mice were detected using the ELISA Kit (Elabscience, E-EL-M0046c) according to the manufacturer's instructions.

### 2.4 | Hematoxylin and eosin staining

Cutaneous lesion margins of mice were harvested and fixed in 4% polyformaldehyde. After paraffin embedding, dewaxing, and hydration, the sections were stained with hematoxylin and eosin (HE) and observed and photographed under optical microscope.

### 2.5 | Flow cytometry

The mice were sacrificed by cervical dislocation and immersed in 75% alcohol for 2 minutes. The mice spleen was dissected and grinded with 200 mesh steel mesh using sterile syringe core, centrifuged at 4°C at 1000 rpm for 10 minutes, and then red blood cells were dissolved with erythrocyte lysate. After centrifugation at 4°C at 1000 rpm for 10 minutes, the cells were collected and suspended in PBS to incubate with CD4<sup>+</sup> (Biolegend, China), CD8<sup>+</sup> (Biolegend, China), and CD11c<sup>+</sup> (Biolegend, China) antibodies at room temperature for 30 minutes. The fluorescence was detected by flow cytometry (Partec GmbH CyFlow Space).

### 2.6 | Real-time quantitative PCR

Total RNA was extracted using the Trizol reagent (Lifetech, China). The RevertAid First Strand cDNA Synthesis Kit (Fermentas) was used to convert RNA into cDNA. Quantitative real-time polymerase chain reaction analysis (qRT-PCR) was conducted using SYBR Green master mix (KAPA, China) on a fluorescent PCR device (ABI 7300). The primers were synthesized by Invitrogen (Guangzhou, China): G-CSF (forward: 5'-TCAA

CTTTCTGCCAGAG-3' and reverse: 5'-TCTCGTCTGACCATAGT-3') and  $\beta$ -actin (forward: 5'-TATGGAATCCTGTGGCATC-3' and reverse: 5'-GTGTTGGCATAGAGGTCTT-3').  $\beta$ -actin was used as an internal control. The results were expressed using the  $2^{-\Delta\Delta Ct}$ . All the experiments were conducted at least three times independently.

## 2.7 | Western blot analysis

100 mg of skin tissue was lysed in 500 mL RIPA lysis buffer (containing 50 mL protease inhibitor) (Beyotime Biotechnology, China) and homogenized by ultrasound. Following centrifugation at 12000 rpm for 20 minutes at 4°C, the supernatants were collected and equal amounts of proteins were separated by sodium dodecyl sulfate polyacrylamide gel electrophoresis (SDS-PAGE) and transferred to polyvinylidene difluoride (PVDF) membranes (Millipore). The membranes were incubated overnight at 4°C with primary antibodies for G-CSF (1:1000; Abcam), CXCL1 (1:2000; Invitrogen, China), IL-12 p40 (1:1000; Invitrogen, China), IL-12 p70 (1:1000; Invitrogen, China), and  $\beta$ -actin (1:2000; Abmart, China). Subsequently, the membranes were incubated with horseradish peroxidase-conjugated secondary antibodies (Goat Anti-Rabbit IgG, 1:2000; Goat Anti-Mouse IgG, 1:2000; Cell Signaling Technology) for 1 hour at room temperature. ECL Western Imprint Detection System (Millipore) and Quantitative ImageJ were used to analyze the bands.

## 2.8 | Statistical analysis

Each experiment was performed at least three times, and all data are presented as the mean  $\pm$  SD. The data were analyzed by *t* test or one-way analysis of variance using GraphPad Prism v.5.0. *P* values  $<$ .05 were considered statistically significant.

