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INVITED REVIEW

Exploring the impact of solar radiation on skin microbiome to develop improved photoprotection strategies

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Abstract

The skin microbiome undergoes constant exposure to solar radiation (SR), with its effects on health well-documented. However, understanding SR's influence on host-associated skin commensals remains nascent. This review surveys existing knowledge on SR's impact on the skin microbiome and proposes innovative sun protection methods that safeguard both skin integrity and microbiome balance. A team of skin photodamage specialists conducted a comprehensive review of 122 articles sourced from PubMed and Research Gateway. Key terms included skin microbiome, photoprotection, photodamage, skin cancer, ultraviolet radiation, solar radiation, skin commensals, skin protection, and pre/probiotics. Experts offered insights into novel sun protection products designed not only to shield the skin but also to mitigate SR's effects on the skin microbiome. Existing literature on SR's influence on the skin microbiome is limited. SR exposure can alter microbiome composition, potentially leading to dysbiosis, compromised skin barrier function, and immune system activation. Current sun protection methods generally overlook microbiome considerations. Tailored sun protection products that prioritize both skin and microbiome health may offer enhanced defense against SR-induced skin conditions. By safeguarding both skin and microbiota, these specialized products could mitigate dysbiosis risks associated with SR exposure, bolstering skin defense mechanisms and reducing the likelihood of SR-mediated skin issues.

KEYWORDS

exposome, photoprotection, skin microbiome, solar radiation, ultraviolet radiation

Abbreviations: 6-HAP, 6-N-hydroxyaminopurine; AK, actinic keratosis; AMP, antimicrobial peptides; IL-6, interleukin 6; MAPK, mitogen-activated protein kinase; MED, minimal erythema dose; MMP, matrix metalloproteinase; NF-κB, nuclear factor kappa-light-chain-enhancer of activated B cells; PAMP/DAMP, pathogen/damage-associated molecular pattern; PLE, polymorphic light eruption; ROS, reactive oxygen species; SC, skin cancer; SCC, squamous cell carcinoma; SR, solar radiation; ssUV, solar-simulated UV; TEWL, transepidermal water loss; TGF-β, transforming growth factor beta; UV, ultraviolet; UVA, ultraviolet A; UVB, ultraviolet B; UVR, ultraviolet radiation; XO, Xanthine oxidase.

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INTRODUCTION

The skin provides a physical, chemical, and immunological protective barrier between the body and the environment.[1,2] It hosts a community of a multitude of commensal microbes named the microbiota which is the collection of all genetic material of all its members.[3,4] The microbiota forms together with the skin the microbiome is the first shield of the human body against external aggression and pathogens.[5,6] The skin microbiome permanently coevolves and a healthy microbiota helps keep the host's health balanced by participating in the host's defense against exposome factors during his entire life.[7–11]

The exposome comprises all external and internal factors to which the body is exposed, including solar radiation (SR), chemicals, pollutants, tobacco smoke as well as lifestyle factors, dietary habits, and infectious agents that a body encounters throughout its lifetime, from conception to death and that impact on its health.[12–17]

A disturbed microbiome may be caused by exposome factors that include medication such as antibiotics, antiseptics, diet, skincare routines, exercise, pollution, and climate parameters as well as SR may result in dysbiosis or an unbalancing of the skin homeostasis.[8,18–23] As a result of dysbiosis, inflammatory skin conditions and, potentially, skin cancer (SC) including melanoma may be observed.[22,24–27] Moreover, exposome factors, are involved in skin photoaging, hyperpigmentary skin disorders, and other photodermatoses, especially in exposed areas of the skin.[28–33]

The commensal microbes differ greatly by site, and the skin microbiome is regularly exposed to SR.[34] While both the positive and negative impact of SR on our health are well defined and understood, the understanding of the role of SR on the microbial dynamics of host-associated commensals of the skin microbiome is still in its infancy.[35]

Currently recommended means to protect from excessive SR include adequate clothing, wearing a hat, and using sunscreens with high sun protection factors as well as avoiding prolonged exposure during midday.[36–39]

Sunscreens have been marketed for decades to primarily protect the skin against SR-induced erythema that is mostly due to UVB exposure. Today, most broad-spectrum sunscreens provide protection against ultraviolet B (UVB) radiation and ultraviolet A (UVA) radiation. But visible and infrared light are also impacting the skin, especially in skin photoaging and hyperpigmentation.[40,41]

The present review provides an overview of what is known about the impact of SR on the skin microbiome and a perspective on innovative sun-protecting products that not only protect the skin but are also able to limit the negative impact of SR on the skin microbiome.

MATERIALS AND METHODS

A group of 10 dermatologists, scientists, and researchers specialized in skin photodamage and photoprotection reviewed 122 publications about the impact of SR, and the skin microbiome published between 1987 and 2023 and available from the PubMed and Research Gate databases.

Keywords alone or combined with each other included skin microbiome, photoprotection, photodamage, skin cancer, ultraviolet radiation (UVR), infrared light, visible light, solar radiation, skin commensals, skin protection, and pre- and probiotics During the second step, the experts discussed the potential of innovative sun protection and future strategies that may be able to leverage the skin microbiome to protect against the deleterious effects of SR.

RESULTS

While a rich literature is available about the impact of SR on the skin and the role of sunscreens to protect the body from SR, only little information is available about the potential impact of SR on the human skin microbiome, its consequences on the host's skin health and about novel approaches to protect the skin from SR exposome damage.[35]

Solar radiation, skin microbiome, and immunity

UVR may directly or indirectly impact the skin microbiome.[42,43] Shifts in the skin microbiome composition with bacteria responding differently, especially to ultraviolet A (UVA) and ultraviolet B (UVB) have been observed.[35,44,45] Figure 1 shows how sun exposure affects the diversity and composition of the skin microbiota and how sunscreens help to maintain healthy skin and its microbiome.

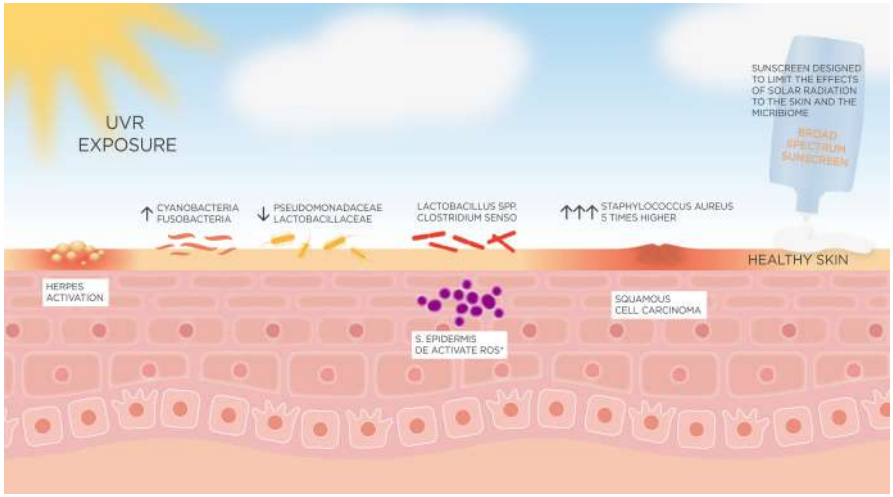

FIGURE 1 Sun exposure affects the diversity and composition of the skin microbiota. Following UVR exposure occurs an increase in *Cyanobacteria* spp., *Fusobacteria* spp, and decreased *Lactobacillaceae* spp. and *Pseudomonadaceae* spp. *Lactobacillus* spp., and *Clostridium* sensu were the most discriminately higher genera in the healthy skin microbiome. *Staphylococcus epidermidis* was shown to be able to generate electrons which deactivate ROS which otherwise cause cellular damage. Increase of Staphylococcus aureus in squamous cell carcinoma and in actinic keratosis.

