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Antioxidative Effects of Boron Minerals in LPS-Induced Psoriasis Model

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Abstract

Objectives: Psoriasis is a chronic inflammatory skin disorder that substantially affects patients' quality of life. Oxidative stress arises when there is a disruption in the balance between reactive oxygen species (ROS) and the body's antioxidant defense mechanisms, significantly contributing to its pathophysiology. This study aimed to investigate the effects of various boron compounds—borax, colemanite, boric acid, and ulexite—on oxidative stress markers in an LPS-induced psoriasis-like cell culture model using HaCaT keratinocytes.

Methods: HaCaT cells were stimulated with LPS to induce a psoriasis-mimicking inflammatory response. Subsequently, cells were treated with different boron compounds. To evaluate antioxidant activity and lipid peroxidation, oxidative stress markers such as superoxide dismutase (SOD), glutathione peroxidase (GPx), and malondialdehyde (MDA) were examined.

Results: Boron compounds modulated oxidative stress parameters by enhancing SOD and GPx activities and reducing lipid peroxidation, as reflected by decreased MDA levels. Among the tested agents, colemanite and ulexite exhibited the strongest antioxidant activities, likely attributable to their distinct mineral compositions.

Conclusion: Findings suggest that boron compounds hold therapeutic potential for reducing oxidative stress in psoriasis-like conditions. Further research is needed to clarify the underlying molecular mechanisms and to refine boron-based therapeutic applications.

Keywords: Psoriasis, LPS, boron compounds, borax, ulexit, colemanite

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LPS ile İndüklenen Psöriasis Modelinde Bor Minerallerinin Antioksidan Etkileri

Öz

Amaç: Psoriasis, hastaların yaşam kalitesini önemli ölçüde etkileyen kronik inflamatuvar bir deri hastalığıdır ve patogenezinde reaktif oksijen türleri (ROS) ile antioksidan savunma mekanizmaları arasındaki dengenin bozulduğu oksidatif stres kritik bir rol oynar. Bu çalışma, LPS ile psöriasis benzeri inflamatuar yanıt oluşturulan HaCaT keratinosit kültüründe bor bileşiklerinin (boraks, kolemanit, borik asit ve üleksit) oksidatif stres belirteçleri üzerindeki etkilerini araştırmayı amaçlamaktadır.

Yöntemler: HaCaT hücrelerinde LPS ile inflamasyon indüklenmiş ve ardından farklı bor bileşikleri uygulanmıştır. Süperoksit dismutaz (SOD), glutatyon peroksidaz (GPx) ve malondialdehit (MDA) düzeyleri ölçülerek antioksidan enzim aktiviteleri ve lipit peroksidasyonundaki değişimler değerlendirilmiştir.

Bulgular: Boron bileşiklerinin SOD ve GPx aktivitelerini artırarak antioksidan yanıtı güçlendirdiği, aynı zamanda MDA düzeylerindeki azalma ile lipit peroksidasyonunu azalttığı belirlenmiştir. Bileşikler arasında özellikle kolemanit ve üleksitin mineral yapılarından kaynaklanabilecek belirgin antioksidan etki gösterdiği saptanmıştır.

Sonuç: Bu bulgular, bor bileşiklerinin psöriasis gibi oksidatif stresle ilişkili deri hastalıklarında terapötik potansiyel taşıdığını göstermektedir. Altta yatan moleküler mekanizmaların aydınlatılması ve bor temelli tedavi stratejilerinin optimize edilmesi için ileri çalışmalara ihtiyaç vardır.

Anahtar kelimeler: Psöriasis, LPS, boraks, üleksit, kolemanit.

INTRODUCTION

Psoriasis is a persistent inflammatory condition of the skin that impacts 2-3% of people worldwide and significantly reducing quality of life. The pathophysiology of psoriasis involves complex and dynamic interactions between skin and immune cells. Many research studies have highlighted the involvement of the immune system, along with its network of interacting leukocytes and cytokines, in the development of psoriasis[1,2]. Free radicals, characterized by their unpaired electrons, often engage with other substances as electron acceptors or oxidizing agents. Among these, reactive oxygen species (ROS) are particularly significant, as they are a leading cause of oxidative damage within cells. Additionally, ROS play a role in generating other reactive species[3].

