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Research Article

Protective effect of boric acid against LPS-induced oxidative damage and inflammation in HaCaT keratinocytes: A simultaneous treatment approach

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Abstract

Objectives: Psoriasis is a chronic inflammatory disease characterized by epidermal dysregulation and increased oxidative stress. This study investigated the protective effects of boric acid (BA) in HaCaT keratinocytes exposed to lipopolysaccharide (LPS)-induced acute inflammatory redox stress using a simultaneous treatment protocol.

Methods: HaCaT cells were assigned to control, LPS (10 µg/mL or 200 ng/mL), BA (20 or 100 µM), and simultaneous treatment (LPS + BA) groups. Cell viability was assessed by CCK-8 assay. Oxidative status was evaluated by malondialdehyde (MDA) and advanced oxidation protein products (AOPP), total sulfhydryl (TSH), and antioxidant parameters [superoxide dismutase (SOD) and catalase (CAT)]. Cell migration was analyzed by a wound healing assay.

Results: LPS exposure did not cause overt cytotoxicity at 24 h but was associated with increased MDA and reduced CAT activity, indicating inflammatory oxidative stress. AOPP levels did not show a marked change under these acute conditions. Simultaneous BA administration maintained keratinocyte viability and attenuated LPS-associated lipid peroxidation, while partially restoring antioxidant defenses and improving wound closure.

Conclusion: BA modulates oxidative stress markers and supports antioxidant defense and migratory capacity in LPS-stimulated keratinocytes. These findings support BA as a candidate redox-modulating compound that warrants validation in immune-competent and in vivo models relevant to psoriasis.

Keywords: Antioxidant defense, boric acid, HaCaT keratinocytes, inflammation, LPS (Lipopolysaccharide), oxidative stress, psoriasis

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Psoriasis is a chronic, systemic inflammatory dermatosis affecting 2–3% of the global population, characterized by a complex interplay of genetic susceptibility, immune dysregulation, and keratinocyte hyperproliferation [1, 2]. Pathogenesis is primarily driven by the aberrant activation of the T-cell/cytokine axis, which triggers a sustained inflammatory cascade within the epidermal microenvironment [3].

Boron is a trace non-metal element for organisms. Boron exists as inorganic salts and organic esters, with boric acid (BA) being its primary form in humans [4]. While inorganic BA is systemically absorbed and excreted unchanged [5], it may exert cytotoxic effects on the microbiota [6]. Conversely, organic boron esters are largely indigestible, with over 95% reaching the colon intact. This highlights their potential as prebiotics that support host-microbiota symbiosis and justify the development of boron-based nutraceuticals targeting the microbiome [4, 6]. BA, a naturally occurring bioactive compound, exhibits significant antioxidant and anti-inflammatory proper-

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ties. Recent evidence suggests that boron can modulate redox homeostasis, scavenge reactive oxygen species (ROS), and enhance wound healing, making it a potential candidate for treating inflammatory skin disorders [4, 6]. However, its specific role in protecting keratinocytes during the acute phases of inflammatory activation mimicking psoriasis flares remains insufficiently characterized.

Beyond classic immunological pathways, oxidative stress has emerged as a fundamental driver of psoriasis progression. An imbalance between excessive ROS production largely from activated neutrophils and keratinocytes and the depletion of endogenous antioxidant defenses (e.g., SOD, CAT, and sulfhydryl compounds) leads to lipid peroxidation and cellular dysfunction [7, 8]. While oxidative stress is central to the disease, clinical biomarker studies often yield inconsistent results due to variability in disease stages and severity, highlighting the need for controlled experimental models to evaluate redox-targeted interventions [9, 10].

Using lipopolysaccharide (LPS)-stimulated HaCaT keratinocytes as a well-established model of inflammatory and oxidative injury, this study investigates the protective efficacy of BA. By employing a simultaneous treatment protocol, we aimed to evaluate the impact of BA on cell viability, antioxidant enzyme kinetics, and migratory capacity. Our findings seek to elucidate whether BA can serve as a prophylactic or early-intervention agent to mitigate oxidative damage and inflammatory signaling in psoriasis-like conditions. Importantly, psoriasis pathogenesis is primarily driven by the IL-23/Th17 axis; therefore, the LPS–HaCaT system is used here to model acute inflammatory and oxidative (redox) stress in keratinocytes rather than to reproduce the full psoriatic immune microenvironment.