A study compared two groups of healthy volunteers exposed to SR. One group, exposed to the sun in the summer, was compared with a group wearing clothes throughout the year including the summer period. The study compared the seasonal effects on the skin microbiome before and after summer. Three skin sites were sampled: the inner forearm, dorsal forearm, and cheek. No significant differences in the diversity of the microbiome were observed and no significant seasonal differences in high-abundance species at any of the sampling sites were observed, in any group. These findings suggest a certain stability of the skin microbiome, even after several months of exposure to SR. However, significant differences were observed in low-abundance species in the unprotected areas of the inner and outer arms in the group exposed to SR during the summer months. These changes in low-abundance species are of interest requiring further research.[48]

Willmott et al.[47] observed in a study published in 2023 a significant change in the microbial beta diversity after 4 weeks of extensive exposure to sunlight at the forearm of the subjects compared to baseline suggesting that sun exposure affects the diversity and composition of the skin microbiota. Moreover, the overall composition of the skin microbiome may be long-term altered following UVA and UVB radiation at the back of the volunteers.[44] A general increase in *Cyanobacteria* spp., *Fusobacteria* spp., *Verrucomicrobia* spp., and *Oxalobacteraceae* spp., was observed. Conversely, *Lactobacillaceae* spp. and *Pseudomonadaceae* spp. decreased following UVR exposure. The latter decreased to a greater extent following UVA exposure.

In parallel, several species developed mechanisms to resist against this aggressor. These defense mechanisms are not only the result of selective pressure but also of the potential of UVR to damage DNA, increasing the mutational rate of microbes.[49] As a result, microbial species exhibit a vast array of natural adaptations to protect against UVR.[50,51] For example, *Staphylococcus epidermidis* generated electrons deactivating ROS which otherwise cause cellular damage.[52] *Micrococcus luteus* which uses carotenoid pigments and a high endonuclease activity limited the bactericidal effects of UVR.[53,54]

The process of the alteration of the skin microbiota by UVR is not completely elucidated. The skin microbiota may be altered by microbial photoproducts including pyrimidine dimers and/or 6–4 photoproducts produced after UVR exposure. UVR may trigger a pathogen/damage-associated molecular pattern (PAMP/DAMP) response resulting in various microbial signals comprising oleic acid, LPS and/or porphyrins.[55–58] The latter affect the overall immune signaling cascade, leading to inflammation, and an altered immune response.

Moreover, an increased production of natural antimicrobial peptides (AMP) by microbes or AMP produced by keratinocytes and controlled by microbes under exposure UVR may contribute to an altered immune response and a change of the microbial load by affecting the microenvironment. Under UVR stress conditions, the microbiome may trigger interleukin-1 and together with directly induced microbial signals, influence skin immunity by the release of various cytokines such as that of Th17 pathway. As a result, the keratinocyte effector function may be influenced through the production of interleukin-17, resulting in an altered AMP production affecting the microbiome.[42]

With UVR being able to suppress the body's immune response to infectious microorganisms, it may also be able to increase the risk of microbial infections or worsen existing infections due to UVR-caused dysbiosis and altered skin homeostasis. However, clinical evidence is still very low.[59]

Table 1 provides an overview of data generated about the impact of solar radiation on the healthy skin microbiome.

Solar radiation and modulation of the skin microbiome response

Due to its capacity to adapt, to a certain extent, to exposome factors, the skin microbiota is able, to protect itself and its host against external aggression.

The release of protease enzymes by commensal microbes is involved in desquamation, renewal of the *stratum corneum*, biofilm, and bacteriocins production, as well as quorum sensing while sebum and free fatty acid production by the host participate in the regulation of the skin pH.[6,60–62] Moreover, the release of lipase enzymes helps in the lipidic film surface breakdown process and urease enzymes participate in urea degradation. Commensal microbes also compete for nutrients and space and thus limit the invasion and reproduction of non-commensal potentially detrimental microbes through the release of antimicrobial peptides (AMPs).[6,63]

A transcriptomic analysis in a mouse model revealed a different gene regulation in the presence or absence of the microbiota after UV exposure.[43] In the absence of the skin microbiota, UV exposure led to an increased release of pro-inflammatory cytokines such as IL-1b, IL-6, and IL-18rap. Conversely, in the presence of the microbiota, immunosuppressive cytokines such as IL-10, IL-10ra, IL-20rb, and IL-7r were more prevalent. These results confirm that microbes or microbial products have immunoregulatory effects and may participate in the protection against UV-induced skin neoplasia and modulate gene expression in the skin. Moreover, they may be able to influence the epidermal development and differentiation and wound healing.[1,51,60,64–66] In addition, certain skin-resident microbes and microbial products are capable to regulate AMP expression induced by UVR, potentially through the production of pro-inflammatory cytokines.[67,68]

UVR, microbiome dysbiosis, and skin cancer

Currently, evidence that UVR may cause skin cancer due to skin microbiome dysbiosis is low.[44,69]

Nakatsuji et al.[51] suggested that a specific strain of *S. epidermidis* that produces 6-N-hydroxyaminopurine (6-HAP) protects in a mouse model of photocarcinogenesis against the development of keratinocyte carcinomas. Additionally, the authors demonstrate that Intravenous injection of 6-HAP in mice suppressed the growth of B16F10 melanoma cells without evidence of systemic toxicity. This molecule inhibited DNA polymerase activity and the proliferation of tumor lines but not that of normal keratinocytes.

An animal study published in 2022 revealed a close link between melanoma progression and dysbiosis of the skin microbiome.[27] A significant difference in microbiome diversity and richness between melanoma tissue and healthy skin was observed. *Lactobacillus* spp., *Clostridium* sensu stricto *1*, and *Corynebacterium 1* were the most discriminately higher genera in the healthy skin microbiome, while *Fusobacterium* spp., *Trueperella* spp., *Staphylococcus* spp., *Streptococcus* spp., *and Bacteroides* spp. were discriminately abundant in the skin microbiome of melanoma lesions.

Kullander et al.[70] reported that *Staphylococcus aureus* (*S. aureus*) and squamous cell carcinoma (SCC) were associated with an almost five times higher colonization rate with *S. aureus* in SCC lesions than in healthy skin. However, the authors also stated that the study design did not allow to determine whether the association implies that *S. aureus* may influence carcinogenesis or if SCC has an increased susceptibility to *S. aureus*. But, with AK lesions being considered precursors of SCC, an increased colonization of *S. aureus* in AK may indicate a carcinogenic process from AK to SCC.[70–72]

Skin microbiome and photoprotection

Today, there is large evidence of acute and chronic skin alteration caused by SR and especially by UVR and the benefits that sunscreens offer to reduce and prevent such damages.[73] However, information still mostly focuses on the protection of the skin as an organ, and data about the protective effect of sunscreens that contain prebiotics or probiotics that also protect the microbiota are sparse.[74,75]

Probiotics and Postbiotics are recognized to help in maintaining human health in supporting disease prevention and management and, in the future, may potentially play a beneficial role in photoprotection.[74,76–80] Table 2 provides a summary of all studies that assessed the benefit of pro and postbiotics as photoprotective ingredients.

