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Antioxidant Techniques And Oxidative Stress In Dermatology

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ABSTRACT

A prooxidant-antioxidant imbalance causes oxidative stress, which damages cells. Free radicals that are produced during normal aerobic metabolism and pathological inflammatory processes, such as reactive oxygen or nitrogen species, mediate it. Skin acts as a defence mechanism that is crucial in preventing both internal and external noxious stimuli and preserving homeostasis. It is becoming more and more clear that oxidative stress plays a role in a variety of skin problems, and that antioxidative tactics can be used as quick and simple ways to treat these conditions. Here, we examine the therapeutic use of antioxidants in dermatology and the dysregulation of antioxidant systems.

Keywords: Oxidative stress, Antioxidant, Dermatology, Skin

INTRODUCTION

Superoxide anion (O2), peroxides, hydroxyl radical (OH°), and singlet oxygen are examples of reactive oxygen species (ROS) (1O2).[1] These chemicals can harm DNA (DNA base damage, DNA single-strand and double-strand breaks, DNA and protein crosslinks, DNA and chromosomal abnormality), lipid membranes, collagen structures, and mitochondrial function. They can promote proliferative and cell survival signals.

In response to signals from cytokines, growth hormones, airborne pollutants, UV radiation, food additives/preservatives, cosmetics, medications, and physiological stimuli, keratinocytes and nearly all types of skin cells create ROS. Antioxidants are often divided into endogenous and exogenous categories. The skin has a robust antioxidant system that includes enzyme-based antioxidants like glutathione peroxidase (GPX), glutathione S-transferase, glutathione reductase, superoxide dismutase (SOD), and catalase, as well as non-enzymatic antioxidants like ascorbic acid (vitamin C), glutathione (GSH), ubiquinol, uric acid, vitamin A, melanin, Other examples of endogenous non-enzymatic antioxidants

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include flavonoids, coenzyme Q10, alpha-lipoic acid, selenium, pyruvate, and bilirubin. Antioxidants can also be obtained exogenously through dietary consumption. Lycopene, curcumin, green tea, Coffea arabica, silymarin, polypodium leucotomos, resveratrol, grape seed extract, pomegranate, pycnogenol, soy isoflavones, propolis, and squalene are examples of this class of antioxidants or foods that contain them.[4] Antioxidants are more abundant in the epidermis of skin than the dermis. These antioxidants are often dispersed in a gradient pattern, with higher quantities being detected in the stratum corneum's deeper layers.[5]

N-Acetylcysteine decreases NF-B binding to the NF-B binding site of the VCAM-1 gene and prevents IL-1-induced mRNA upregulation and expression of E-selectin and vascular cell adhesion molecule 1 (VCAM-1) in endothelial cells. [11] Resveratrol, acetylcysteine, and tea polyphenols prevent ROS-regulated MMP expression. [12] Although cutaneous lymphocyte-associated antigen (CLA) production and functional activity are inhibited by N-acetylcysteine, alpha-tocopherol, and ascorbate in isolated human T-lymphocytes,[13] these antioxidants do not prevent contact dermatitis when applied topically.[14] Cell-permeable SOD inhibits TNF-induced MMP-9 in keratinocytes; as a result, SOD is hypothesised to function as an immunomodulatory substance in inflammatory skin conditions. [15] In irritating and ACD models, a topical PPAR-alpha activator promotes antioxidant enzymes and lowers inflammation.[16]

Scleroderma

It is believed that oxidative stress is a significant factor in the emergence of disease. Patients with scleroderma had higher levels of the oxidative stress marker, 8-isoprostane, in their serum. [16] Similar to TAC, serum TOS and OSI levels that are elevated in systemic sclerosis were also elevated. TAC may also operate as a signal for predicting the likelihood of lung and gastrointestinal involvement. [17]

N-acetylcysteine was recently demonstrated to minimise skin fibrosis in a mouse model of scleroderma produced by bleomycin; this antioxidant also markedly decreased the MDA and protein carbonyl contents in mouse skin.[18]

Pemphigus foliaceus as well as Pemphigus vulgaris

In patients with pemphigus vulgaris (PV), serum GPX, catalase, and GSH in erythrocyte and plasma, plasma -carotene, vitamin E, and vitamin A are decreased, but MDA in erythrocyte and plasma is raised.[19] PV patients have higher levels of lipid hydroperoxide (LOOH) and serum TOC. 30 While GPX, vitamin C, selenium, and bilirubin levels in PV patients were comparable to controls, plasma uric acid was reduced. [21]In patients with pemphigus foliaceus (PF), MDA, conjugated dienes, catalase, and SOD activities are elevated while protein thiol levels are lowered.[22]

Acne

Patients with acne have lower leukocyte SOD and GPX activity and higher serum TBARS and MDA levels. 18 Acne tissue scrapings show elevated levels of SOD, catalase, GSH, MDA, and adenosine deaminase. 19 Acne patients had lower plasma levels of vitamins A and E. 20 Squalene peroxide levels,