Materials and Methods

Keratinocyte culture

The human keratinocyte cell line HaCaT was obtained from the Cell Line Service, Heidelberg, Germany. The cells were maintained at 37°C in a 5% CO_2 atmosphere with 95% humidification in complete RPMI 1640 Medium (RPMI 1640; Gibco, USA) containing 10% heat-inactivated fetal bovine serum (FBS; Sigma-Aldrich, USA), 2 mM L-glutamine, 100 U/mL penicillin, and 100 µg/mL streptomycin.

Cell viability assays

HaCaT cells were grown in 96-well plates at a density of 15×10^3 cells/well in complete RPMI 1640 medium. Cells were treated with 10 µg/ml and 200 ng/ml LPS and BA at concentrations of 20, 100 µM for 24 h. Cell viability was determined by using a colorimetric CCK8 (Abbkine, China) assay. Briefly, CCK8 solution was added to the cell culture at 10 µL. After 3 h of incubation, the obtained optical density was measured at 450 nm by using a microplate reader (Bio-Tek, Winooski, VT, USA). All experiments were performed in at least three independent biological replicates (n=3) to ensure reproducibility.

Wound healing assay

Cells were seeded at a high density into 6-well plates. After allowing the cells to adhere and reach 80% confluence, a scratch was created in the center of each well using a sterile 100 µL pipette tip to simulate a wound, followed by washing with 1x PBS [11]. At this stage, the wound was examined under a microscope, and the initial image was recorded. Subsequently, LPS and BA were administered at predetermined doses. The control group received an equivalent volume of medium. Wound closure kinetics were monitored at 24 h post-scratching using an inverted microscope. The denuded areas were digitized, and the migration rate was determined by calculating the change in wound width over time. The extent of wound closure was quantified by measuring the mean distance between the wound edges at multiple representative points, and the results were normalized to the initial baseline (0 h) measurements.

Measurement of oxidative stress biomarkers

All biochemical analyses were performed using cell lysate samples. The second indicator, malondialdehyde (MDA) levels were determined by a modified thiobarbituric acid (TBA) method [12]. Both assays were adapted to the cell lysate matrix and reaction volumes were optimized to allow microplate format. To evaluate the damage caused by oxidative stress on protein structures, advanced oxidative protein products (AOPP) levels were determined spectrophotometrically. In this assay, the method described by Hanasand et al. [13] was adapted to the cell lysate matrix and the analysis conditions were optimized by making the necessary modifications to enable microplate format. Total thiol (–SH) content was determined according to the colorimetric method described by Sedlak and Lindsay [14]. Catalase (CAT) activity was measured spectrophotometrically using the method of Aebi [15], which is based on the rate of hydrogen peroxide (H_2O_2) decomposition. The decrease in absorbance of H_2O_2 over time was monitored, and enzyme activity was calculated accordingly. Superoxide dismutase (SOD) levels were determined using a commercially available enzyme-linked immunosorbent assay (ELISA) kit (AbbKine, Inc., Cat No: KTB1030, Wuhan, China) according to the manufacturer's instructions in cell lysate.

Statistical analysis

The results were presented as means±standard deviation (SD). Statistical analyses were conducted using GraphPad Prism 8 software (San Diego, USA). ANOVA was performed to compare the quantitative data across the groups. When appropriate, post hoc comparisons were performed using Tukey's multiple comparison test. Statistical significance for all analyses was set at a significance level of $p<0.05$.