UVR and the use of sun protective measures were reported to impact the relative bacterial abundances of the skin microbiome.[104] The proximity of *Micrococcus luteus* to *Corynebacterium* spp., *Streptococcus* spp., and *Staphylococcus* spp. in the observed interaction network associated with sunscreen was hypothesized to be an example of how *Micrococcus luteus* may be able to influence

TABLE 1 Impact of solar radiation on a healthy microbiome.

Waveband	Wavelength	Dose	Study design	Subjects	Collection method	Analysis method	Impact of SR	Reference
UVB	308 nm	100, 150, 200, 250, 300, and 350 mJ/cm^2	Varying doses of UVB applied to the back Swab samples acquired prior to, immediately after, and 24 h after UV exposure	Male ($n=6$) Fitzpatrick phototype I/II	Swabbing	16S rRNA sequencing	Phylum level increase in *Cyanobacteria*, *Fusobacteria* and *Verrucomicrobia* Family level decreases in *Lactobacillaceae* and *Pseudomonadaceae* Family level increase in *Oxalobacteraceae*	Burns et al.[44]
UVA1	340–400 nm	22, 27, 33, 39, and 47 J/cm^2	Varying doses of UVB applied to the back Swab samples taken prior to, immediately after, and 24 h after UV exposure.	Male ($n=6$) Fitzpatrick skin types I-II	Swabbing	16S rRNA sequencing	Phylum level increase in *Cyanobacteria*, *Fusobacteria* and *Verrucomicrobia* Family level decreases in *Lactobacillaceae* and *Pseudomonadaceae* Family level increase in *Oxalobacteraceae*.	Burns et al.[44]
Solar-simulated radiation (UVA/UVB)	ND	2 MED	Single-blind clinical trial SPF 20 sunscreen and placebo applied to two test areas on the back of prior to UV irradiation Treated and untreated zones exposed to 2 MED of UVA/UVB Skin surface swabs collected 2 h after UV exposure	Female ($n=10$) Fitzpatrick skin type II–III Aged 20–45 years	Swabbing	16S rRNA sequencing	UV exposure led to higher abundance of *Cutibacterium* compared to *Lactobacillus* Application of SPF20 sunscreen prevented this relative change Placebo had no effect	Schuetz et al.[46]
Natural sunlight	NA	ND	Skin swabs taken from extensor forearm prior to (d0) taking a holiday in a sunny destination (minimum of 7 days duration) Swabbing repeated upon return at d1, d28 and d84	Male ($n=4$) Female ($n=17$) Healthy, white Northern European Mean age ± SD: 33.6 ± 6.4 years	Swabbing	16S rRNA sequencing	Reduction of *Proteobacteria* at 28 days post-holiday in subjects that actively sought sun No differences by D28 and D84 post-holiday	Willmott et al.[47]
Natural sunlight	NA	ND	Two groups: (1) Lifeguards, exposed yearly to direct sunlight and seawater throughout summer; (2) Ultraorthodox and always protected from direct sunlight Three skin sites were sampled: the inner forearm, dorsal forearm, and cheek.	Male	Swabbing	16S rRNA sequencing	Microbiomes of both groups generally similar Significant differences in microbial composition between lifeguards and ultraorthodox detected in sun exposed sites (cheek, outer and inner arms) No significant seasonal difference in the microbiome within the ultraorthodox group	Harel et al.[48]
Natural sunlight	NA	ND	Lifeguards exposed to SR throughout the summer Swabs collected at the beginning (May) and end (September) of summer	Lifeguards ($n=10$)	Swabbing	16S rRNA sequencing	Increase in the abundance of *Sphingomonas* and *Erythrobacteraceae* after the summer	Harel et al.[48]

TABLE 2 Pro- and postbiotics for photoprotection—An overview of all studies performed with pro- or postbiotics.

Route of administration	Probiotic/Postbiotic	Species	Model	Stress	Photoprotective effects	References
Topical	6-N-hydroxyaminopurine-producing *Staphylococcus epidermidis*	*Staphylococcus epidermidis*	SKH-1 mouse	UVB	Suppresses UVB-induced tumor formation.	Nakatsuji et al.[51]
Topical	Fermentable metabolite of *Zymomonas mobilis*	*Zymomonas mobilis*	SKH-1 hairless mouse	UVB	Restores TGF-β signaling Restores Procollagen I expression Reduces wrinkle formation	Tanaka et al.[81]
Topical	*Limosilactobacillus fermentum* XJC60	*Limosilactobacillus fermentum*	Guinea pig	UVB	Prevents skin damage Reduces abnormal keratinization, hyperplasia, and edema Reduces MMP-1 expression Reduces inflammatory cell infiltration	Chen et al.[82]
Topical	*Staphylococcus epidermidis* (+ glycerol)	*Staphylococcus epidermidis*	ICR mouse	UVB	Reduces erythema and ulceration Reduces IL-6 levels Reduces hyperplasia	Keshari et al.[83]
Topical	*Staphylococcus epidermidis* ATCC 12228	*Staphylococcus epidermidis*	ICR mice	UVB	Reduces lipid peroxidation Reduces DNA damage	Balasubramaniam et al.[84]
Topical	Yeast	*Saccharomyces cerevisiae*	BALB/c mice	UVB	Reduces skin damage Reduces inflammatory cell infiltration	Lu et al.[85]
Topical	*Lactobacillus reuteri* DSM 17938	*Lactobacillus reuteri*	Reconstructed Human Epidermis (EpiDerm™; EPI-200, Mattek)	UVB	Reduces inflammatory cytokine production	Khmaladze et al.[86]
Topical	*Lactobacillus reuteri* DSM 17938 lysate	*Lactobacillus reuteri*	Ex vivo human skin explant Reconstructed Human Epidermis (EpiDerm™; EPI-200, Mattek)	UVB UVB	Reduced inflammatory cytokine production Reduces inflammatory cytokine production	Khmaladze et al.[86] Khmaladze et al.[86]
Topical	*Vitreoscilla filiformis* extract	*Vitreoscilla filiformis*	Clinical study on back skin (Placebo-controlled; $n = 5$)	UVB	Reduces sunburn cell formation	Mahé et al.[87]
Oral	*Bifidobacterium breve*	*Bifidobacterium breve*	Hos:HR-1 hairless mice	UVB	Reduces erythema Reduces edema Reduces skin thickening Increases skin elasticity Reduces elastase expression Reduces inflammatory cytokine expression	Sugimoto et al.[88]
			Hos:HR-1 hairless mice	UVB	Reduces TEWL Increased hydration levels Reduces lipid and protein oxidation Reduces XO activity	Ishii et al.[89]
			Hos:HR-1 hairless mice	UVB	Reduces wrinkle formation Reduces erythema Reduces TEWL Increases hydration levels Reduces skin thickening Reduces histological damage Reduces inflammatory cytokine production	Satoh et al.[90]

TABLE 2 (Continued)