## 3 | RESULTS

### 3.1 | Effects of NB-UVB irradiation on inflammatory factors in patients with psoriasis

First, we collected blood samples from patients with psoriasis (before and after NB-UVB irradiation) and healthy control. ELISA results

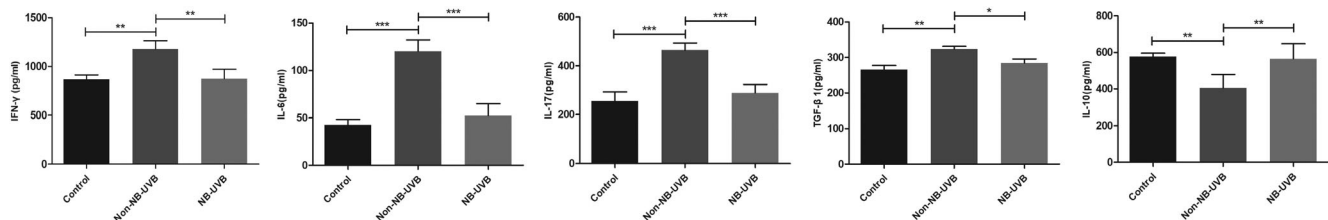
showed that the contents of pro-inflammatory factors in the blood of patients with psoriasis increased significantly (Figure 1,  $P <$  .05,  $P <$  .01,  $P <$  .001). NB-UVB irradiation significantly inhibited the expression of TGF- $\beta$ 1, IL-17, IL-6, and IFN- $\gamma$ . However, the content of anti-inflammatory factor IL-10 in the blood of patients with psoriasis decreased significantly, while NB-UVB irradiation significantly increased the expression of IL-10. Therefore, we hypothesized that the effects of NB-UVB irradiation on psoriasis may be related to the regulation of inflammation.