Results

Cell viability

The effects of LPS, BA, and their combinations on HaCaT keratinocyte viability and migratory capacity were evaluated after

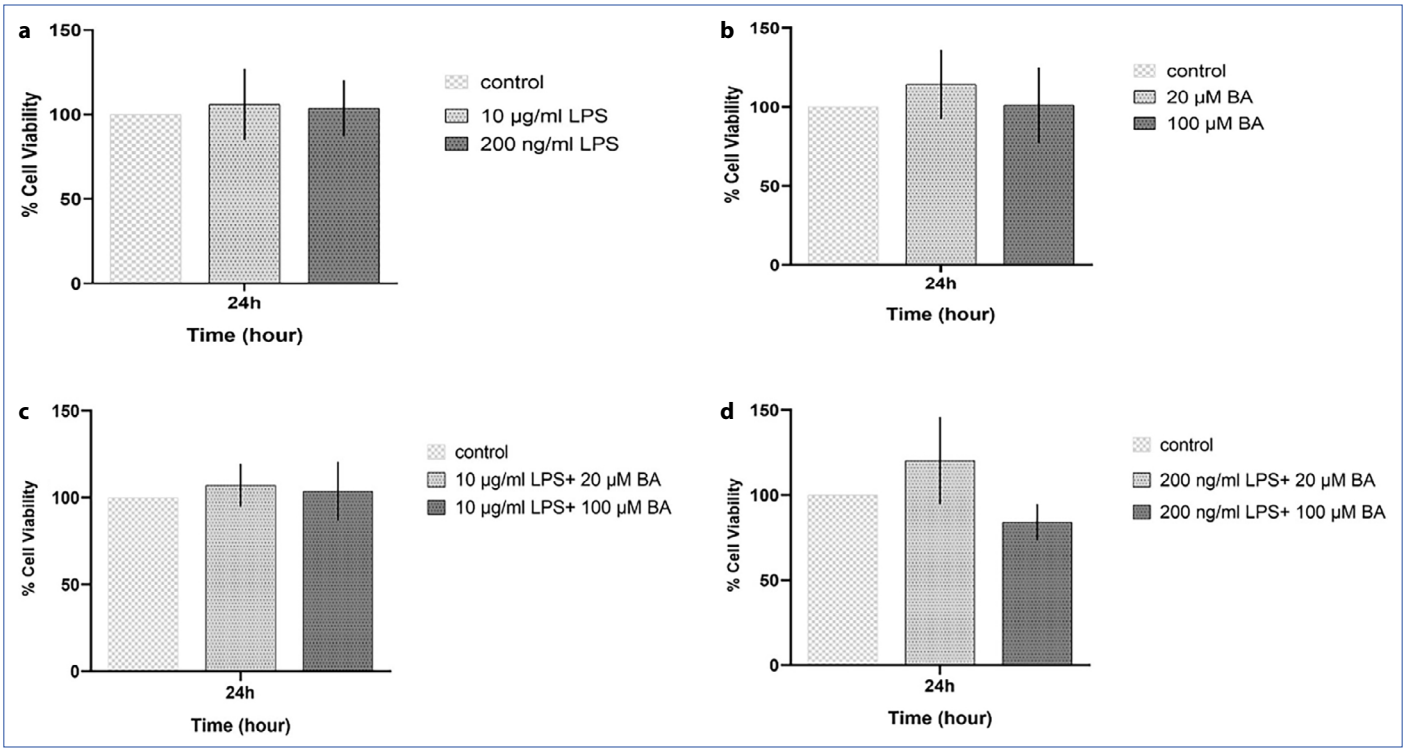

Figure 1. HaCaT cells were treated with different concentrations and combinations of LPS and Boric acid for 24 h and percentage cell viability was determined from CCK-8 results.

LPS: Lipopolysaccharide.

24 h of treatment. As shown in Figure 1, exposure of HaCaT cells to LPS and BA at different concentrations, either alone or in combination, did not induce overt cytotoxicity after 24 h. Cell viability remained comparable to control levels across most treatment groups. Notably, co-treatment with lower concentrations of BA in the presence of LPS resulted in preserved or slightly increased cell viability, indicating that BA does not adversely affect keratinocyte survival under inflammatory conditions at the tested doses.

