

Nutraceuticals which are used as skin shield: A review of the evidence

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ABSTRACT

Nutraceuticals are essential for maintaining healthy skin. Nutraceuticals like as probiotics, phenolics, and vitamins are among the nutraceuticals that may help prevent and manage dermatologic diseases. Probiotics, vitamin E, and green tea catechins may provide the most comprehensive set of skin-protective mechanisms, with probiotics having the broadest therapeutic application. Recent study has focused on the effects of probiotics on atopic dermatitis and opportunistic infections of skin burns. This contains a $p=0.02$ improvement in Scoring Atopic Dermatitis index scores when intact *Lactobacillus rhamnosus* Goldin and Gorbach (LGG) was used instead of heat inactivated LGG or a placebo. *Lactobacillus reuteri*, whether given

before or after a *Staphylococcus aureus* infection, can improve epidermal keratinocyte survival, $p=0.01$. It's possible that phenolics haven't been thoroughly researched for atopic dermatitis or skin burns. Phenolics, on the other hand, play a role in photoprotection. The phenolic rutin enhances UVB radiation filter reactive oxygen species scavenging by 75%, $p=0.002$, and peak wavelength absorption by 10%, $p=0.001$. While oral and topical probiotics offer unexplored potential in the treatment of atopic dermatitis and the prevention of skin infections, phenolics will become more widely employed in photoprotection. Nutraceuticals will become increasingly important for good skin care as bioavailability, dose, and formulation are improved.

Key Words: *Atopic dermatitis; Green tea; Human skin; Keratinocyte; Moisturizer; Nutraceuticals; Photoprotection; Polyphenols; Probiotics; Vitamin E*

INTRODUCTION

Nonmelanoma Skin Cancers (NMSC) are one of the most prevalent human malignancies, having an 18- to 20-fold higher incidence than melanoma [1]. They are made up mostly of Basal Cell Carcinomas (BCC) and Squamous Cell Carcinomas (SCC). As a result, photocarcinogenesis prevention is critical. The incidence of NMSC varies around the world. BCC is most frequent in Australia and the United States of America (USA), with over 1000 and 450 cases per 100,000 person-years [2]. BCC is the least well-documented disease in Africa, with a prevalence of less than one per 100,000 person-years [3]. Meanwhile, SCC occurs at a third of the rate of BCC in England, which is 22.65 per 100,000 person-years [2,3]. At least 1.3 million NMSCs occur each year in the United States, impacting one in every five people and costing more than \$650 million USD. The intrinsic photocarcinogenesis prevention organ is the skin, which is a multifunctional organ. The skin regulates body temperature and water content, preventing dehydration. Infection, physical injury, and Ultraviolet Radiation (UVR) are all protected by the skin [4]. Heat, wetness, UVR, and direct mechanical trauma are all modalities that can cause skin damage. Immunosuppression, UVR-dependent acute inflammation with dermal leukocytic infiltration, and erythema (sunburn) are all dose-dependent photo-carcinogenic and photo-damaging effects of UVR from a 200 nm to 400 nm spectrum [5]. Acute inflammation activates cytosolic phospholipase A2, which raises free arachidonic acid, the substrate for eicosanoid production by cyclooxygenases and lipoxygenases, and results in prostaglandin E2 (PGE2) and 12-lipoxygenase-derived 12-hydroxyeicosatetraenoic acid (HETE), the two main players in sunburn [6].

UVA, with a wavelength range of 320 nm to 400 nm, is the deepest penetrating UVR, penetrating 1000 m into the epidermis and dermis and accounting for 90% to 95% of UVR. UVA disrupts melanogenesis by inducing oxidative stress and impairing antioxidant defences, whereas UVB and UVC damage DNA directly [1,7]. UVA increases tyrosinase concentration and activity, as well as Reactive Oxygen Species (ROS) and 8-hydroxy-2'-deoxyguanosine (8-OHdG), while decreasing glutathione S-transferase (GST) [7]. UVA causes nuclear factor E2-related factor 2 (Nrf2) nuclear translocation and transcriptional activity to decrease one hour after exposure. Downregulation of Glutamate Cysteine Ligase Catalytic subunit (GCLC), GST, and

nicotinamide adenine dinucleotide quinone oxidoreductase 1 (NQO1) has been linked to UVA radiation. UVA inhibits Extracellular Signal-Related Kinases (ERK), Jun Nuclear Kinase (JNK), and p38 mitogen-activated protein kinases (MAPK) in the same way as it inhibits Nrf2 nuclear translocation [7]. Keratinocytes are the main cells of the epidermis, the outermost layer of the skin. The stratum corneum outer horny layer contains the most differentiated keratinocytes, while the stratum basale nearest to the dermis has the least differentiated keratinocytes. Collagen, elastin, fibroblasts, and immune cells make up the dermis, which is largely connective tissue [8]. The dermis and epidermis receive vascular and neuronal networks from the underlying hypodermis. The stratum corneum, or outside horny skin layer, is elastic and moisturised when it is healthy. Cracking, pruritus, and scaling are symptoms of a skin barrier problem caused by a dehydrated, inelastic, and damaged outer horny skin layer [8].