Mitochondria, peroxisomes, inflammatory cells, flavins, adrenaline, dopamine, quinones, the cytochrome P450 enzyme complex, NADPH oxidase, and xanthine oxidase are among the internal sources of reactive oxygen species (ROS). External contributors can include environmental pollutants, radiation, optical radiation, anticancer medications, and chemical substances like tobacco and alcohol. The primary forms of ROS are superoxide anion (O_2^-), hydroxyl radical (OH), and hydrogen peroxide (H_2O_2)[3].

ROS can also damage cellular structures such as DNA, RNA, proteins, and lipids, with lipid peroxidation being a prominent consequence in lipids. Lipid peroxidation is a process where polyunsaturated fatty acids found in the membranes of mammalian cells react with free oxygen radicals. This interaction results in the creation of compounds such as hydroxy fatty acids, alcohols, aldehydes, peroxides, pentane, ethane, and malondialdehyde (MDA). Measuring the amount of MDA, one of the byproducts of this process, allows for the assessment of lipid peroxidation through an MDA test[4].

Antioxidants serve as the body's defense mechanisms against oxidants, maintaining redox balance and neutralizing reactive species. Key antioxidant enzymes include catalase

(CAT), glutathione peroxidase (GPx), and superoxide dismutase (SOD). In addition to these, there are non-enzymatic antioxidants such as vitamins E, C, and A, flavonoids, albumin, glutathione, thioredoxins, uric acid, and polyphenol metabolites. Measuring these antioxidants provides information about the antioxidant status of cells or tissues[3].

Recent studies suggest that increased production of ROS, impaired antioxidant function, and oxidative stress may contribute to the pathogenesis of psoriasis[1,5,6]. Recent progress in comprehending autoimmune inflammatory pathways and new insights into the pathogenesis of psoriasis have paved the way for the creation of biological drugs that precisely target elements of effector immune mechanisms. These biological therapies have transformed the management of psoriasis. Nonetheless, these current biological treatments may not work for every patient or might lose their effectiveness over time. Coupled with the substantial costs of these medications, these challenges have spurred the continuous pursuit of alternative treatment options[5,7]. Given the strong evidence that ROS-mediated oxidative stress is involved in numerous biological responses that may contribute to the pathogenesis of inflammatory skin diseases, including psoriasis, leveraging this knowledge to develop new therapeutic strategies is of considerable interest for managing psoriasis.

Intense oxidative stress can result in cellular aging and even cell death, whereas moderate oxidative stress might enhance cell survival by triggering widespread alterations in gene expression and modifications of proteins after they are translated[8]. Psoriasis involves the activation and growth of skin and immune cells, suggesting that mild oxidative stress might be more influential in its development than intense oxidative stress.

Boron (B) is a metalloid with a molecular weight of 10.81 g/mol and an atomic number of 5. It is an essential element for humans, absorbed through food intake[8]. In nature, boron is not found in its free form but in an oxygen-bound state known as borates. There are approximately 250 different boron minerals in nature, with minerals containing Na, Ca, and Na-Ca playing important roles in biological processes. Sodium-containing boron minerals include borax, calcium-containing boron minerals include colemanite, and sodium-calcium-containing minerals include ulexite. Although this element has physiological functions at low concentrations, it has been reported to have toxic effects at higher concentrations[9].

Oxidative stress biomarkers can include the measurement of free radicals, damage products of lipids, proteins, and DNA, and the assessment of antioxidant defense systems. One of the most frequently used methods for evaluating oxidative status is through the measurement of Total Antioxidant Status (TAS) and Total Oxidant Status (TOS). Additionally, the measurement of oxidative stress biomarkers such as SOD, GPx, and MDA provides insights into the oxidative stress status of cells or tissues[10,11].

In vitro studies on psoriasis frequently utilize the HaCaT keratinocyte cell line. Treating HaCaT cells with lipopolysaccharide (LPS) is a widely used method for inducing a psoriasis model by stimulating proinflammatory cytokines[12].

