Clinical, Cosmetic and Investigational Dermatology

Dovepress
Taylor & Francis Group

Open Access Full Text Article

REVIEW

Mechanisms and Therapeutic Roles of Medicinal Plants in Skin Photoaging

Xinxin Lei

Department of Dermatology, Hangzhou Third People's Hospital, Hangzhou Third Hospital Affiliated to Zhejiang Chinese Medical University, Hangzhou, 310009, People's Republic of China

Correspondence: Xinxin Lei, Department of Dermatology, Hangzhou Third People's Hospital, Hangzhou Third Hospital Affiliated to Zhejiang Chinese Medical University, West Lake Road38, Hangzhou, 310009, People's Republic of China, Tel +86-15273121767, Email leixinxin2025@163.com

Abstract: Photoaging refers to the cumulative skin damage primarily caused by exposure to ultraviolet (UV) radiation. This process results in the harmful effects of UV irradiation on skin cells, leading to alterations in the extracellular matrix, which consequently impacts the appearance and function of the skin. This review aims to elucidate how bioactive compounds from medicinal plants counteract UV-induced damage, as identified by current mechanistic and molecular studies. Over the past few decades, medicinal plants have garnered increasing attention for their potential therapeutic benefits in treating various human diseases. Numerous studies have explored the anti-photoaging properties of herbal remedies, revealing that various active compounds, extracts, and combinations of medicinal plants can mitigate photoaging in different skin cells through diverse signaling pathways. Compared to synthetic agents, herbal remedies offer lower toxicity profiles and are often perceived as safer alternatives, making them attractive options for long-term skin care and photoaging prevention. These findings suggest that herbal plants can reduce UV-induced skin damage primarily by inhibiting the production of reactive oxygen species (ROS), matrix metalloproteinases (MMPs), and inflammatory mediators, while simultaneously promoting collagen synthesis. The underlying mechanisms of these effects are associated with key cellular pathways, including MAPK, NF-κB, Nrf2, and TGF-β/Smad. These findings suggest that herbal plants can reduce UV-induced skin damage by inhibiting reactive oxygen species (ROS), matrix metalloproteinases (MMPs), and inflammatory mediators, while promoting collagen synthesis. This review provides novel insight into cell-specific mechanisms by which medicinal plants mitigate photoaging, laying the groundwork for their potential therapeutic application.

Keywords: photoaging, medicinal plants, herbs, matrix metalloproteinases, MMPs, reactive oxygen species, ROS, collagen

Introduction

Skin aging has become a significant challenge in contemporary society. It comprises two distinct categories: intrinsic and extrinsic aging. Intrinsic aging, also referred to as chronological aging, occurs as a result of a natural series of physiological processes.[1] In contrast, extrinsic aging is influenced by external factors such as exposure to sunlight, smoking, chemical agents, stress, and pollution.[2] Ultraviolet (UV) radiation from sunlight is the most critical factor in photoaging. UV radiation can be classified into three types: UVA (320–400 nm), UVB (290–320 nm), and UVC (100–290 nm). Only UVA and UVB reach the Earth's surface and cause damage to human health.[3]

According to the updated UNEP Environmental Effects Assessment Panel report, UV radiation continues to pose major public health risks, particularly in the context of skin photoaging. Photoaging is a chronic skin condition caused by cumulative UV exposure, which significantly contributes to premature aging phenotypes.[4] Clinically, photoaged skin is characterized by coarse, lax wrinkles, irregular pigmentation, depigmentation, rough texture, telangiectasia, and atrophy. Histological alterations in photodamaged skin include increased epidermal thickness, reduced collagen content, fragmentation of normal elastic fibers, excessive deposition of aberrant elastic fibers, and increased glycosaminoglycans.[5,6] Moreover, photoaging can stimulate the proliferation of fibroblasts and inflammatory cells. In mildly damaged skin, the vascular walls thicken; in severely damaged skin, vasodilation and vessel wall thinning occur.[7] The mechanism of skin damage is closely related to its optical properties. While part of UV radiation is reflected at the skin surface, a significant

Received: 4 May 2025
Accepted: 28 June 2025
Published: 11 July 2025

portion is absorbed or transmitted, with UVA penetrating into the dermis and UVB primarily affecting the epidermis, thereby triggering oxidative stress and DNA damage.