Route of administration	Probiotic/Postbiotic	Species	Model	Stress	Photoprotective effects	References
Oral	*Bifidobacterium animalis* subs. *lactis* MG741	*Bifidobacterium animalis*	HR-1 hairless mice	UVB	Reduces wrinkle formation Reduces epidermal thickening Increases hydration Reduces TEWL Reduces MMP-3 expression Inhibits MAPK signaling Reduces inflammatory cytokine expression	Lee et al.[91]
Oral	*Bifidobacterium longum*	*Bifidobacterium longum*	SKH-1 hairless mice	UVB	Reduces TEWL Reduces erythema Reduces wrinkle formation	Hong et al.[92]
			SKH-1 hairless mice	UVB	Increases hydration Reduces wrinkle formation Reduces epidermal thickening Increases antioxidant enzyme levels Increases Type I collagen expression Reduces inflammatory cytokine expression Reduces MMP expression Inhibits MAPK and NK-κB signaling	Kim et al.[93]
Oral	*Bifidobacterium longum* + Galactooligosaccharide	*Bifidobacterium longum*	SKH-1 hairless mice	UVB	Reduces TEWL Reduces erythema	Hong et al.[92]
			SKH-1 hairless mice	UVB	Increases hydration Reduces wrinkle formation Reduces epidermal thickening Increases antioxidant enzyme levels Increases Type I collagen expression Reduces inflammatory cytokine expression Reduces MMP expression Inhibits MAPK and NF-κB signaling	Kim et al.[93]
Oral	Lactobacillus acidophilus	*Lactobacillus acidophilus*	HR-1 hairless mice	UVB	Reduces TEWL Reduces epidermal thickening Inhibits MMP expression Inhibits MAPK signaling	Im et al.[94]
			Hos:HR-1 hairless mice	UVB	Reduces skin thickening Increases collagen levels Increases skin hydration Reduces TEWL Inhibits MMP expression Increases antioxidant enzyme expression Inhibits inflammatory cytokine production Inhibits MAPK signaling	Im et al.[95]

(Continues)

TABLE 2 (Continued)

Route of administration	Probiotic/Postbiotic	Species	Model	Stress	Photoprotective effects	References
Oral	Lactobacillus johnsonii La1	*Lactobacillus johnsonii*	Skh:hr1 mice	ssUV	Reduces edema Prevents UV-induced immunosuppression	Guéniche et al.[96]
Oral	Lipoteic acid from *Lactobacillus rhamnosus* GG		Crl:SKH-1-hrBR hairless mice	UVB + UVA	Reduces epidermal thickening Prevents apoptosis Reduces inflammatory cell infiltration Prevents immunosuppression Delays tumor formation	Weill et al.[97]
			C57/BL6 mice	UVB	Prevents UV-induced immunosuppression	Friedrich et al.[98]
			Crl:SKH-1-hrBR hairless mice	UVB	Reduces tumor growth	Friedrich et al[98]
Oral	Lactobacillus plantarum HY7714	*Lactobacillus plantarum*	Hairless mice	UVB	Reduces epidermal thickening Reduces TEWL Increases hydration Increases ceramide levels	Ra et al.[99]
Oral	Lactobacillus johnsonii La1	*Lactobacillus johnsonii*	Clinical study on back skin (RCT; $n = 54$)	ssUV	Prevents UV-induced immunosuppression	Peguet-Navarro et al.[100]
			Clinical study ($n = 60$ with Polymorphic Light Eruption) (skin area n/a)	UVA1	Reduces PLE scores	Marini et al.[101]
Oral	Lactobacillus johnsonii La1 + Carotenoids	*Lactobacillus johnsonii*	Clinical study ($n = 16$) (skin area: buttocks)	ssUV	Prevents UV-induced decrease in Langerhans cells Reduces melanin content	Bouilly-Gauthier et al.[102]
Oral	Lactobacillus johnsonii La1 + Carotenoids	*Lactobacillus johnsonii*	Clinical study (RCT; $n = 43$) (skin area: upper back)	ssUV	Increases MED Reduces ΔE^*	Bouilly-Gauthier et al.[102]
Oral	Lactobacillus johnsonii La1 + Carotenoids	*Lactobacillus johnsonii*	Clinical study ($n = 80$) (skin area: whole body dermatological evaluation)	Natural sunlight	Prevents sunburn Prevents sun intolerances (benign summer light eruption; labial herpes) Prevents sunspots More homogenous pigmentation	Bouilly-Gauthier et al.[102]
Oral	Lactiplantibacillus plantarum PBS067 (DSM24937), Lacticaseibacillus rhamnosus LRH020 (PBS070, DSM25568), and Limosilactobacillus reuteri PBS072 (DSM25175)	*Lactiplantibacillus plantarum*, *Lacticaseibacillus rhamnosus*, *Limosilactobacillus reuteri*	SKH-1 hairless mice	UVB	Reduces TEWL Increases hydration levels Reduces erythema Reduces skin thickening Reduces wrinkle formation Inhibits MMP expression Increases Type I collagen expression Increases expression of hydration-related proteins Reduces inflammatory cytokine expression Increases antioxidant enzyme levels Reduces lipid peroxidation Reduces ROS levels Inhibits MAPK signaling	Seo et al.[103]

Abbreviations: IL-6, Interleukin 6; MAPK, Mitogen-activated protein kinase; MED, Minimal erythema dose.; MMP, Matrix metalloproteinase; NF-κB, Nuclear factor kappa-light-chain-enhancer of activated B cells; PLE, Polymorphic Light Eruption; ROS, Reactive oxygen species; ssUV, Solar-simulated UV; TEWL, Transepidermal water loss; TGF-β, Transforming growth factor beta; UVA, Ultraviolet A; UVB, Ultraviolet B; XO, Xanthine oxidase.

nearby organisms or even the host skin and their abilities to increase their resistance against UVR by sharing metabolites.

In a recently published study, new insights into the response of the skin microbiome to UVR, as well as the potential protective effects of a sun-protecting sunscreen on the microbiome of UV-irradiated skin of the volunteers' back were given.[46] The authors reported that the alpha diversity of the skin microbes was more modified by inter-individual differences and by the application of sunscreens rather than by SR. After UVR, the abundance of *Lactobacillus crispatus* was negatively impacted without any sun protection, while the application of sunscreen provided a protection against UVR. Moreover, the use of sunscreen helped to maintain an interaction network with Micrococcus genus. UVA and UVB impacted differently on the survival rates of *L. crispatus*, *Cutibacterium acnes*, and *Staphylococcus epidermidis*. These findings may help for a better understanding of the complex interaction between the skin microbiome and UVR, highlighting the importance of sun protection in maintaining a healthy skin ecosystem to avoid dysbiosis that may damage the skin. Significant interindividual differences in the microbial composition were observed in each subject.