Cell migration was assessed using the wound healing assay, and representative light microscope images (4× magnification) are presented in Figure 2. At 24 h, LPS-treated cells exhibited delayed wound closure compared with the control group, reflecting impaired migratory capacity. In contrast, cells treated with BA, either alone or in combination with LPS, demonstrated enhanced wound closure, suggesting improved migratory behavior. Quantitative analysis of wound diameter changes at 0 and 24 h is shown in Figure 3. LPS treatment significantly inhibited wound closure compared with control, whereas BA co-treatment significantly reduced wound diameter and accelerated gap closure. The improvement in wound healing observed in BA-treated groups was statistically significant ($p<0.001$), indicating that BA effectively counteracts the inhibitory effects of LPS on keratinocyte migration.

Biochemical parameters

The effects of LPS, BA, and their simultaneous administration on oxidative stress markers and antioxidant defense parameters in HaCaT keratinocytes after 24 h are shown in Figure 4. MDA levels, an indicator of lipid peroxidation, were elevated in LPS-treated cells compared with control. Simultaneous administration of BA attenuated this increase, with both BA concentrations showing reduced MDA levels relative to LPS alone. This effect was more pronounced in the co-treatment groups, indicating a potential inhibitory effect of BA on lipid peroxidation under inflammatory conditions.

AOPP levels did not show a marked change following LPS treatment alone compared with the control group. Similarly, BA treatment alone and simultaneous LPS + BA treatments resulted in comparable AOPP levels, indicating no pronounced protein oxidation under the applied experimental conditions.

TSH levels exhibited modest variations among treatment groups. While LPS treatment alone tended to slightly reduce TSH levels relative to control, co-treatment with BA particularly at higher concentrations was associated with preserved or mildly increased TSH levels compared with LPS-treated groups, suggesting partial maintenance of thiol-based antioxidant capacity.

CAT activity was significantly decreased following LPS exposure compared with the control group. In contrast, BA co-treatment partially restored CAT activity, with both low and high BA concentrations showing higher CAT levels than LPS alone. BA treatment alone maintained CAT activity near control levels.

SOD levels remained largely unchanged following LPS treatment alone. However, BA administration both alone and in