Bor derivatives such as borax, colemanite, and ulexite have diverse applications. Borax is commonly used in various household laundry and cleaning products, including laundry boosters, powdered hand soaps, and some tooth whitening formulas[13]. Colemanite and ulexite are widely used in sectors such as glass, metallurgy, fiberglass, fertilizer, detergent, and cosmetics[14].

In this study, a cell culture psoriasis model was established by treating the HaCaT keratinocyte cell line with LPS. This model was used to investigate the effects of borax, colemanite, and ulexite on oxidative stress indices and oxidative stress markers, specifically SOD, GPx, and MDA.

METODS

Cell Culture

HaCaT (RRID: CVCL_0038) keratinocyte cells were grown using DMEM-high glucose (Gibco) enriched with 10% FBS (Gibco) and 1% penicillin-streptomycin (Gibco). The cultivation took place in an incubator with 5% CO2 and 95% air humidity, maintained at 37°C. The culture medium was replaced every 2-3 days. Once the cells reached 80 to 90% confluence, they were detached with a 0.25% trypsin-EDTA solution and then replated for further growth.

Based on the previous data, cultured cells were treated with 200 ng/ml LPS (Sigma, L2630, USA) for 24 hours to create psoriasis model[6,15].

MTT assay

The effects of the following agents were tested: borax ($Na_2B_4O_7 \cdot 10H_2O$, CAS No.1303-96-4), colemanite ($Ca_2B_6O_{11} \cdot 5H_2O$, CAS No. 1318-33-8), boric acid, ulexite. The substances were procured from Eti Mine Works General Management in Turkey. Borax, colemanite, boric acid and ulexite's impact on psoriasis model cell proliferation was assessed using the MTT assay, following the manufacturer's protocol (MTT Cell Viability Assay Kit; Biotium, cat no: 30006). Cells were plated in 96-well plates at a density of 1×10^4 cells per well. After 24 hours of incubation, the cells were exposed to borax, colemanite, boric acid, and ulexite solutions at concentrations of 250 µg/ml, 500 µg/ml, 750 µg/ml, and 1000 µg/ml dissolved in dH2O for durations of 24 and 48 hours. Control cells remained untreated either LPS or boron compounds. The selected concentrations were determined in accordance with previously published study[6].After the incubation period, the MTT solution was introduced, and the formation of formazan was measured at 570 nm, using 630 nm as a reference wavelength, with a Biotek microplate reader. To standardize the absorbance values, the background absorbance was deducted from the signal absorbance.

The viability percentage was calculated using the formula:

Viability (%) = Absorbance of experiment well / Absorbance of control well × 100

Total Antioxdant Status (TAS) and Total Oxidant Status (TOS)

HaCaT (control) and Psoriasis model cells were cultured in T-25 flasks (Sarstedt) at a concentration of 1×10^6 cells. Following a 24-hour incubation, psoriasis model cells were exposed to 1000 µg/ml of borax, 250 µg/ml of colemanite, 1000 µg/ml of boric acid, and 1000 µg/ml ulexite for 24 hours.

TAS levels were determined using TAS assay kits (Cat.No: RL0017, Relassay, Turkey). This novel automated method relies on the reduction of a more stable ABTS (2,2′-Azino-bis(3-ethylbenzothiazoline-6-sulfonic acid)) radical cation by antioxidants, resulting in the bleaching of its characteristic color. TAS levels were expressed in mmol Trolox equivalent/L.

TOS levels were measured using TOS assay kits (Cat.No: RL0024, Relassay, Turkey). In this assay, oxidants in the sample oxidize the ferrous ion-o-dianisidine complex to ferric ion. Glycerol molecules in the reaction medium enhance this oxidation reaction. In an acidic environment, the ferric ion interacted with xylenol orange to create a colored complex. The strength of this color, determined through spectrophotometry, is directly related to the total quantity of oxidant molecules in the sample. The assay was standardized using hydrogen peroxide, and the TOS levels were reported as micromolar hydrogen peroxide equivalents per liter (μmol H2O2 equivalent/L).