Atopic dermatitis (eczema; AD) is linked to a thickening of the outer horny skin layer [5]. AD affects about 14% of children under the age of four [9]. In their lifetime, up to 20% of people worldwide may get Alzheimer's disease. AD costs roughly \$1 billion per year in the United States. Externally applied moisturisers improve the barrier function of dry skin and reduce inflammation, which is part of the treatment for Alzheimer's disease [5,9]. Immunologic components are present in skin barrier diseases such as Alzheimer's disease [5]. Extrinsic or allergic AD, which involves IgA, IgE, and regulatory T-cells, accounts for up to 80% of AD [9].

The hygiene hypothesis proposes that a lack of antigen exposure due to cleanliness reduces self-reactive T-cell suppression, resulting in increased autoimmunity and immunologic system-based diseases, such as atopic dermatitis. The hygiene hypothesis has been updated to include a link between early childhood immune system development and the hygiene hypothesis, as well as an altered gut microbiota. As a result, allergy and autoimmune disease susceptibility rises. The connection of filaggrin mutation and increased *Staphylococcus aureus* in 42 percent of AD patients supports the hygiene theory of decreased microbial antigen exposure as an AD risk factor [10].

Probiotics

In childhood, the Actinobacteria *Propionibacterium* and *Corynebacterium*,

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Firmicutes Staphylococcus, Proteobacteria, and Bacteroidetes dominate the skin commensal microbiome. Skin commensals directly protect against pathogenic microorganisms and modify immune responses mediated by type 1 T-helper (Th1) and type 2 T-helper (Th2) cells [10]. The makeup of skin commensals varies in AD [10]. In 20% of persons, stable *S. aureus* colonisation with less than 10⁶ cfu/cm² of skin occurs, while intermittent carriage occurs in 60% of people. *S. aureus* at a concentration of 10⁶ cfu/cm² of skin is normally considered an infection [8]. On the other hand, generates dose-dependently severe opportunistic infections in heat damaged (burned) skin as well as cracked, dehydrated AD skin.

As a result, any skin barrier disruption, such as burns, Alzheimer's disease, or acne vulgaris, puts 80% of persons at risk of opportunistic *S. aureus* infections. A change in skin commensal bacterial microflora has also been linked to Alzheimer's disease and acne vulgaris [9].

Probiotics are live, active microbial cultures that are Generally Recognized as Safe (GRAS) when ingested in amounts that benefit the host [8,9]. Bacteria that have been heat-inactivated or destroyed are not probiotics. Probiotics have not been found to improve the gastrointestinal barrier function or general health of preterm newborns. Probiotics kill pathogenic microorganisms by increasing ceramide formation without affecting the host and increasing mucin secretions that trap pathogens through acid and bacteriocin secretion.

Probiotics also defend against pathogenic microorganisms by preventing them from attaching to cell receptors and competing for nutrients. For added protection against harmful microorganisms, probiotics can modulate and regulate the host's innate immune responses [8]. Lactobacilli, particularly *Lactobacillus plantarum*, have been proven to stimulate and regulate the immune system in a nonspecific way. Lactobacilli prevent pathogen-induced apoptosis in phagocytes. Probiotics, like the skin commensal microbiota, have anti-pathogenic and anti-inflammatory immunologic activities. As a result, it is scientifically plausible that probiotics can act in place of the typical skin commensal microbiome when the skin commensal microbiome composition changes in AD and heat injured skin [7].

Polyphenols: Flavonoids, phenolic acids, stilbenes, and proanthocyanidins

UVR absorption by pigmented polyphenols is largely in the UVB range, with some UVA and UVC absorption as well [1]. Camellia sinensis-derived tea is the world's second most popular drink, accounting for 20% of all green tea consumed. UVR-induced p53 expression has been shown to be reduced by topical green tea extract [1]. Through dihydrofolate reductase inhibition, Epigallocatechin-3-Gallate (EGCG) has been linked to poor in vitro nucleotide biosynthesis repair. UVB-induced photodamage is inhibited by topical resveratrol, silymarin, oral resveratrol, silymarin, and grape seed proanthocyanidins in mouse models. Polyphenols' anti-photo-carcinogenesis activity is mediated by this anti-inflammatory action [1,4].