### 3.2 | NUB irradiation reduced the proportions of CD4<sup>+</sup>, CD8<sup>+</sup>, CD11c<sup>+</sup> cells

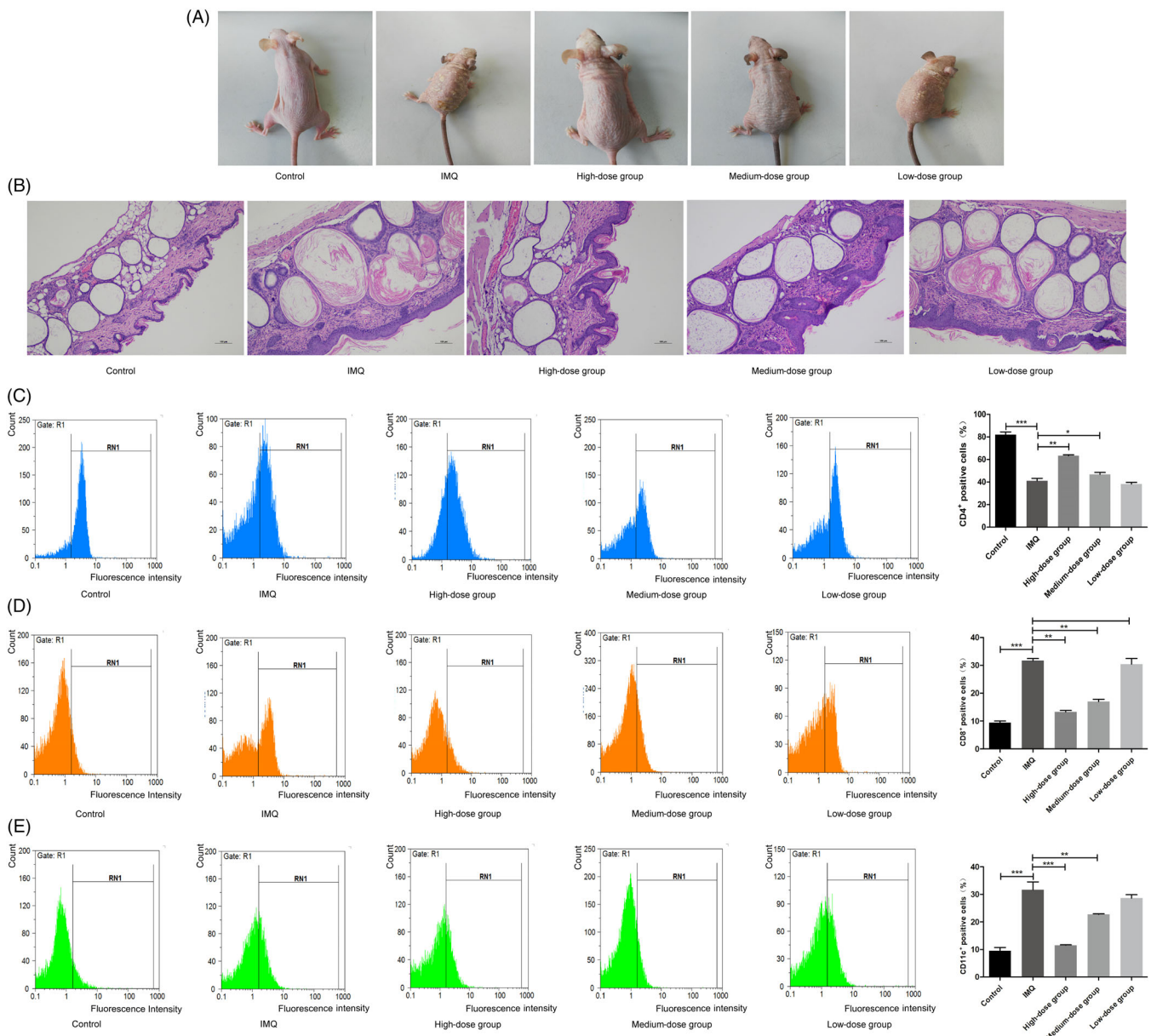
To further confirm our hypothesis, we applied IMQ on the skin of mice to induce psoriasis. After 10 days of continuous administration of IMQ, the changes of mice skin were observed (Figure 2A), which indicated successful construction of animal model of psoriasis. In order to find the best dose of irradiation, different doses of NB-UVB irradiation were administrated to mice. HE staining showed that granular layer became thinner, basal layer became thicker, and epidermal crest was prolonged in the model of psoriasis induced by IMQ (Figure 2B). However, there was no significant difference between control group and the high dose group. Furthermore, mononuclear cells were isolated from mouse spleens and the proportions of CD4<sup>+</sup>, CD8<sup>+</sup>, and CD11c<sup>+</sup> cells were detected by flow cytometry. The ratio of CD8<sup>+</sup> and CD11c<sup>+</sup> cells decreased significantly and the ratio of CD4<sup>+</sup> increased after NB-UVB irradiation (Figure 2C,  $P <$  .05,  $P <$  .01 and  $P <$  .001), which proved that the mechanism of NB-UVB irradiation in the treatment of psoriasis may be related to the regulation of immunological response.

### 3.3 | NB-UVB irradiation-regulated inflammation in psoriasis

Next, we wondered whether inflammation was involved in the inhibition of psoriasis by NB-UVB irradiation. ELISA assay of the serum of mice in model group showed that NU-UVB irradiation significantly reduced IMQ-induced increase of GM-CSF, IFN- $\gamma$ , IL-9, and IL-17 but increased the expression of IL-10 (Figure 3A,  $P <$  .05,  $P <$  .01, and



**FIGURE 1** Effect of NB-UVB irradiation on inflammatory factors in patients with psoriasis. TGF- $\beta$ 1, IL-17, IL-10, IL-6, and IFN- $\gamma$  levels in serum of patients and healthy people ( $n = 10$ ) were detected by ELISA. Values are shown as the mean  $\pm$  SD of at least three independent experiments. \* $P <$  .05, \*\* $P <$  .01, \*\*\* $P <$  .001