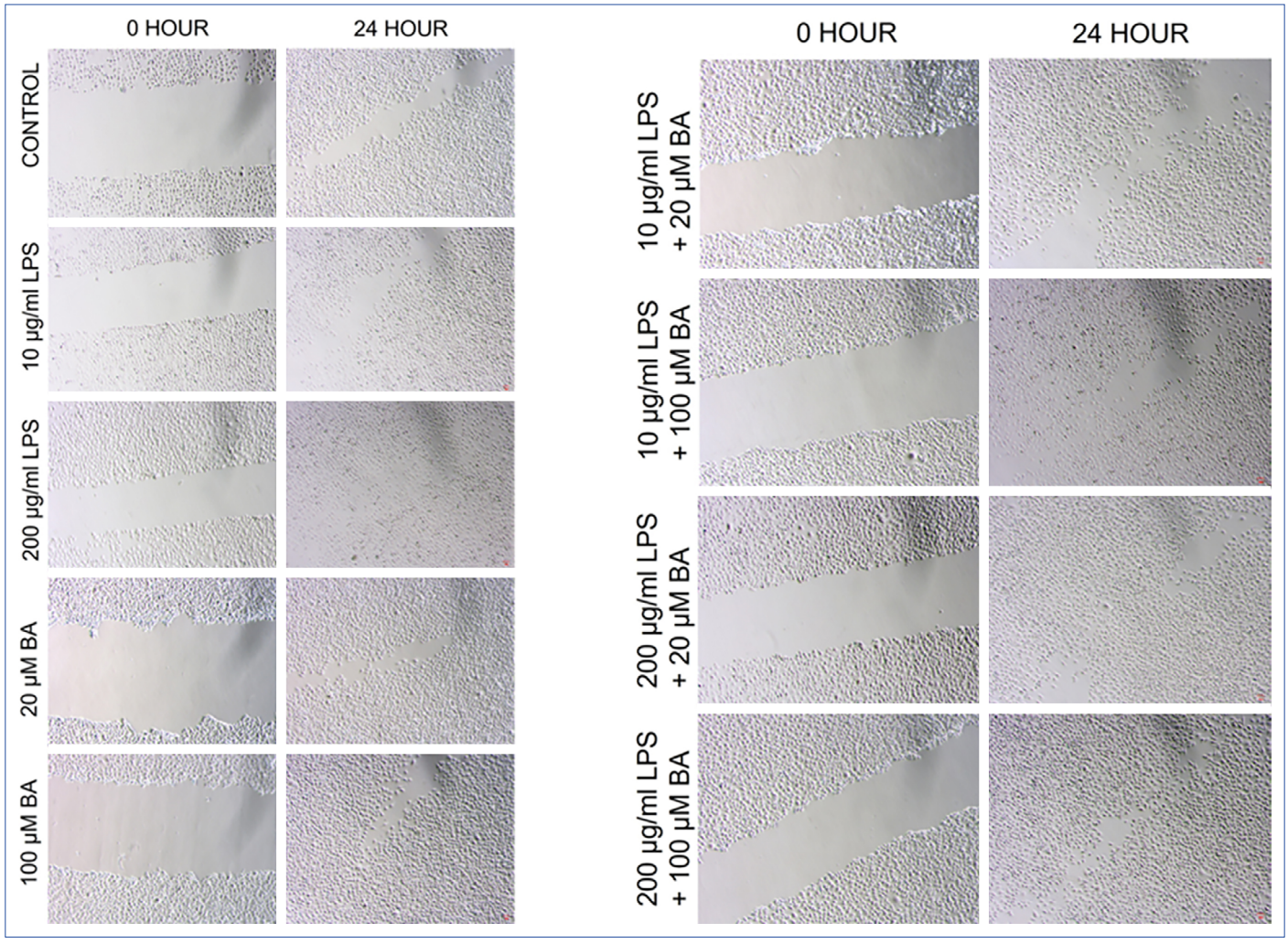

Figure 2. Light microscope images of HaCAT cell migration determined by wound healing method (4X).

combination with LPS was associated with increased SOD levels compared with control, with the highest values observed in the BA-treated groups, indicating enhancement of enzymatic antioxidant defense.

Discussion

The present study demonstrates that BA exerts a concentration-dependent modulatory effect on HaCaT keratinocyte viability under LPS-induced inflammatory conditions. In our experimental model, LPS exposure alone did not induce overt cytotoxicity, which is consistent with previous studies indicating that LPS primarily acts as a proinflammatory stimulus rather than a direct cytotoxic agent in keratinocytes [16, 17]. Notably, simultaneous treatment with low-dose BA (20 µM) significantly enhanced cell viability, whereas higher-dose BA (100 µM) attenuated this effect. These findings suggest that BA supports keratinocyte survival within an optimal concentration range and emphasize the importance of dose-dependent responses. Previous studies have reported that boron compounds promote epithelial cell proliferation and wound repair under physiological conditions [18, 19]; however, our

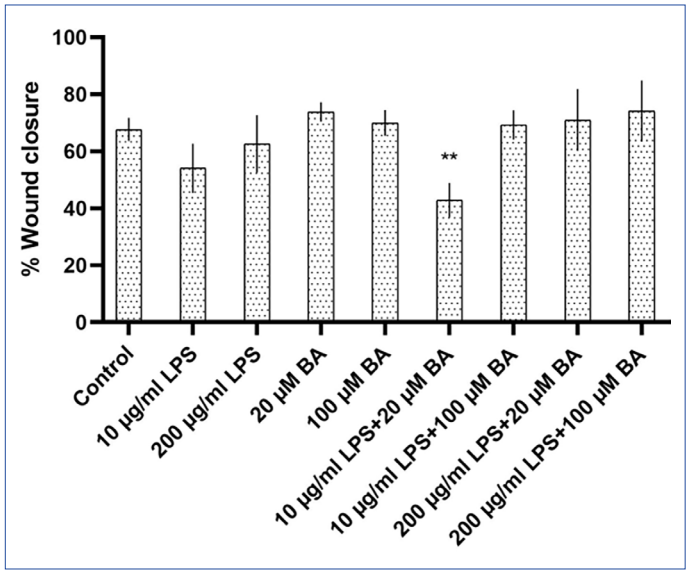

Figure 3. Wound healing effects LPS and Boric acid on HaCaT cells on wound diameter change at 0 and 24 hours (**p<0.001).

