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Short communication

Oxidative stress in psoriasis: biomarkers, antioxidant defense and clinical implications

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Introduction

Psoriasis is a chronic immune-mediated inflammatory skin disease characterized by abnormal keratinocyte proliferation and persistent activation of inflammatory pathways. Although traditionally regarded as a dermatological disorder, it is now recognized as a systemic inflammatory disease associated with comorbidities such as psoriatic arthritis, metabolic syndrome, and cardiovascular disease (Armstrong et al., 2025). Alongside immune dysregulation, oxidative stress has emerged as an important biochemical dimension of psoriasis. Oxidative stress reflects an imbalance between reactive oxygen species (ROS) production and antioxidant defense capacity, leading to damage of cellular lipids, proteins, and nucleic acids and potentially amplifying inflammatory signaling. Although numerous studies have reported altered oxidative stress markers and disturbances in antioxidant defense in psoriasis, the precise clinical significance of these findings remains incompletely defined (Bilski et al., 2024; Dobrică et al., 2022). In this context, oxidative stress represents a relevant biochemical perspective for understanding the inflammatory and systemic complexity of psoriasis.

Inflammation and oxidative stress

Inflammation and oxidative stress are closely interconnected processes in psoriasis. The persistent inflammatory environment characteristic of the disease promotes increased production of reactive oxygen species by activated immune cells, including neutrophils, macrophages, and T cells. In turn, excessive ROS generation further amplifies inflammatory signaling, creating a positive feedback loop that sustains immune activation and tissue injury. Key cytokines involved in psoriasis, particularly TNF-α, IL-17, and IL-23, have been associated with increased oxidative burden and disruption of redox homeostasis. Excess ROS may induce lipid peroxidation, protein oxidation, and DNA damage, while also disrupting normal keratinocyte differentiation and function. These observations support the view that oxidative stress is not merely a secondary consequence of inflammation, but an active contributor to the initiation and maintenance of psoriatic pathophysiology (Dobrică et al., 2022; Pleńkowska et al., 2020).

Biomarkers and antioxidant defense

Numerous studies indicate that psoriasis is accompanied by alterations in both oxidative

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damage biomarkers and antioxidant defense systems. Among the most frequently investigated markers, malondialdehyde (MDA), a by-product of lipid peroxidation, is commonly reported at elevated levels in serum and psoriatic lesions, reflecting increased oxidative damage to cellular membranes. Increased levels of 8-hydroxy-2′-deoxyguanosine (8-OHdG) and total oxidative status (TOS) further support the presence of oxidative injury and systemic oxidative burden. At the same time, several components of the antioxidant defense system appear to be compromised. Reduced glutathione (GSH), a major intracellular antioxidant, is often decreased, while lower activities of enzymes such as superoxide dismutase (SOD) and glutathione peroxidase (GPx) suggest impaired ability to counteract reactive oxygen species. Findings for catalase (CAT) remain less consistent, but total antioxidant capacity (TAC) is frequently reduced, indicating depletion of antioxidant reserves. Interpretation of these findings remains complicated by differences in sample type, analytical methodology, and patient characteristics across studies. Although variability exists among studies, the overall evidence supports disturbed redox homeostasis in psoriasis, characterized by increased oxidative damage and weakened antioxidant protection (Bilski et al., 2024; Dobrică et al., 2022; Göçer Gürok et al., 2025; Pleńkowska et al., 2020).

Clinical implications

The potential clinical relevance of oxidative stress in psoriasis has received increasing attention. Several studies suggest associations between oxidative stress parameters and disease severity, most commonly assessed using the Psoriasis Area and Severity Index (PASI). Elevated levels of markers such as MDA and TOS, together with reduced antioxidant capacity, may reflect a greater inflammatory and oxidative burden in more severe disease. In addition, improvements in clinical status after treatment have in some cases been accompanied by partial normalization of redox-related parameters, suggesting a possible link between oxidative balance and therapeutic response. However, the clinical utility of these biomarkers in routine assessment, prognosis, or treatment monitoring remains insufficiently established. At present, these markers appear more useful for mechanistic interpretation than as established tools for routine clinical decision-making. This is partly due to the lack of standardized methodologies, variability in biomarker selection, and heterogeneity among study populations. Moreover, it remains unclear whether oxidative stress acts primarily as a pathogenic driver or as a secondary consequence of chronic inflammation. Further studies are therefore needed to clarify the mechanistic and clinical significance of redox-related biomarkers in psoriasis (Bilski et al., 2024; Dobrică et al., 2022).

Conclusion

Current evidence identifies oxidative stress as one of the most consistent biochemical themes in psoriasis. Increased markers of oxidative damage together with alterations in antioxidant defense systems support the presence of a significant redox imbalance associated with disease pathophysiology. However, despite promising findings, the clinical utility of oxidative stress biomarkers still requires validation in standardized and well-designed studies. A more integrated understanding of oxidative stress in psoriasis may improve biochemical interpretation of the disease and support future translational and patient-oriented research.

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