Oxıdatıve Stress Index (OSI)

OSI was determined as the ratio of TOS to TAS[16,17]. To calculate OSI, TAS results were converted to micromoles per liter (μmol/L), and then OSI was computed using the following formula:

OSI (arbitrary unit) = TOS (μol H2O2 equivalent/L) / TAC (μol Trolox equivalent/L).

SOD, GPx, MDA ELISA

HaCaT and psoriasis model cells were seeded at a density of 1x106 cells per well in a 6-well plate. After a 24-hour incubation period, the cells were exposed to 250 μg/ml of Colemanite, 1000 μg/ml of Borax, 1000 μg/ml boric acid, and 1000 μg/ml ulexite. Following treatment, the culture medium was collected after 24 hours, and the levels of SOD, MDA, and GPx were assessed using enzyme-linked immunosorbent assay (ELISA). The collected media were stored at -20°C until ELISA analysis was conducted. ELISA assays were performed in accordance with the manufacturer's instructions using kits with the following catalog numbers: E4502Hu, E3921Hu, and SH0020 from BT-Lab, China.

RESULTS

Effect of Borax, Boric Acid, Colemanite and Ulexite on Psoriasis model

The impact of borax, boric acid, colemanite, and ulexite on a psoriasis model was assessed using the MTT assay. This approach allowed for the examination of how boron compounds affect the psoriasis model over time and at varying doses. The IC50 dose for borax was determined to be 1000 μg/ml at the 24-hour mark, while colemanite's IC50 dose was 250 μg/ml at the same time point. Both boric acid and ulexite had an IC50 dose of 1000 μg/ml (Fig 1).

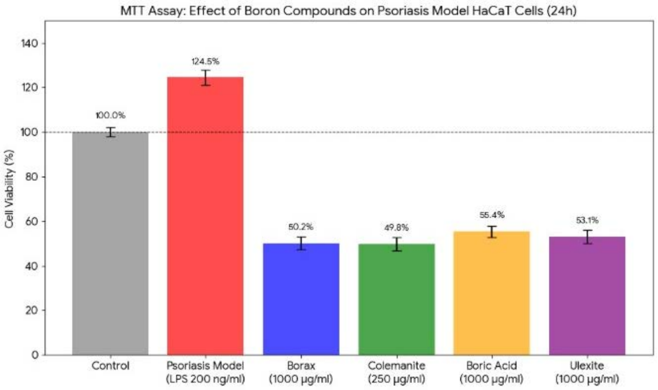

Figure 1. MTT results of borax, boric acid, colemanite and ulexite.

Oxidant/Antioxidant effect of boron compounds in LPS-induced keratinocytes

To assess the oxidant and antioxidant activities of boron compounds in keratinocytes treated with LPS, both the total oxidant and antioxidant statuses were measured, and the OSI was determined. The OSI levels in LPS-treated cells showed a significant difference compared to the HaCaT control group, with values of 2.310 and 0.407, respectively ($p<0.05$). When comparing LPS-treated cells to those treated with both borax and LPS, the OSI levels were 2.310 and 2.139, respectively ($p<0.05$). Additionally, cells treated with colemanite and LPS exhibited a statistically significant difference from the LPS-treated group, with OSI values of 0.691 and 2.310, respectively ($p<0.05$). Furthermore, the OSI values for cells treated with both ulexite and LPS compared to the LPS-treated group were 2.008 and 2.310, respectively ($p<0.05$). LPS-treated cells and the group which is treated both with boric acid and LPS OSI levels were 2.310 vs 1.634, respectively ($p<0.05$) (Fig 2.).

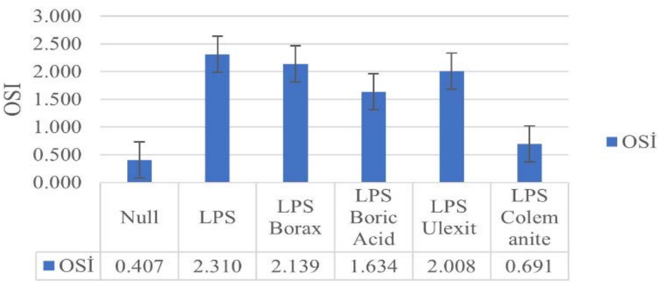

Figure 2. Oxidative stress index values of cell groups.