LPS: Lipopolysaccharide.

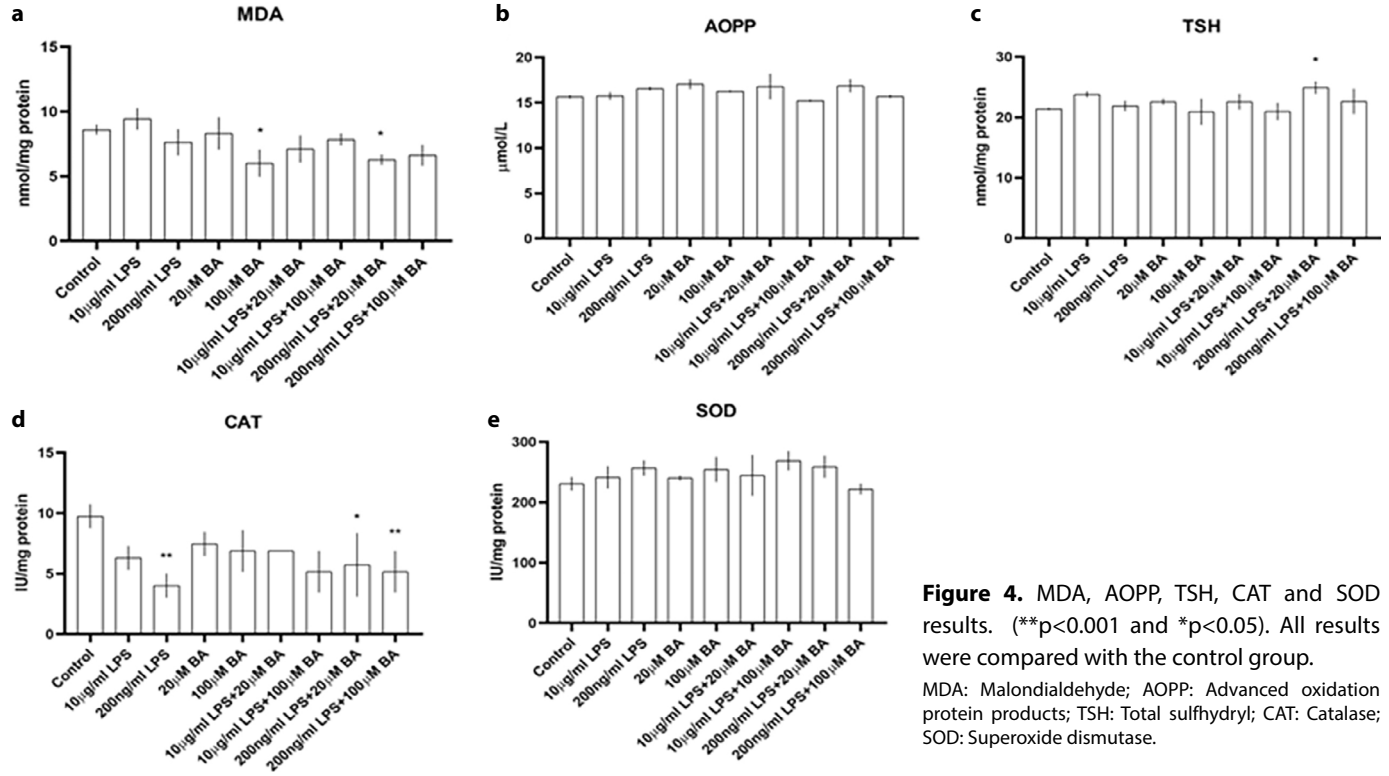

Figure 4. MDA, AOPP, TSH, CAT and SOD results. (**p<0.001 and *p<0.05). All results were compared with the control group.