DISCUSSION

The skin microbiome is permanently exposed to external aggressors including SR. Genetic predisposition plays a significant role in determining individual susceptibility to solar radiation-induced dysbiosis and skin conditions. Impacting the host's microbiome results in modifications of the microbiota composition and of the host immunity including both innate and acquired immunity.[7,8,42,105]

While evidence of the impact of SR on the skin as an organ is abundant, to date there are only few data available about the impact of SR on the skin microbiome. These data do yet not allow to confirm the hypothesis that SR components impact not only directly the immune responses of the body but also indirectly through modifications of the skin microbiome.[35] While the beneficial and deleterious impact of SR components on inflammatory skin diseases such as atopic dermatitis, psoriasis, and acne has been well described the microbiota may also play a role in the host's immune response during or following exposure to SR.[13,42,44,106–118]

Investigating the global metabolic profile of the skin regarding the microbiome and UVR, as well as together with other environmental factors such as pollution or climate change, provided interesting and new insights into the dynamics and interactions between the skin metabolome, microbiome, and UVR creating new axes for the development of metabolite- or lipid-based claims to maintain skin health.[119]

While, again, there is no direct evidence that UVR has a deleterious impact on the skin microbiome and through this impact causes or worsens existing skin conditions, there is evidence that UVR destroys substances such as porphyrins which are absorbed in the long UVA range and especially in the visible range and which are produced by several microorganisms including the skin commensal *Cutibacterium acnes* and *Pseudomona aeruginosa*. Thus, UVR may impact on the skin microbiome composition leading to dysbiosis and an altered skin barrier.[120,121]

Sun protective means exist to shield the body, and especially the skin, against UVR. These products were developed to protect the skin as an organ in itself but without considering their effect on its commensal inhabitants. Therefore, protecting the exposed areas of the skin and its commensal inhabitants may help to further protect the host and maintain a balanced skin microbiome. Novel sun-protecting products containing pre and probiotics and other beneficial ingredients may potentially help to protect the skin microbiota from SR damages and help to limit the exposome-induced immunosuppression of the host.[74,76–80]

In addition to topical sunscreens, the use of oral photoprotective supplements rich in antioxidants such as *Polypodium leucotomos* extract and green tea may play a crucial role in mitigating the dysbiosis induced by solar radiation exposure, thus maintaining a healthier skin microbiome.[122]

In 2021, Souak et al.[74] reported that the skin microbiota may be a source of compounds with indirect photoprotective properties and several commensal bacteria are able to block UV radiation or absorb its deleterious impact, while others have anti-inflammatory and antioxidative properties. Clinical investigations have supported the concept that probiotics may be beneficial in preventing or reversing the negative impact of UVR on the body.[35] Other work supported this idea of potential benefits of novel therapeutic strategies using pro- and pre-biotics to modulate the skin response to UVR including not only the protection of the host but also to enhance the therapeutic effects of UVR in inflammatory skin conditions.[123]

While this concept seems appealing, currently only sparse information is available that shows that modulating the skin microbiota in supplying such care helps to reduce immunosuppression and strengthen the skin microbiome against SR.

In conclusion, safeguarding both the skin and its microbiota can help reduce the risk of dysbiosis caused by solar radiation (SR), thereby supporting skin defense

mechanisms and lowering the likelihood of SR-related skin conditions. Furthermore, the incorporation of topical sunscreens with film-forming properties as a vehicle not only offers effective sun protection but also enhances the skin barrier, potentially aiding in the preservation of a healthy microbiome by reducing the penetration of harmful UV radiation and environmental stressors. Among our recommendations, it is imperative to conduct more studies to comprehend the influence of solar radiation and its individual component wavebands on the skin microbiome. Also, more studies about the potential long-term consequences of solar radiation exposure on the skin microbiome and the implications for chronic skin conditions and aging. Additionally, there is a need for further investigations assessing the impact of sunscreens on the skin microbiome. Moreover, more high-quality clinical studies are needed to explore the use of oral and topical pro- and postbiotics and other therapeutic interventions targeting the skin microbiome to mitigate the effects of SR. Additional research is necessary to validate this emerging concept of microbiota protection against solar radiation.

AUTHOR CONTRIBUTIONS

All authors participated in the discussion, literature review, and read and approved the manuscript.

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DATA AVAILABILITY STATEMENT

The data that support this work are available from the corresponding author upon reasonable request.

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REFERENCES

1. Belkaid Y, Segre JA. Dialogue between skin microbiota and immunity. *Science (New York, NY)*. 2014;346(6212):954-959.
2. Lee H-J, Kim M. Skin barrier function and the microbiome. *Int J Mol Sci*. 2022;23(21):13071.
3. Gill SR, Pop M, Deboy RT, et al. Metagenomic analysis of the human distal gut microbiome. *Science*. 2006;312:1355-1359.
4. Grice EA, Segre JA. The skin microbiome. *Nat Rev Microbiol*. 2011;9:244-253.
5. Ratanapokasatit Y, Laisuan W, Rattananukrom T, Petchlorlian A, Thaipisuttikul I, Sompornrattanaphan M. How microbiomes affect skin aging: the updated evidence and current perspectives. *Life (Basel, Switzerland)*. 2022;12:936.
6. Baldwin HE, Bhatia ND, Friedman A, Eng RM, Seite S. The role of cutaneous microbiota harmony in maintaining a functional skin barrier. *J Drugs Dermatol*. 2017;16(1):12-18.
7. Balato A, Cacciapuoti S, Di Caprio R, et al. Human microbiome: composition and role in inflammatory skin diseases. *Arch Immunol Ther Exp*. 2019;6:1-18.
8. Belizário JE, Napolitano M. Human microbiomes and their roles in dysbiosis, common diseases, and novel therapeutic approaches. *Front Microbiol*. 2015;6:1050.
9. Bhatt AP, Redinbo MR, Bultman SJ. The role of the microbiome in cancer development and therapy. *CA Cancer J Clin*. 2017;67:326-344.
10. Dominguez-Bello MG, Godoy-Vitorino F, Knight R, Blaser MJ. Role of the microbiome in human development. *Gut*. 2019;68:1108-1114.
11. Chambers ES, Vukmanovic-Stejic M. Skin barrier immunity and ageing. *Skin Barrier Immunity and Ageing Immunology*. 2020;160:116-125.
12. Vrijheid M. The exposome: a new paradigm to study the impact of environment on health. *Thorax*. 2014;69:876-878.
13. Agache I, Miller R, Gern JE, et al. Emerging concepts and challenges in implementing the exposome paradigm in allergic diseases and asthma: a Practall document. *Allergy*. 2019;74:449-463.
14. Vineis P, Robinson O, Chadeau-Hyam M, Dehghan A, Mudway I, Dagnino S. What is new in the exposome? *Environ Int*. 2020;143:105887.
15. Buck Louis GM, Sundaram R. Exposome: time for transformative research. *Stat Med*. 2012;31:2569-2575.
16. Miller GW, Jones DP. The nature of nurture: refining the definition of the exposome. *Tocicol Sci*. 2014;137:1-2.
17. Wild CP. The exposome: from concept to utility. *Int J Epidemiol*. 2012;41:24-32.
18. Kim BE, Leung DYM. Significance of skin barrier dysfunction in atopic dermatitis. *Allergy, Asthma Immunol Res*. 2018;10:207-215.
19. Kobayashi T, Glatz M, Horiuchi K, et al. Dysbiosis and *Staphylococcus aureus* colonization drives inflammation in atopic dermatitis. *Immunity*. 2015;42:756-766.
20. McDonald D, Ackermann G, Khailova L, et al. Extreme dysbiosis of the microbiome in critical illness. *mSphere*. 2016;1:e00199-e00206.
21. Lai A, Owens K, Patel S, Nicholas M. The impact of air pollution on atopic dermatitis. *Curr Allergy Asthma Rep*. 2023;23:435-442.
22. Dréno B, Dagnelie MA, Khammari A, Corvec S. The skin microbiome: a new actor in inflammatory acne. *Am J Clin Dermatol*. 2020;21(Suppl 1):1-24.
23. Celebi Sozener Z, Özbey Yücel Ü, Altiner S, et al. The external exposome and allergies: from the perspective of the epithelial barrier hypothesis. *Front Allergy*. 2022;3:887672.