To elucidate the alterations in the oxidative stress index, we assessed the activities of the antioxidant enzymes GPx and, SOD as well as the levels of the lipid peroxidation product MDA. These measurements were subjected to statistical analysis to evaluate their significance in reflecting changes in oxidative stress. Twenty-four-hour borax treatment caused alterations in the antioxidant enzyme activities of psoriasis model. GPx level decreased with LPS treatment on HaCaT control group (9.0 vs 5.2, $p<0.05$) but treatment with LPS and borax restore the GPx level in the control group (8.2 vs 9.0, $p>0.05$). Boric acid treatment did not affect the GPx level on LPS-treated psoriasis model (5.5 vs 5.2 respectively, $p>0.05$). Ulexit treatment decreases (3.2 vs 5.2, $p>0.05$) and colemanite treatment increases GPx level on LPS-treated group (Fig 3.).

SOD level is decreased with LPS treatment on HaCaT control group (0.29 vs 1.51, $p<0.05$) and borax treatment increases the SOD level on LPS-treated psoriasis model (0.29 vs 0.44, $p<0.05$). Boric acid also increases the SOD level on LPS-treated psoriasis model (0.59 vs 0.44, $p<0.05$). SOD level increased with colemanite application on LPS- treated group (0.62 vs 0.29, $p<0.05$) and ulexit treatment also increases the SOD level on LPS-treated psoriasis model (0.62 vs 0.29, $p<0.05$) (Fig 3.).

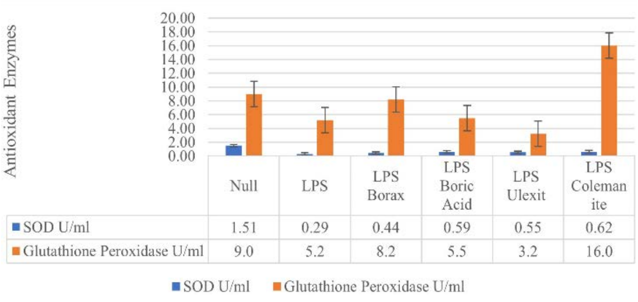

Figure 3. SOD and GPx levels of cell groups.

MDA level is increased with LPS treatment on HaCaT control group (0.70 vs 0.49, $p<0.05$) and borax treatment decreases the MDA level on LPS-treated psoriasis model (0.66 vs 0.70, $p<0.05$). Boric acid also decreases the MDA level on LPS-treated psoriasis model (0.67 vs 0.70, $p<0.05$). MDA level decreased with colemanite application on LPS- treated group (0.28 vs 0.70, $p<0.05$) and ulexit treatment also decreases the MDA level on LPS-treated psoriasis model (0.36 vs 0.70, $p<0.05$) (Fig 4.).

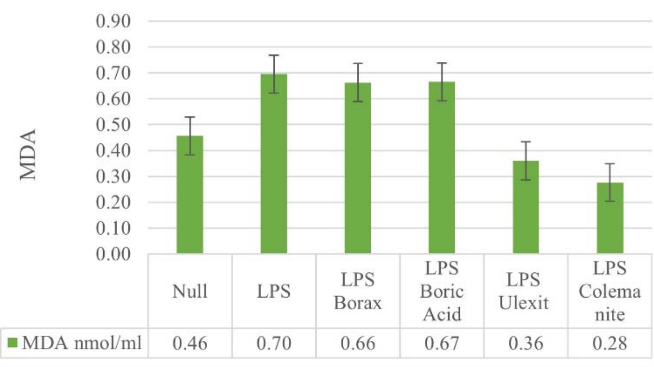

Figure 4. MDA levels of cell groups.

DISCUSSION

Psoriasis is a multifactorial skin disease marked by chronic and recurrent inflammatory episodes, with oxidative stress recognized as a significant factor in its pathophysiology. In psoriasis, oxidative stress arises when there is a disruption in the balance between the generation of ROS and the body's antioxidant defenses, with elevated ROS levels causing harm to cells[18]. Excessive ROS generated in skin and immune cells can harm critical biomolecules, including DNA, RNA, proteins, and lipids, exacerbating inflammation and advancing disease progression.