MDA: Malondialdehyde; AOPP: Advanced oxidation protein products; TSH: Total sulfhydryl; CAT: Catalase; SOD: Superoxide dismutase.

data extend these observations by demonstrating a protective effect of BA on keratinocyte viability specifically during acute inflammatory activation relevant to psoriasis.

Keratinocyte viability is a critical determinant of epidermal homeostasis in psoriasis, where inflammatory stress, abnormal proliferation, and impaired differentiation coexist [20]. Preservation of cell viability during early inflammatory responses may contribute to maintenance of the epidermal barrier and controlled regeneration. Importantly, BA alone did not exert cytotoxic effects at the tested concentrations, supporting earlier toxicological and nutritional studies indicating that BA is well tolerated by epithelial cells within defined dose limits [18, 21]. Collectively, these findings demonstrate that BA preserves HaCaT cell viability and promotes keratinocyte migration under LPS-induced inflammatory conditions, supporting its potential role in maintaining epidermal repair capacity during inflammatory stress.

Beyond its effects on cell survival, our findings highlight a significant role for BA in modulating oxidative stress pathways. LPS stimulation resulted in increased lipid peroxidation, as evidenced by elevated MDA levels, along with suppression of antioxidant enzyme activity, particularly CAT. These results are in line with extensive evidence demonstrating that oxidative stress is a key contributor to psoriasis pathogenesis, driven by excessive ROS production from activated keratinocytes and infiltrating neutrophils [22–25]. Clinical studies evaluating oxidative stress markers in psoriasis have frequently reported elevated MDA levels and altered antioxidant enzyme activities; however, findings have been inconsistent, likely due to heterogeneity in disease severity, stage, and treatment status [26, 27]. Contrasting results have been reported by Gabr and colleagues, who demonstrated significantly elevated MDA levels together with reduced activities of the antioxidant enzymes SOD and CAT in patients with psoriasis [26]. The concomitant increase in lipid peroxidation and depletion of enzymatic antioxidant defenses in their study underscores the presence of pronounced oxidative imbalance in psoriasis. Importantly, these alterations were correlated with disease severity, suggesting that progressive impairment of SOD and CAT activity contributes to the accumulation of oxidative damage, as reflected by increased MDA levels. These findings are in line with our observations, where inflammatory stimulation was associated with enhanced lipid peroxidation and suppression of antioxidant enzymes, while BA treatment attenuated MDA levels and restored SOD and CAT activity. Together, these data support the concept that dysregulation of SOD and CAT plays a pivotal role in oxidative stress–mediated keratinocyte damage in psoriasis and highlight lipid peroxidation as a key downstream consequence of impaired antioxidant defense. Ikonomidis et al. [27] demonstrated that oxidative stress and inflammation-related mechanisms contribute not only to cutaneous pathology but also to systemic vascular dysfunction in patients with psoriasis, showing parallels with coronary artery disease. Their findings highlight increased oxidative burden and impaired antioxidant defenses as key drivers of disease-related tissue damage. Consistent with these observations, our results show that inflammatory stimulation induces oxidative imbalance in keratinocytes, characterized by increased lipid peroxidation and suppression of antioxidant enzymes, while BA treatment restores redox homeostasis by

enhancing SOD and CAT activity and reducing MDA levels. Together, these findings support the concept that oxidative stress represents a common mechanistic link between local keratinocyte dysfunction and systemic complications in psoriasis.

Simultaneous administration of BA markedly attenuated LPS-induced lipid peroxidation and partially restored CAT activity, while enhancing SOD levels. These results suggest that BA reinforces enzymatic antioxidant defense mechanisms and limits early oxidative damage, particularly at the level of membrane lipids. Previous experimental studies have shown that boron modulates redox homeostasis by regulating antioxidant enzyme activity and scavenging ROS [28, 29]. Our study adds to this body of evidence by demonstrating that BA exerts these antioxidant effects specifically under inflammatory stress conditions rather than under basal states alone.