The mechanisms underlying cutaneous photoaging involve complex biochemical processes that primarily affect the connective tissue of the skin. UV radiation (UVR) can directly damage DNA, and it can also induce excessive production of reactive oxygen species (ROS), leading to secondary damage to DNA, membranes, and proteins, or triggering complex molecular pathways. These pathways involve cell surface receptors, signal transduction cascades, transcription factors, and enzymes such as collagenase, elastase, and hyaluronidase.[8] This cascade of events results in further DNA damage, extracellular matrix (ECM) degradation, inflammation, apoptosis, melanogenesis disorders, vascular damage, and immune dysfunction, affecting various skin cell types, including keratinocytes, fibroblasts, melanocytes, endothelial cells, and Langerhans cells.

Currently, there are several approaches for addressing photodamaged skin, such as the use of retinoids, antioxidants, DNA repair enzymes, α-hydroxy acids, and hormones. For instance, retinoids regulate cellular proliferation and differentiation, prevent collagen loss, and stimulate collagen synthesis in sun-exposed skin. Antioxidants, including vitamins C and E and procyanidins, mitigate oxidative damage to the skin by neutralizing free radicals and converting them into less reactive molecules.[7] However, chemical-based treatments can lead to various adverse effects, such as allergic contact dermatitis, irritant contact dermatitis, and phototoxicity.[9] Therefore, there is an urgent need to identify safe and effective natural alternatives with fewer toxic side effects.

The use of medicinal herbs dates back thousands of years. Today, many cosmetic products and functional foods in Asian countries contain herbal plants and their metabolites. These products are often perceived as safe and therapeutically beneficial. As a result, there has been growing interest in researching natural extracts and compounds as novel inhibitors of skin photoaging. Scientific studies have demonstrated that herbal extracts and their metabolites possess photoprotective and anti-aging properties. These compounds can inhibit oxidative processes as well as the activity of collagenase, elastase, and hyaluronidase.[8] This review aims to evaluate the effects and underlying mechanisms of herbal medicines in the treatment of photoaging.

The Mechanism of Skin Photoaging

Oxidative Stress

UV radiation-induced ROS is a major factor contributing to photodamage, playing a critical role in the signaling pathways involved in photoaging. During the energy conversion from UV-absorbing chromophores to molecular oxygen, ROS are generated. This process can enhance the scavenging ability of the ROS elimination system. However, when the accumulation of ROS exceeds the system's capacity for neutralization, it causes various types of damage, including direct DNA damage, protein degradation, dermal matrix disruption, melanogenesis disorders, and inflammation. ROS encompasses many different species, and the type of ROS produced depends on the UV wavelength. UVA primarily generates singlet oxygen ($^1O_2$) through a photosensitive reaction with endogenous chromophores, while UVB predominantly produces superoxide anion ($\bullet O_2^-$) via the activation of NADPH oxidase and respiratory chain reactions. UVA can also activate NADPH oxidase and photosensitize advanced glycation products to generate $\bullet O_2^-$, which can further be converted into hydrogen peroxide (H_2O_2) spontaneously or by superoxide dismutase (SOD). Subsequently, H_2O_2 can be converted into highly reactive hydroxyl radicals through the Fenton reaction.[10,11] UV irradiation also increases the expression of inducible nitric oxide synthase (iNOS), which produces nitric oxide (NO). The reaction between NO and superoxide results in the formation of peroxynitrite and other ROS.[12,13] These compounds directly damage DNA, lipids, and proteins and activate downstream proteins such as Raf, protein tyrosine phosphatases (PTPs), and MEKK1, leading to the activation of MAPKs and NF-κB.[11]

DNA Damage

DNA can be damaged both directly and indirectly by UV radiation, with UVB being the primary cause of direct damage.[14] UV irradiation primarily affects telomeres and mitochondrial DNA (mtDNA). Damage to telomeres activates DNA damage response proteins, such as p53, which can trigger cell apoptosis.[7] Additionally, mtDNA, which is located

near the respiratory chain where ROS are substantially produced, is highly susceptible to oxidative damage. Accumulation of ROS can impair mtDNA repair capacity, resulting in increased mutations, further ROS generation, and exacerbated cellular damage.[15]