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which lower GSH, are higher and vitamin D levels, which boost GSH, are lower in acne-related sebum. Patients with acne had considerably lower plasma levels of vitamin E, vitamin A, and zinc; there was also a negative link between the severity of the acne and vitamin E and zinc levels.[21]Numerous studies have demonstrated the efficacy of various antioxidants for treating acne, including topical or oral zinc, nicotinamide, and sodium ascorbyl phosphate, a precursor to vitamin C.[22] A multi-nutrient antioxidant capsule including zinc, vitamin C, carotenoids, D-alpha-tocopherol acetate, chromium, selenium, and vitamin E, as well as lactoferrin, is another antioxidant that has shown effectiveness in treating acne.[23]

However, oral isotretinoin therapy for severe acne reduces serum paraoxonase-1 activity by increasing oxidative stress and increases erythrocyte lipid peroxidation, GSH, and GPX. It was proposed that this behaviour constituted a pathomechanism for the adverse effects of isotretinoin.[24]

Rosacea

According to some theories, ROS produced by inflammatory cells, including neutrophils in rosacea, may be linked to inflammation. [25] Patients with rosacea have higher serum levels of peroxide and cutaneous ferritin while having lower serum levels of total antioxidant capacity.[16] Antioxidant activity is exhibited by several effective rosacea treatments, such as metronidazole, tetracyclines, azelaic acid, and azithromycin.[17] There were mixed outcomes when zinc sulphate was used orally in regards to the treatment of rosacea.[18]

Persistent venous ulcer

Potentially, oxidative stress impairs wound healing.[19] It has been shown that the levels of 8-isoprostane and the allantoin:uric acid percentage ratio (AUR) have increased in chronic wounds. 50 GPX activity, selenium, zinc, and iron levels are all lowered, whereas superoxide and iron deposition with enhanced ROS are increased. [21] It was discovered that endothelial cells and macrophages both displayed increased inducible cyclooxygenase-2 activity.The activities of MMPs and serine proteases as well as the total amount of iron present in chronic exudates are all elevated [22,24] Upregulated levels of SOD, MDA, and NO in valve tissues suggest higher oxidative stress.[25]

UV causes the depletion of endogenous antioxidants as well as the production of ROS and lipid peroxidation products (TBARS). In the skin, ultraviolet light depletes GPX, ascorbate, GSH, SOD, catalase, alpha-tocopherol, and ubiquinol. The epidermis exhibits greater antioxidant system damage than the dermis. [16]

Prior to exposure to UV light, antioxidants are crucial for protecting the skin. Photoprotective effects can be produced by antioxidants like vitamin C, vitamin E, coenzyme Q10, lycopene, carotenoids, tretinoin, GSH, zinc, resveratrol, genistein, cocoa, selenium, and polypodium leucotomos. Melatonin, green tea, silymarin, soy isoflavones, lutein, and zeaxanthin are additional antioxidants with photoprotective qualities. [17]

Psoriasis

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The mouse psoriasis model caused by imiquimod displays an abnormal antioxidant system.In mouse skin, myeloperoxidase (MPO) and GSH/GSH disulfide (GSSG), one of the markers of oxidative stress, are elevated, whereas SOD activity and levels are lowered.[18] Redox sensitive cellular signalling pathways that are involved in the development of psoriasis include NF-B, Janus kinase-signaling transducers, and mitogen-activated protein kinase/activator protein 1, among others.[19]

Dimethylfumarate has anti-inflammatory effects via upregulating GSH and NAD(P)H:quinone oxidoreductase 1 (NQO1) and activating the Nrf2 transcriptional pathway. These effects include the downregulation of cytokines and adhesion molecules.[20]High levels of ROS are present in psoriatic dermatitis caused by imiquimod; therefore, appropriately elevated levels of ROS may prevent psoriasis by promoting indoleamine 2,3-dioxygenase (IDO) expression and Treg function. It is possible to link the effectiveness of phototherapy and hyperbaric oxygen therapy for the treatment of psoriasis to the elevation of ROS levels, which improves Treg function. [21]

CONCLUSION

Despite claims to the contrary, oxidative stress has a significant impact on the development of several cutaneous conditions by different redox-sensitive routes, with reported inconsistent outcomes thinking about the oxidant/antioxidant states in these illnesses.The following can be used to explain this discrepancy:

(1) the amounts of inherent compounds in various tissue samples

(2) ROS can impact various complicated signalling pathways. oxidative stress, biochemical processes, and (2) is not always the source of inflammation.

Conclusions defending the effectiveness of antioxidant Still elusive are effective treatments. However, there have Numerous papers discuss the use of powerful antioxidants treating cutaneous illness, in addition to traditional medications which have antioxidant properties. focusing on oxidative

Various skin conditions may respond well to stress; Therefore, additional research is necessary to establish a foundation for antioxidant treatment strategies for each illness.

Acknowledgements

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