24. Marson J, Bhatia N, Graber E, et al. Supplement Article: The role of epidermal barrier dysfunction and cutaneous microbiome dysbiosis in the pathogenesis and manageme.
25. Edslev SM, Agner T, Andersen PS. Skin microbiome in atopic dermatitis. *Acta Derm Venereol*. 2020;100:adv.
26. Woo YR, Cho SH, Lee JD, Kim HS. The human microbiota and skin cancer. *Int J Mol Sci*. 2022;23(3):1813.
27. Mekadim C, Skalnikova HK, Cizkova J, et al. Dysbiosis of skin microbiome and gut microbiome in melanoma progression. *BMC Microbiol*. 2022;22:63.
28. Bensadoun RJ, Humbert P, Krutman J, et al. Daily baseline skin care in the prevention, treatment, and supportive care of skin toxicity in oncology patients: recommendations from a multinational expert panel. *Cancer Manag Res*. 2013;5:401-408.
29. Krutmann J. Ultraviolet a radiation-induced biological effects in human skin: relevance for photoaging and photodermatosis. *J Dermatol Sci*. 2000;23(Suppl 1):S22-S26.
30. Krutmann J, Bouloc A, Sore G, Bernard BA, Passeron T. The skin aging exposome. *J Dermatol Sci*. 2017;85:152-161.
31. Krutmann J, Granger C, Trullàs C, Passeron T. Daily photoprotection: what does it really mean? *Photodermatol Photoimmunol Photomed*. 2021;37:569-570.
32. Krutmann J, Morita A, Chung JH. Sun exposure: what molecular photodermatology tells us about its good and bad sides. *J Invest Dermatol*. 2012;132:976-984.
33. Krutmann J, Passeron T, Gilaberte Y, et al. Photoprotection of the future: challenges and opportunities. *J Eur Acad Dermatol Venereol*. 2020;34:447-454.
34. Liebert A, Bicknell B, Johnstone DM, Gordon LC, Kiat H, Hamblin MR. "Photobiomics": can light, including Photobiomodulation, Alter the microbiome? *Photobiomodul Photomed Laser Surg*. 2019;37:681-693.
35. Rai S, Rai G, Kumar A. Eco-evolutionary impact of ultraviolet radiation (UVR) exposure on microorganisms, with a special focus on our skin microbiome. *Microbiol Res*. 2022;260:127044.
36. Lautenschlager S, Wulf HC, Pittelkow MR. Photoprotection. *Photoprotection Lancet*. 2007;370:528-537.
37. Garnacho Saucedo GM, Salido Vallejo R, Moreno Giménez JC. Effects of solar radiation and an update on photoprotection. *An Pediatr (Engl Ed)*. 2020;92:377.e1-e9.
38. Li H, Colantonio S, Dawson A, Lin X, Beecker J. Sunscreen application, safety, and Sun protection: the evidence. *J Cutan Med Surg*. 2019;23:357-369.
39. Kurz B, Berneburg M, Singer S. Sonnenschutz der menschlichen Haut: Grundlagen. *Hautarzt*. 2022;73:251-256.
40. Guan LL, Lim HW, Mohammad TF. Sunscreens and photoaging: a review of current literature. *Am J Clin Dermatol*. 2021;22:819-828.
41. Searle T, Al-Niaimi F, Ali FR. Visible light and hyperpigmentation: the invisible culprit. *Clin Exp Dermatol*. 2021;46:995-997.
42. Patra V, Byrne SN, Wolf P. The skin microbiome: is it affected by UV-induced immune suppression? *Front Microbiol*. 2016;7:1235.
43. Patra V, Wagner K, Arulampalam V, Wolf P. Skin microbiome modulates the effect of ultraviolet radiation on cellular response and immune function. *iScience*. 2019;15:2112-2122.
44. Burns EM, Ahmed H, Isedeh PN, et al. Ultraviolet radiation, both UVA and UVB, influences the composition of the skin microbiome. *Exp Dermatol*. 2019;28:136-141.
45. Fernández RO, Pizarro RA. Lethal effect induced in *Pseudomonas aeruginosa* exposed to ultraviolet-a radiation. *Photochem Photobiol*. 1996;64:334-339.
46. Schuetz R, Claypool J, Sfriso R, Vollhardt JH. Sunscreens can preserve human skin microbiome upon erythemal UV exposure. *Int J Cosmet Sci*. 2024;46(1):71-84.
47. Willmott T, Campbell PM, Griffiths CEM, et al. Behaviour and sun exposure in holidaymakers alters skin microbiota composition and diversity. *Front Aging*. 2023;4:1217635.
48. Harel N, Reshef L, Biran D, Brenner S, Ron EZ, Gophna U. Effect of solar radiation on skin microbiome: study of Two populations. *Microorganisms*. 2022;10:1523.
49. Smith M, O'Neill C, Dickinson M, Chavan B, McBain A. Exploring associations between skin, the dermal microbiome, and ultraviolet radiation: advancing possibilities for next-generation sunscreens. *Front Microbiomes*. 2023;2:1-11.
50. Kong HH, Segre JA. Skin microbiome: looking back to move forward. *J Invest Dermatol*. 2012;132:933-939.
51. Nakatsuji T, Chen TH, Butcher AM, et al. A commensal strain of *Staphylococcus epidermidis* protects against skin neoplasia. *Sci Adv*. 2018;4:eaao4502.
52. Balasubramaniam A, Adi P, Do Thi TM, Yang JH, Labibah AS, Huang CM. Skin bacteria mediate glycerol fermentation to produce electricity and resist UV-B. *Microorganisms*. 2020;8:1092.
53. Shiota S, Nakayama H. UV endonuclease of *Micrococcus luteus*, a cyclobutane pyrimidine dimer-DNA glycosylase/abasic lyase: cloning and characterization of the gene. *Proc Natl Acad Sci USA*. 1997;94:593-598.
54. Mohana DC, Thippeswamy S, Abhishek RU. Antioxidant, antibacterial, and ultraviolet-protective properties of carotenoids isolated from *Micrococcus* spp. *Rad Protect Env*. 2013;36:168.
55. Biragyn A, Ruffini PA, Leifer CA, et al. Toll-like receptor 4-dependent activation of dendritic cells by beta-defensin 2. *Science*. 2002;298:1025-1029.
56. Chadebech P, Goidin D, Jacquet C, Viac J, Schmitt D, Staquet MJ. Use of human reconstructed epidermis to analyze the regulation of beta-defensin hBD-1, hBD-2, and hBD-3 expression in response to LPS. *Cell Biol Toxicol*. 2003;19:313-324.
57. Niyonsaba F, Ushio H, Nakano N, et al. Antimicrobial peptides human beta-defensins stimulate epidermal keratinocyte migration, proliferation and production of proinflammatory cytokines and chemokines. *J Invest Dermatol*. 2007;127:594-604.
58. Kolls JK, McCray PB Jr, Chan YR. Cytokine-mediated regulation of antimicrobial proteins. *Nat Rev Immunol*. 2008;8:829-835.
59. Chapman RS, Cooper KD, De Fabo EC, et al. Solar ultraviolet radiation and the risk of infectious disease: summary of a workshop. *Photochem Photobiol*. 1995;61:223-247.
60. Meisel JS, Sfyroera G, Bartow-McKenney C, et al. Commensal microbiota modulate gene expression in the skin. *Microbiome*. 2018;6:20.
61. Williams MR, Costa SK, Zaramela LS, et al. Quorum sensing between bacterial species on the skin protects against epidermal injury in atopic dermatitis. *Sci Transl Med*. 2019;11:eaat8329.
62. Boxberger M, Cenizo V, Cassir N, La Scola B. Challenges in exploring and manipulating the human skin microbiome. *Microbiome*. 2021;9:125.
63. Nakatsuji T, Chen TH, Narala S, et al. Antimicrobials from human skin commensal bacteria protect against Staphylococcus