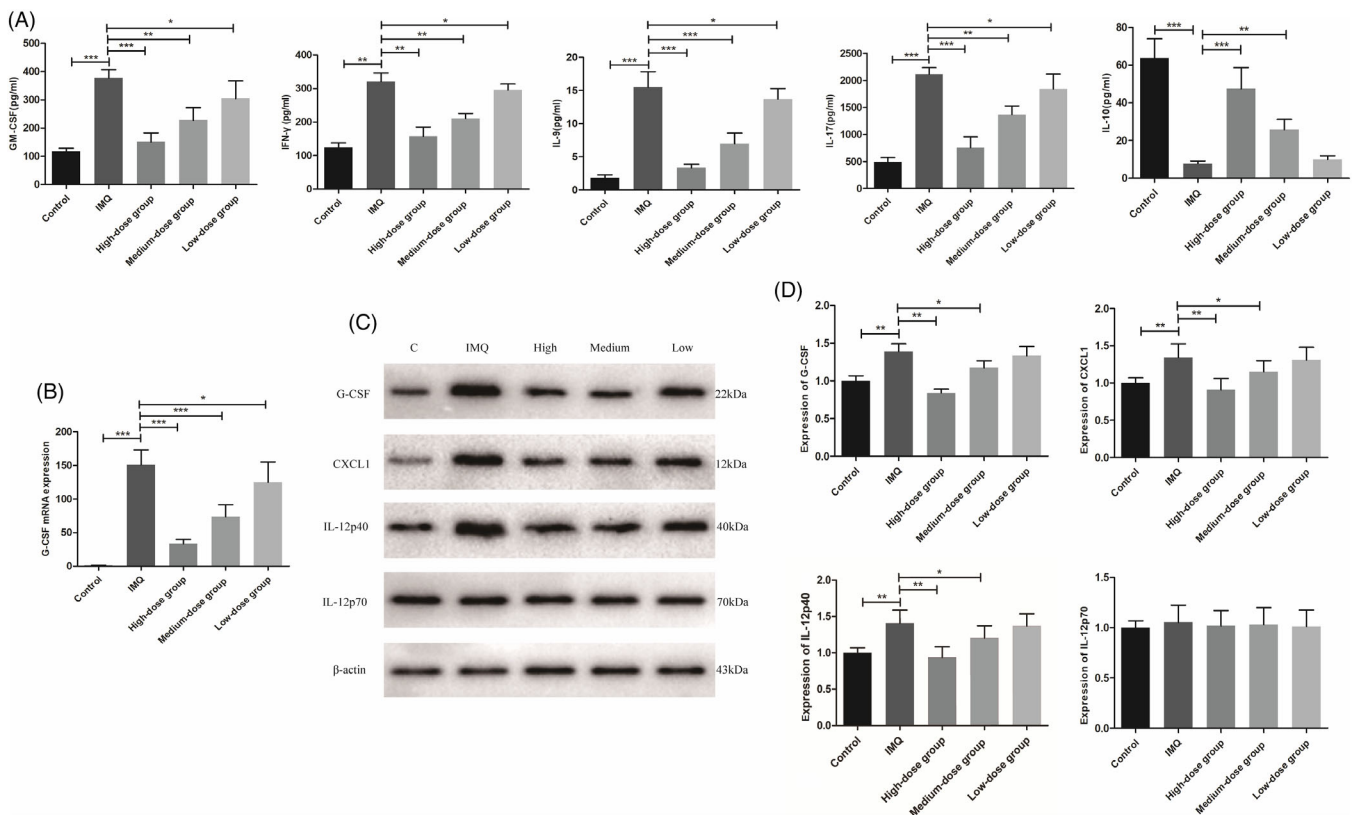


**FIGURE 2** NUB irradiation inhibits inflammation in mouse model of psoriasis. A, Skin changes in psoriatic mice treated with NB-UVB irradiation ( $n = 3$ ). B, H&E staining of skin sections of mice treated with IMQ or/and high, medium, or low doses of NB-UVB irradiation (scale bar: 100  $\mu\text{m}$ ) ( $n = 3$ ). C-E, Flow cytometry analysis of the proportions of CD4<sup>+</sup>, CD8<sup>+</sup>, and CD11c<sup>+</sup> cells ( $n = 3$ ). Values are shown as the mean  $\pm$  SD of at least three independent experiments. \* $P < .05$ , \*\* $P < .01$ , \*\*\* $P < .001$