Signal Transduction

UV radiation activates several cytokine and growth factor receptors, including the epidermal growth factor receptor (EGF-R), tumor necrosis factor (TNF)-α receptor, platelet-activating factor (PAF) receptor, interleukin (IL)-1 receptor, and platelet-derived growth factor (PDGF) receptor.[16] These receptors activate multiple signal transduction pathways. Photooxidative stress regulates several key pathways, such as the mitogen-activated protein kinase (MAPK), nuclear factor-kappa B (NF-κB)/p65, and nuclear factor erythroid 2-related factor 2 (Nrf2) pathways.[17] The primary MAPK pathways include extracellular signal-regulated kinases (ERK), c-Jun N-terminal kinase (JNK), and p38 MAPK. Activation of MAPK leads to the phosphorylation of transcription factor activator protein-1 (AP-1), which regulates the expression of matrix metalloproteinases (MMPs). Nrf2, a key regulator of the antioxidant response, controls the expression of endogenous antioxidant systems and mitochondrial bioenergetics. UV radiation suppresses Nrf2 expression and its target genes. Additionally, NF-κB activation is associated with oxidative modifications of cellular membranes induced by UV radiation, leading to the activation of inflammatory cytokines such as IL-1, IL-6, vascular endothelial growth factor (VEGF), TNF-α, and TNF-β, particularly in fibroblasts, keratinocytes, leukocytes, and endothelial cells.[17]

Changes in the Extracellular Matrix

The extracellular matrix (ECM) is the structural foundation of the skin, composed primarily of collagen, elastin, and glycosaminoglycans (GAGs). Collagen, the most abundant ECM component, provides structural support and is essential for the skin's tensile strength.[18] Elastin contributes to skin elasticity, while GAGs are responsible for skin hydration. ECM degradation occurs through the action of MMPs, whose transcription is primarily regulated by the transcription factor AP-1, activated by MAPK pathways. MMPs, which include MMP-1, MMP-3, and MMP-9, are zinc-dependent endopeptidases. MMP-1 is responsible for the degradation of collagen types I and III, while MMP-9 further degrades collagen fragments. MMP-3 targets collagen type IV, which forms the basement membrane. Following UV exposure, keratinocytes upregulate MMP-9 expression, which can exacerbate the inflammatory response by stimulating the production of TNF-α and IL-1β.[19] The transforming growth factor β (TGF-β)/Smad pathway promotes the growth of dermal fibroblasts and ECM synthesis. TGF-β binds to its receptor, activating the Smad2/3 transcription factors, which, in conjunction with Smad4, transmit signals to the nucleus to initiate the transcription of collagen genes such as COL1A1 and COL3A1.[20] Furthermore, Smad7 downregulates Smad2/3 activation by interacting with the TGF-β receptor. AP-1-induced downregulation of TGF-β receptor expression impairs the TGF-β/Smad signaling pathway.[21] Thus, UV radiation activates the MAPK pathway, stimulating AP-1, which in turn drives the transcription of MMPs, inhibits procollagen gene expression, and disrupts the TGF-β/Smad pathway. These events contribute to the morphological and histological changes observed in photoaged skin, including coarse wrinkles, rough texture, reduced collagen content, abnormal elastic fiber deposition, and increased glycosaminoglycan levels.

Inflammation and Immunosuppression

After UV irradiation, key inflammatory pathways are activated, including the NF-κB, p38/MAPK, and PI3K/Akt pathways. These pathways trigger the release of various inflammatory cytokines and enzymes, such as IL-1, IL-6, VEGF, TNF-α, TNF-β, and COX-2.[17,22] Skin cells, such as keratinocytes, fibroblasts, leukocytes, and endothelial cells, release these inflammatory factors, further activating the inflammatory pathways and stimulating the production of MMP-1, leading to more inflammation and ECM degradation. Furthermore, inflammatory cytokines damage the mitochondria, which results in increased ROS accumulation. NF-κB typically forms an inactive complex with the inhibitory molecule IκB in the cytoplasm. After UV radiation, IκB is degraded, allowing NF-κB to translocate into the nucleus and induce the expression of proinflammatory target genes.[23,24] Additionally, these proinflammatory cytokines activate NF-κB, leading to the production of MMP-1 and basic fibroblast growth factor (bFGF).[25,26] UVB-induced ROS formation can also activate the PI3K/Akt pathway, which regulates COX-2 expression.[27] Furthermore, UV radiation can have an

immunosuppressive effect on the skin, primarily by affecting Langerhans cells (LCs). UVR reduces the number of LCs and impairs their antigen-presenting capacity.[28,29] UVR induces the migration of epidermal LCs to lymph nodes, resulting in the depletion of LCs in the epidermis.[30] Additionally, keratinocytes can suppress antigen-presenting cell (APC) function by releasing immunosuppressive factors, such as IL-10 and TNF-α.