aureus and are deficient in atopic dermatitis. *Sci Transl Med*. 2017;9:eaah4680.
64. Canesso MC, Vieira AT, Castro TB, et al. Skin wound healing is accelerated and scarless in the absence of commensal microbiota. *J Immunol*. 2014;193:5171-5180.
65. Lai Y, Di Nardo A, Nakatsuji T, et al. Commensal bacteria regulate toll-like receptor 3-dependent inflammation after skin injury. *Nat Med*. 2009;15:1377-1382.
66. Harrison OJ, Linehan JL, Shih HY, et al. Commensal-specific T cell plasticity promotes rapid tissue adaptation to injury. *Science*. 2019;363:eaat6280.
67. Gläser R, Navid F, Schuller W, et al. UV-B radiation induces the expression of antimicrobial peptides in human keratinocytes in vitro and in vivo. *J Allergy Clin Immunol*. 2009;123:1117-1123.
68. Gallo RL, Hooper LV. Epithelial antimicrobial defence of the skin and intestine. *Nat Rev Immunol*. 2012;12:503-516.
69. Faergemann J, Larko O. The effect of UV-light on human skin microorganisms. *Acta Derm Venereol*. 1987;67:69-72.
70. Kullander J, Forslund O, Dillner J. Staphylococcus aureus and squamous cell carcinoma of the skin. *Cancer Epidemiol Biomarkers Prevent*. 2009;18:472-478.
71. Wood DLA, Lachner N, Tan JM, et al. A natural history of actinic keratosis and cutaneous squamous cell carcinoma microbiomes. *MBio*. 2018;9:e01432-18.
72. Madhusudhan N, Pausan MR, Halwachs B, et al. Molecular profiling of keratinocyte skin tumors links Staphylococcus aureus overabundance and increased human β-Defensin-2 expression to growth promotion of squamous cell carcinoma. *Cancer (Basel)*. 2020;12:541.
73. Young AR, Claveau J, Rossi AB. Ultraviolet radiation and the skin: photobiology and sunscreen photoprotection. *J Am Acad Dermatol*. 2017;76:S100-S109.
74. Souak D, Barreau M, Courtois A, et al. Challenging cosmetic innovation: the Skin microbiota and probiotics protect the skin from UV-induced damage. *Microorganisms*. 2021;9:936.
75. Teng Y, Huang Y, Danfeng X, Tao X, Fan Y. The role of probiotics in skin photoaging and related mechanisms: a review. *Clin Cosmet Investig Dermatol*. 2022;15:2455-2464.
76. Habeebuddin M, Karnati RK, Shiroorkar PN, et al. Topical probiotics: more than a skin deep. *Pharmaceutics*. 2022;14:557.
77. Valdéz JC, Peral MC, Rachid M, Santana M, Perdigón G. Interference of lactobacillus plantarum with Pseudomonas aeruginosa in vitro and in infected burns: the potential use of probiotics in wound treatment. *Clin Microbiol Infect*. 2005;11:472-479.
78. Gueniche A, Philippe D, Bastien P, Blum S, Buyukpamukcu E, Castiel-Higounenc I. Probiotics for photoprotection. *Dermato Endocrinol*. 2009;1:275-279.
79. Baquerizo Nole KL, Yim E, Keri JE. Probiotics and prebiotics in dermatology. *J Am Acad Dermatol*. 2014;71:814-821.
80. Shaheen AE, Gebreel HM, Moussa LA, Zakaria AE, Nemr WA. Photoprotection against UV-induced skin damage using hyaluronic acid produced by *Lactiplantibacillus plantarum* and *Enterococcus durans*. *Curr Microbiol*. 2023;80:262.
81. Tanaka H, Yamaba H, Kosugi N, Mizutani H, Nakata S. Fermentable metabolite of Zymomonas mobilis controls collagen reduction in photoaging skin by improving TGF-beta/Smad signaling suppression. *Arch Dermatol Res*. 2008;300:S57-S64.
82. Chen H, Li Y, Xie X, et al. Exploration of the molecular mechanisms underlying the anti-photoaging effect of *Limosilactobacillus fermentum* XJC60. *Front Cell Infect Microbiol*. 2022;12:838060.
83. Keshari S, Balasubramaniam A, Myagmardoloonjin B, Herr DR, Negari IP, Huang CM. Butyric acid from probiotic Staphylococcus epidermidis in the skin microbiome Down-regulates the ultraviolet-induced pro-inflammatory IL-6 cytokine via short-chain fatty acid receptor. *Int J Mol Sci*. 2019;20:4477.
84. Balasubramaniam A, Adi P, Tra My DT, et al. Repurposing INCI-registered compounds as skin prebiotics for probiotic *Staphylococcus epidermidis* against UV-B. *Sci Rep*. 2020;10:21585.
85. Lu Y, Liu M, Cao Y, et al. Hydrogel sunscreen based on yeast /gelatin demonstrates excellent UV-shielding and skin protection performance. *Colloids Surf B Biointerfaces*. 2021;205:111885.
86. Khmaladze I, Butler É, Fabre S, Gillbro JM. *Lactobacillus reuteri* DSM 17938-a comparative study on the effect of probiotics and lysates on human skin. *Exp Dermatol*. 2019;28:822-828.
87. Mahé YF, Martin R, Aubert L, et al. Induction of the skin endogenous protective mitochondrial MnSOD by *Vitreoscilla filiformis* extract. *Int J Cosmet Sci*. 2006;28:277-287.
88. Sugimoto S, Ishii Y, Izawa N, et al. Photoprotective effects of Bifidobacterium breve supplementation against skin damage induced by ultraviolet irradiation in hairless mice. *Photodermatol Photoimmunol Photomed*. 2012;28:312-319.
89. Ishii Y, Sugimoto S, Izawa N, Sone T, Chiba K, Miyazaki K. Oral administration of *Bifidobacterium breve* attenuates UV-induced barrier perturbation and oxidative stress in hairless mice skin. *Arch Dermatol Res*. 2014;306:467-473.
90. Satoh T, Murata M, Iwabuchi N, et al. Effect of *Bifidobacterium breve* B-3 on skin photoaging induced by chronic UV irradiation in mice. *Benefic Microbes*. 2015;6:497-504.
91. Lee JY, Park JY, Kim Y, Kang CH. Protective effect of *Bifidobacterium animalis* subs. Lactis MG741 as probiotics against UVB-exposed fibroblasts and hairless mice. *Microorganisms*. 2022;10:2343.
92. Hong KB, Jeong M, Han KS, Hwan Kim J, Park Y, Suh HJ. Photoprotective effects of galacto-oligosaccharide and/or *Bifidobacterium longum* supplementation against skin damage induced by ultraviolet irradiation in hairless mice. *Int J Food Sci Nutr*. 2015;68:923-930.
93. Kim D, Lee KR, Kim NR, Park SJ, Lee M, Kim OK. Combination of *Bifidobacterium longum* and Galacto-oligosaccharide protects the skin from photoaging. *J Med Food*. 2021;24:606-616.
94. Im AR, Kim HS, Hyun JW, Chae S. Potential for tyndalized lactobacillus acidophilus as an effective component in moisturizing skin and anti-wrinkle products. *Exp Ther Med*. 2016;12:759-764.
95. Im AR, Lee B, Kang DJ, Chae S. Skin moisturizing and antiphotodamage effects of *Tyndallized lactobacillus* acidophilus IDCC 3302. *J Med Food*. 2018;21:1016-1023.
96. Gueniche A, Benyacoub J, Buetler TM, Smola H, Blum S. Supplementation with oral probiotic bacteria maintains cutaneous immune homeostasis after UV exposure. *Eur J Dermatol*. 2006;16:511-517.
97. Weill FS, Cela EM, Paz ML, Ferrari A, Leoni J, González Maglio DH. Lipoteichoic acid from *Lactobacillus rhamnosus* GG as an oral photoprotective agent against UV-induced carcinogenesis. *Br J Nutr*. 2013;109:457-466.
98. Friedrich AD, Campo VE, Cela EM, et al. Oral administration of lipoteichoic acid from *Lactobacillus rhamnosus* GG overcomes UVB-induced immunosuppression and impairs skin tumor growth in mice. *Eur J Immunol*. 2019;49:2095-2102.