$P < .001$ ). PCR analysis of the level of G-CSF in mouse skin showed the same trend as ELISA (Figure 3B,  $P < .05$  and  $P < .001$ ). Western blot analysis showed that G-CSF, CXCL1, and IL-12p40 were upregulated in the skin of psoriasis model mice induced by IMQ, but were significantly decreased compared with IMQ model group after NB-UVB irradiation (Figure 3C,D,  $P < .05$  and  $P < .01$ ). In addition, IL-12p70 showed no significant change, which was consistent with that previously reported.<sup>21</sup> These data confirmed that NB-UVB irradiation regulated the expression of inflammatory factors in psoriasis progression.

## 4 | DISCUSSION

In this study, we found that NB-UVB irradiation downregulated the expression of pro-inflammatory factors and upregulated anti-inflammatory factors in serum samples from patients with psoriasis. In addition, similar results were confirmed in psoriasis mouse model induced by IMQ after irradiation with NB-UVB. Based on previous studies,<sup>16,20,22,23</sup> we speculated that different doses of NB-UVB irradiation altered the expression of immune inflammatory factors through NF- $\kappa$ B signaling pathway.



**FIGURE 3** NB-UVB irradiation regulated inflammatory factors in mouse model of psoriasis. A, Serum levels of GM-CSF, IFN- $\gamma$ , IL-9, IL-17, and IL-10 were detected by ELISA ( $n = 3$ ). B, G-CSF expression levels in mouse skin were detected by qRT-PCR. C,D, Western blot analysis of the expression of G-CSF, CXCL1, IL-12p40, and IL-12p70 in mouse skin ( $n = 3$ ). Values are shown as the mean  $\pm$  SD of at least three independent experiments. \* $P < .05$ , \*\* $P < .01$ , \*\*\* $P < .001$

During psoriasis, keratinocyte differentiation and proliferation are regulated by cytokines and inflammatory mediators released by inflammatory cells and are accompanied by pathological changes.<sup>15,23,24</sup> The downstream effects of IL-17 such as the production of IL-1, IL-6, IL-8, TNF- $\alpha$ , and G-CSF may be regulated by NF- $\kappa$ B and promote the pathogenesis of psoriasis.<sup>4</sup> Psoriasis is a chronic inflammatory disease mediated by type 1 memory T cells.<sup>25</sup> Because IL-12p70 promotes the development of type 1 memory T cells,<sup>21,26</sup> we detected the expression of IL-12p70. Interestingly, there was no significant change in the expression of IL-12p70 between control and treatment groups, and it is possible that the severity of psoriasis is not related to the expression of IL-12p70.<sup>21</sup>

The epidermis is the external part of the human body, mainly composed of KC, which absorbs most NB-UVB irradiation.<sup>15</sup> The efficacy of NB-UVB irradiation in the treatment of psoriasis may be related to the induction of lymphocyte apoptosis, the reduction of pro-inflammatory cytokine production, the downregulation of Th17 signaling pathway, and the reduction or depletion of T cells.<sup>19,27,28</sup> Our results showed that the expression of inflammatory factors was regulated by high-dose NB-UVB irradiation in both human and mice. Therefore, high dose of NB-UVB irradiation inhibits the expression of pro-inflammatory factors and increases the expression

of anti-inflammatory factors, thereby reducing or clearing psoriasis plaque. This may be an important mechanism for the treatment of psoriasis.<sup>28</sup> However, whether NB-UVB irradiation induces apoptosis to treat psoriasis remains to be further explored.