TSH levels were preserved in BA-treated groups, indicating maintenance of thiol-based redox buffering capacity. In contrast, AOPP did not show marked changes across treatment groups, suggesting that protein oxidation may represent a later-stage oxidative event not prominently induced under the acute inflammatory conditions applied in this study. This observation is consistent with previous reports indicating that lipid peroxidation is a more sensitive early marker of oxidative stress in keratinocytes than advanced protein oxidation [30–32]. In addition to redox modulation, BA significantly improved keratinocyte migratory capacity in the wound healing assay, counteracting the inhibitory effects of LPS. Impaired keratinocyte migration is a recognized feature of inflammatory skin diseases and contributes to delayed epidermal repair [18]. Earlier studies have demonstrated that boron-containing compounds promote wound healing and epithelial regeneration, potentially through redox-sensitive signaling and cytoskeletal reorganization [18, 33]. In the context of psoriasis, enhanced keratinocyte migration may facilitate controlled epidermal renewal during inflammatory flare-ups. Overall, these results demonstrate that while LPS induces oxidative imbalance primarily through lipid peroxidation and suppression of antioxidant enzymes, simultaneous treatment with BA modulates oxidative stress markers and supports antioxidant defense mechanisms in HaCaT keratinocytes.

Akçaalan et al. [34] demonstrated that BA significantly stimulates wound closure in HaCaT keratinocytes and modulates epithelial–mesenchymal transition (EMT)–related gene expression, highlighting its role in keratinocyte migration and regenerative responses. These findings are highly consistent with our results, in which BA markedly improved wound closure and counteracted LPS-induced impairment of keratinocyte migration. While Akçaalan et al. [34] focused on EMT-associated transcriptional regulation under non-inflammatory conditions, our study extends these observations by demonstrating that BA preserves migratory capacity even in the presence of inflammatory and oxidative stress. Together, these data suggest that BA supports epidermal repair through complementary mechanisms involving both redox modulation and migration-related cellular programs.

Study Limitations and Future Perspectives

Several limitations of this study should be acknowledged. First, the findings are based on an *in vitro* HaCaT keratinocyte model, which does not fully recapitulate the complex immune–epidermal interactions present in psoriatic skin. The absence of immune cells, such as T lymphocytes and dendritic cells, limits direct extrapolation to *in vivo* disease mechanisms. Second, the study focused on acute inflammatory responses induced by LPS and therefore does not reflect chronic or recurrent inflammatory conditions characteristic of long-standing psoriasis. Third, molecular signaling pathways underlying BA-mediated protection such as NF-κB, MAPK, or Nrf2 signaling were not directly investigated. In addition, inflammatory cytokine profiling (e.g., IL-6, IL-8, TNF-α) and psoriasis-relevant axis stimulation (e.g., IL-17A/IL-23) were not assessed; because the revision relied on archived lysates, additional cytokine assays could not be performed in this cycle. Finally, pathway-level validation (e.g., NF-κB, MAPK, Nrf2/HO-1) and intracellular ROS measurement were not included, and mechanistic inferences should be interpreted cautiously.

Conclusion

Collectively, our findings demonstrate that BA confers protective effects in LPS-stimulated HaCaT keratinocytes by supporting cell viability, attenuating oxidative stress, enhancing antioxidant defenses, and promoting cell migration. These results provide experimental support for the potential role of BA as a candidate redox-modulating compound targeting oxidative and inflammatory processes relevant to psoriasis. Further studies using immune–keratinocyte co-culture systems, chronic inflammation models, and *in vivo* approaches are warranted to clarify the underlying molecular mechanisms and to evaluate the translational potential of BA in psoriasis management.

Disclosures

Ethics Committee Approval: Ethics Committee approval is not required for cell culture studies.

Informed Consent: Informed consent was obtained from all participants.

Conflict of Interest Statement: None declared.

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