Research suggests that mild oxidative stress may play a more prominent role in psoriasis pathogenesis compared to severe oxidative stress, likely due to continuous, low-level activation of inflammatory pathways[19]. This underscores the importance of antioxidants as the body's primary defense against ROS. Both enzymatic antioxidants, like SOD and GPx, and non-enzymatic antioxidants work to neutralize ROS and prevent oxidative damage. In this study, treatments with boron compounds, especially colemanite and ulexite, significantly increased SOD and GPx activities, indicating that these compounds may enhance the cellular antioxidant defense system against oxidative stress.

Lipid peroxidation, a critical indicator of oxidative stress, significantly impacts cell membrane integrity and initiates inflammatory signaling. In psoriasis, ROS attack polyunsaturated fatty acids in cell membranes, triggering a chain reaction that leads to lipid peroxide formation and secondary products like MDA. Our study showed that colemanite treatment significantly reduced MDA levels, suggesting that boron compounds may inhibit lipid peroxidation and preserve cellular structure under oxidative stress[20]. By mitigating lipid peroxidation, boron compounds may thus help limit cellular damage and inflammation in psoriasis.

The differential effects of various boron compounds on oxidative stress markers highlight their potential for targeted therapeutic applications. For instance, colemanite and ulexite had distinct effects on antioxidant enzyme activities, with colemanite showing a particularly strong effect on both SOD and GPx. These findings suggest that different boron compounds might selectively modulate antioxidant defenses, providing a basis for developing targeted therapies. Additionally, the concentration-dependent effects observed in this study are important to consider, as lower concentrations appeared beneficial, while higher doses could potentially induce toxicity[8].

In a study by Turkez et al. investigating the effects of boron compounds on glioblastoma cell lines, it was demonstrated that boric acid and borax altered oxidative stress parameters. These compounds increased MDA levels while also enhancing SOD and catalase activities. Additionally, boric acid application regulated inflammatory responses by reducing IL-1α, IL-6, and TNF-α levels. This highlights the modulatory effects of boron compounds on inflammation and oxidative stress[21]. Boron compounds are also known to modulate immune responses and play a role in inflammatory processes. A review by Romero-Aguilar et al. emphasized the effects of boron compounds on innate and adaptive immunity. Boric acid was shown to promote T cell and B cell proliferation while regulating the secretion of specific cytokines. This suggests the potential use of boron compounds in therapeutic strategies targeting inflammatory processes within the immune system[22].

The anti-inflammatory effects of boron compounds are extensively documented in the literature. Particularly, a study by Demirci et al. demonstrated that boric acid and sodium pentaborate suppressed iNOS and COX-2 gene expression and reduced NO production in LPS-stimulated macrophages[23]. These effects are critical for suppressing inflammation and accelerating wound healing. Considering the inflammatory processes in psoriasis pathogenesis, these findings support the therapeutic potential of boron compounds.

In summary, boron compounds hold promise as therapeutic agents for managing oxidative stress and inflammation in psoriasis. By enhancing key antioxidant enzyme activities and reducing lipid peroxidation, compounds like colemanite may support cellular defenses against oxidative damage. The ability of boron compounds to modulate antioxidant systems, as evidenced in this study, indicates they could be integral to innovative psoriasis treatment strategies.

CONCLUSION

This study highlights the potential of boron compounds to reduce oxidative stress and enhance antioxidant defenses in inflammatory conditions like psoriasis. The observed effects on oxidative stress parameters, especially the increased activity of antioxidant enzymes, suggest that boron compounds may mitigate oxidative damage in psoriasis. These findings provide a foundation for further research into boron-based therapies, potentially leading to novel strategies for psoriasis management and treatment.

Ethics Committee Approval: Ethical approval was not required for this study as it involved only cell culture experiments.

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Conflict of interest: The authors declare that there are no conflicts of interest regarding the publication of this paper.

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