In conclusion, therapeutic doses of NB-UVB irradiation could regulate the expression of inflammatory factors, which may be the mechanism by which NB-UVB irradiation ameliorates and cures psoriasis. Further studies are needed to investigate whether NB-UVB irradiation regulates the expression of inflammatory factors via NF- $\kappa$ B signaling to attenuate psoriasis. Taken together, our findings suggest that inflammatory factors are potential therapy targets for psoriasis.

#### CONFLICT OF INTEREST

The authors declare no conflict of interest.

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#### REFERENCES

1. Armstrong AW, Harskamp CT, Armstrong EJ. Psoriasis and the risk of diabetes mellitus: a systematic review and meta-analysis. *JAMA Dermatol.* 2013;149:84-91.

2. Colombo E, Galleri G, Erre GL, et al. Peripheral blood CD8+ T-cell profiles in patients with psoriatic arthritis: a cross-sectional case-control study. *Eur Rev Med Pharmacol Sci*. 2017;21:5166-5171.
3. Kingue S, Ngoe CN, Menanga AP, et al. Prevalence and risk factors of hypertension in urban areas of Cameroon: a Nationwide population-based cross-sectional study. *J Clin Hypertens (Greenwich)*. 2015;17:819-824.
4. Moorchung N, Kulaar JS, Chatterjee M, Vasudevan B, Tripathi T, Dutta V. Role of NF- $\kappa$ B in the pathogenesis of psoriasis elucidated by its staining in skin biopsy specimens. *Int J Dermatol*. 2014;53:570-574.
5. Zheng Y, Danilenko DM, Valdez P, et al. Interleukin-22, a T(H)17 cytokine, mediates IL-23-induced dermal inflammation and acanthosis. *Nature*. 2007;445:648-651.
6. Fitch E, Harper E, Skorcheva I, Kurtz SE, Blauvelt A. Pathophysiology of psoriasis: recent advances on IL-23 and Th17 cytokines. *Curr Rheumatol Rep*. 2007;9:461-467.
7. Baker H. Psoriasis: a review part I. *Dermatologica*. 1975;150:16-25.
8. Blauvelt A, Chiricozzi A. The immunologic role of IL-17 in psoriasis and psoriatic arthritis pathogenesis. *Clin Rev Allergy Immunol*. 2018;55:379-390.
9. Arican O, Aral M, Sasmaz S, Ciragil P. Serum levels of TNF-alpha, IFN-gamma, IL-6, IL-8, IL-12, IL-17, and IL-18 in patients with active psoriasis and correlation with disease severity. *Mediators Inflamm*. 2005;2005:273-279.
10. Shukuya R, Hasegawa T, Niwa Y, Okuma K, Ikeda S. Granulocyte and monocyte adsorption apheresis for generalized pustular psoriasis. *J Dermatol*. 2011;38:1130-1134.
11. Piskin G, Tursen U, Sylva-Steenland RM, Bos JD, Teunissen MB. Clinical improvement in chronic plaque-type psoriasis lesions after narrow-band UVB therapy is accompanied by a decrease in the expression of IFN-gamma inducers—IL-12, IL-18 and IL-23. *Exp Dermatol*. 2004b;13:764-772.
12. Touw CR, Hakkaart-Van Roijen L, Verboom P, Paul C, Rutten FF, Finlay AY. Quality of life and clinical outcome in psoriasis patients using intermittent cyclosporin. *Br J Dermatol*. 2001;144:967-972.
13. Sharma S, Parsad D, Bhattacharjee R, Muthu SK. A prospective right-left comparative study to evaluate the efficacy and tolerability of combination of NB-UVB and topical bimatoprost 0.03% eye drops versus NB-UVB given alone in patients of vitiligo vulgaris. *J Eur Acad Dermatol Venereol*. 2018;32:e330-e331.