Flavonoids

Green Tea Catechins (GTC), which are anti-inflammatory, antioxidant, and polyphenolic, inhibit cyclooxygenase-2 and lipoxygenases [6]. Orally ingested GTC conjugates and metabolites are absorbed into the skin and have been identified in skin biopsy tissues [6]. Oral GTC and EGCG were found to protect mice from UVR-induced photodamage and photocarcinogenesis [1,6]. Green tea's principal catechin and most bioactive component is EGCG. Topical GTC before UVR exposure reduced photodamage, and oral GTC reduced photodamage by increasing the skin erythema (sunburn) threshold in open, uncontrolled human trials [6]. In controlled human experiments, pre-exposure to UVR with EGCG (1 mg/cm²) reduced photodamage. Sunburn was decreased by 38.9% in a 34-day, 39-person controlled experiment comparing topical 4% green tea extract (OM24) versus GTC prior to UVB exposure [1].

Phenolic acids

Dietary phenolic acids are largely UVA-absorbing antioxidants that have an indirect regulatory influence on the Nrf2-ARE pathway. Nrf2 inhibits melanogenesis and tyrosinase protein expression in primary Human Epidermal Melanocytes (HEMn) and B16F10 melanoma cells. Partially UVA protectants include caffeine and ferulic acid. UVA protection is provided by quercetin and rutin. Avobenzene is a good UVA filter that doesn't have any antioxidant properties [7]. UVA causes B16F10 melanoma cells to produce more melanin, increase tyrosinase activity, and increase tyrosinase protein expression. Caffeic acid, ferulic acid, quercetin, rutin, and avobenzene reduced melanin synthesis and tyrosinase activity in B16F10 melanoma cells.

Quercetin is the most potent inhibitor of melanin formation and tyrosinase activity, with an IC₃₀ of 7.8 1.4 μM against melanin production and 10.1

3.1 μM against tyrosinase activity. Rutin, caffeic acid, and avobenzene all have similar but less effective action than quercetin, with p values of 0.05, 0.01, and 0.001, respectively. Ferulic acid is the least active against melanin formation and tyrosinase activity, with an IC₃₀ >30 [7]. Despite this, quercetin, caffeic acid, and avobenzene have been shown to counteract UVA-induced Nrf2 nuclear translocation, Nrf2-ARE downregulation, and GCLC, GST, and NQO1 decrease. When rutin is introduced to UVB filters, ethylhexyl methoxycinnamate, ethylhexyl dimethyl PABA, and octocrylene organic UV filters, ROS scavenging is improved by 75% (p=0.002) and peak wavelength absorption is raised (p=0.001), although being less effective than quercetin [7].

Vitamins C and E

Vitamins C and E are natural components of the skin. UVR-induced erythema is unaffected, although oral vitamin C administration of 500 mg daily increases skin and plasma vitamin C levels. Topical vitamin C at a concentration of up to 10% does not irritate or sensitise human skin. α-, β-, δ-, and γ-tocopherols, as well as α-, β-, δ-, and γ-tocotrienols are understood by vitamin E. Importantly, tocopherols and tocotrienols have separate bioactive distribution compartments. Although dose-dependent -tocopherol depletion by UVB with lipophilic penetration in the brain and liver and even intramembrane positioning close to membrane surfaces is consistent with vitamin E inactivation of ROS from UVR exposure, tocotrienols are more powerful antioxidants than α-tocopherol [6].

CONCLUSION

The preceding sections, as well as the synopsis of selected references, demonstrated a wide range of skin-protecting nutraceuticals. A particular nutraceutical can be helpful for distinct pathologic diseases via diverse modes of action. Probiotics are helpful for immunologic-based AD, preventing skin dryness and infections, and promoting wound healing through many modes of action. With a growing body of research, recommendations for maternal oral probiotic use during pregnancy and breastfeeding have gained traction over time. The delivery route influences the microbiota of a person. Oral maternal combination probiotics are a way to give people who are born by caesarean a microbiome that is similar to people who are born vaginally. Given the reduced AD severity indicated by SCORAD, p=0.022, the *L. salivarius* symbiotic should be explored for AD treatment. The efficacy of probiotics is influenced by their formulation. Before significant effects on AD may be shown with *B. breve* as a combination probiotic in fermented foods, more research is needed.

AUTHOR CONTRIBUTIONS

Researchers conceived and designed the commentary, performed the literature search, analysed the retrieved articles, and wrote the commentary.

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CONFLICTS OF INTEREST

The author declares no conflict of interest.

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