99. Ra J, Lee DE, Kim SH, et al. Effect of oral administration of *Lactobacillus plantarum* HY7714 on epidermal hydration in ultraviolet B-irradiated hairless mice. *J Microbiol Biotechnol*. 2014;24:1736-1743.
100. Peguet-Navarro J, Dezutter-Dambuyant C, Buetler T, et al. Supplementation with oral probiotic bacteria protects human cutaneous immune homeostasis after UV exposure-double blind, randomized, placebo controlled clinical trial. *Eur J Dermatol*. 2008;18:504-511.
101. Marini A, Jaenicke T, Grether-Beck S, et al. Prevention of polymorphic light eruption by oral administration of a nutritional supplement containing lycopene, β-carotene, and *Lactobacillus johnsonii*: results from a randomized, placebo-controlled, double-blinded study. *Photodermatol Photoimmunol Photomed*. 2014;30:189-194.
102. Bouilly-Gauthier D, Jeannes C, Maubert Y, et al. Clinical evidence of benefits of a dietary supplement containing probiotic and carotenoids on ultraviolet-induced skin damage. *Br J Dermatol*. 2010;163:536-543.
103. Seo E, Song HH, Kim H, et al. Oral Administration of Mixed Probiotics Improves Photoaging by modulating the Cecal microbiome and MAPK pathway in UVB-irradiated hairless mice. *Mol Nutr Food Res*. 2023;67:e2200841.
104. Tao K, Noda A, Yonei S. The roles of different excision-repair mechanisms in the resistance of *Micrococcus luteus* to UV and chemical mutagens. *Mutat Res*. 1987;183:231-239.
105. Lossius AH, Sundnes O, Ingham AC, et al. Shifts in the skin microbiota after UVB treatment in adult atopic dermatitis. *Dermatology*. 2022;238:109-120.
106. Appenzeller BMR, Chadeau-Hyam M, Aguilar L. Skin exposome science in practice: current evidence on hair biomonitoring and future perspectives. *J Eur Acad Dermatol Venereol*. 2020;34(Suppl 4):26-30.
107. Cecchi L, D'Amato G, Annesi-Maesano I. External exposome and allergic respiratory and skin diseases. *J Allergy Clin Immunol*. 2018;141:846-857.
108. Celebi Sözener Z, Cevhertas L, Nadeau K, Akdis M, Akdis CA. Environmental factors in epithelial barrier dysfunction. *J Allergy Clin Immunol*. 2020;145:1517-1528.
109. Dreno B, Bettoli V, Araviiskaia E, Sanchez Viera M, Bouloc A. The influence of exposome on acne. *J Eur Acad Dermatol Venereol*. 2018;32:812-819.
110. Dreno B, Shourick J, Kerob D, Bouloc A, Taïeb C. The role of exposome in acne: results from an international patient survey. *J Eur Acad Dermatol Venereol*. 2020;34:1057-1064.
111. Molina-García M, Malvehy J, Granger C, Garre A, Trullàs C, Puig S. Exposome and skin. Part 2. The influential role of the exposome, beyond UVR, in actinic keratosis, Bowen's disease and squamous cell carcinoma: a proposal. *Dermatol Ther (Heidelb)*. 2022;12:361-380.
112. Morgado-Carrasco D, Granger C, Trullas C, Piquero-Casals J. Impact of ultraviolet radiation and exposome on rosacea: key role of photoprotection in optimizing treatment. *J Cosmet Dermatol*. 2021;20:3415-3421.
113. Passeron T, Krutmann J, Andersen ML, Katta R, Zouboulis CC. Clinical and biological impact of the exposome on the skin. *J Eur Acad Dermatol Venereol*. 2020;34(Suppl 4):4-25.
114. Passeron T, Zouboulis CC, Tan J, et al. Adult skin acute stress responses to short-term environmental and internal aggression from exposome factors. *J Eur Acad Dermatol Venereol*. 2021;35:1963-1975.
115. Piquero-Casals J, Morgado-Carrasco D, Rozas-Muñoz E, et al. Sun exposure, a relevant exposome factor in acne patients and how photoprotection can improve outcomes. *J Cosmet Dermatol*. 2023;22:1919-1928.
116. Stefanovic N, Flohr C, Irvine AD. The exposome in atopic dermatitis. *Allergy*. 2020;75(1):63-74.
117. Assarsson M, Duvetorp A, Dienus O, Söderman J, Seifert O. Significant changes in the skin microbiome in patients with chronic plaque psoriasis after treatment with narrowband ultraviolet B. *Acta Derm Venereol*. 2018;98(4):428-436.
118. Reid C, Griffiths CEM. Psoriasis and treatment: past, present and future aspects. *Acta Derm Venereol*. 2020;100(3):70-80.
119. Patra V, Bordag N, Clement Y, et al. Ultraviolet exposure regulates skin metabolome based on the microbiome. *Sci Rep*. 2023;13:7207.
120. Amin RM, Bhayana B, Hamblin MR, Dai T. Antimicrobial blue light inactivation of Pseudomonas aeruginosa by photoexcitation of endogenous porphyrins: in vitro and in vivo studies. *Lasers Surg Med*. 2016;48:562-568.
121. Wang Y, Zhu W, Shu M, et al. The response of human skin commensal bacteria as a reflection of UV radiation: UV-B decreases porphyrin production. *PLoS One*. 2012;7:e47798.
122. Torres AE, Luk KM, Lim HW. Botanicals for photoprotection. *Plast Aesthet Res*. 2020;7:57.
123. Patra V, Gallais Sérézal I, Wolf P. Potential of skin microbiome, pro- and/or pre-biotics to affect local cutaneous responses to UV exposure. *Nutrients*. 2020;12:1795.

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