14. Uitentuis SE, Narayan VS, Wind BS, Bekkenk MW, de Rie MA, Wolkerstorfer A. Patient reported outcomes for intensified versus conventional NB-UVB treatment in non-segmental vitiligo. *J Dermatolog Treat*. 2019;30:594-597.
15. Zhuang, Y., C. Han, B. Li, L. Jin, E. Dang, H. Fang, H. Qiao & G. Wang. (2018). NB-UVB irradiation downregulates keratin-17 expression in keratinocytes by inhibiting the ERK1/2 and STAT3 signaling pathways. 310, 147-156.
16. Piskin G, Sylva-Steenland RM, Bos JD, Teunissen MB. T cells in psoriatic lesional skin that survive conventional therapy with NB-UVB radiation display reduced IFN-gamma expression. *Arch Dermatol Res*. 2004;295:509-516.
17. Batycka-Baran A, Besgen P, Wolf R, Szepletowski JC, Prinz JC. The effect of phototherapy on systemic inflammatory process in patients with plaque psoriasis. *J Photochem Photobiol B*. 2016;161:396-401.
18. Taguchi K, Fukunaga A, Ogura K, Nishigori C. The role of epidermal Langerhans cells in NB-UVB-induced immunosuppression. *Kobe J Med Sci*. 2013;59:E1-E9.
19. Wang X, Wang G, Gong Y, et al. Disruption of circulating CD4+ T-lymphocyte subpopulations in psoriasis patients is ameliorated by narrow-band UVB therapy. *Cell Biochem Biophys*. 2015;71:499-507.
20. Kim Y, Lee SK, Bae S, et al. The anti-inflammatory effect of alloferon on UVB-induced skin inflammation through the down-regulation of pro-inflammatory cytokines. *Immunol Lett*. 2013;149:110-118.
21. Litjens NH, van der Plas MJ, Ravensbergen B, et al. Psoriasis is not associated with IL-12p70/IL-12p40 production and IL12B promoter polymorphism. *J Invest Dermatol*. 2004;122:923-926.
22. Chung JH, Youn SH, Koh WS, et al. Ultraviolet B irradiation-enhanced interleukin (IL)-6 production and mRNA expression are mediated by IL-1 alpha in cultured human keratinocytes. *J Invest Dermatol*. 1996;106:715-720.
23. Mavropoulos A, Varna A, Zafiriou E, et al. IL-10 producing Bregs are impaired in psoriatic arthritis and psoriasis and inversely correlate with IL-17- and IFN $\gamma$ -producing T cells. *Clin Immunol*. 2017;184:33-41.
24. Guasch Murillo D, Martín Escalona I. Plasma rico en plaquetas versus paracetamol oral para el tratamiento de la osteoartritis temprana de rodilla. *Estudio preliminar boletim do instituto geológico*. 2014;54:272-272.
25. Piskin G, Sylva-Steenland RM, Bos JD, Teunissen MB. In vitro and in situ expression of IL-23 by keratinocytes in healthy skin and psoriasis lesions: enhanced expression in psoriatic skin. *J Immunol*. 2006;176:1908-1915.
26. Khader SA, Pearl JE, Sakamoto K, et al. IL-23 compensates for the absence of IL-12p70 and is essential for the IL-17 response during tuberculosis but is dispensable for protection and antigen-specific IFN-gamma responses if IL-12p70 is available. *J Immunol*. 2005;175:788-795.
27. Avramidis G, Krüger-Krasagakis S, Krasagakis K, Fragiadaki I, Kokolakis G, Tosca A. The role of endothelial cell apoptosis in the effect of etanercept in psoriasis. *Br J Dermatol*. 2010;163:928-934.
28. Sigmundsdottir H, Johnston A, Gudjonsson JE, Valdimarsson H. Narrowband-UVB irradiation decreases the production of pro-inflammatory cytokines by stimulated T cells. *Arch Dermatol Res*. 2005;297:39-42.

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