Melanogenesis

Several factors influence pigmentation, including ROS, cytokines, and hormones. The composition of melanin is closely related to tyrosine (TYR) and tyrosinase-related protein (TYRP), and the expression of TYR is induced by the microphthalmia-associated transcription factor (MITF).[31,32] It has been found that the MAPK and cAMP/PKA signaling pathways play significant roles in melanogenesis after UV irradiation. Activation of the cAMP/PKA pathway stimulates MITF expression, which in turn promotes the expression of melanogenesis-related genes. Moreover, after UV exposure, higher ROS levels in melanocytes activate the MAPKs pathway, enhancing MITF and TYR expression and leading to increased synthesis and transport of melanin.[32–34] Additionally, UVB radiation stimulates keratinocytes and fibroblasts to secrete numerous paracrine cytokines that regulate melanogenesis. These cytokines increase cAMP levels in melanocytes, activating PKA and enhancing MITF expression.

Photodamaged Vascular Endothelial Cells and Blood Vessels

UV radiation induces damage to microvasculature, leading to both deterioration and abnormal vascularization.[35,36] Aging of vascular endothelial cells (VECs) results in the loss of normal capillaries and irregular dilation of the remaining vessels, particularly in the upper dermis, where extensive degenerative changes occur in the connective tissue matrix. Moreover, expansive vessels embedded in elastin fibers exacerbate the degradation of VECs and activate abnormal VECs with an increased number of organelles and pinocytic vesicles. This leads to impaired blood circulation, reduced skin temperature, subcutaneous hematomas, and delayed wound healing.[37]

Apoptosis

UV-induced apoptosis primarily occurs through two pathways: the mitochondrial pathway and the death receptor pathway. The mitochondrial pathway involves the downregulation of Bcl-2 and the activation of Bax, which leads to the release of cytochrome C (CytoC) from mitochondria. CytoC, together with apoptosis-activating factor and deoxyadenosine triphosphate, activates caspase-9, which cleaves downstream caspase-3, resulting in cell death. The death receptor pathway includes Fas/FasL, TNFR, and TNF-related apoptosis-inducing ligand (TRAIL) signaling pathways. Fas/FasL activation leads to the activation of caspase-8, which downregulates Bcl-2 and initiates apoptosis through the mitochondrial pathway.[38] Additionally, UV-induced DNA damage activates the tumor suppressor protein P53, inducing apoptosis in keratinocytes and fibroblasts.[39]

The Role of Medicinal Plants in Photoaging Prevention

Photoaging damages various skin cells, including fibroblasts, keratinocytes, melanocytes, endothelial cells, and Langerhans cells, resulting in clinical changes. Among these, fibroblasts and keratinocytes are most susceptible. Recently, herbal medicines have garnered increasing attention and are widely used in many medical fields. These plants often contain active ingredients that determine their therapeutic effectiveness. Moreover, herbal formulations that combine different herbs can enhance each other's therapeutic effects, which is a common practice in many Asian countries. Various studies have shown that medicinal plants can influence different skin cells through multiple mechanisms, effectively delaying the aging process. This review focuses on the roles and mechanisms of traditional herbal medicines in inhibiting photoaging across different skin cells. The roles of medicinal plants and their potential mechanisms are summarized in Tables 1–3.

The Effect of Medical Plants on Fibroblasts

Several medicinal plant compounds activate the Nrf2/Keap1 pathway, leading to increased transcription of antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase, thereby enhancing the skin's intrinsic defense system against UV-induced ROS.

Table 1 The Anti-Photoaging Effect of Active Ingredients and Extracts from Plants on Fibroblasts

Categories	Names	Location	Extract Method	Experiments/Dose		UV Types and Doses	Mechanism		Ref.
				in vitro	in vivo		Pathways	Molecules	
Effective compounds	***Gallic acid***	–	Ethanol	NHDFs/0.1, 1, and 10 μM	Mice /1%,5%	UVB (144mJ/cm2)	AP-1↓/ TGF-β1↑	MMP-1↓, ROS↓, IL-6↓	[40]
	Magnolol	-	50% ethanol	NHDFs /5,10,20 μM	Mice /20 μM	UVB (4 J/cm2)	NF-κB↓	MMP-1 ↓, bFGF ↓	[41]
	(-)Epigallocatechin gallate	-	-	Human dermal fibroblasts /1,10,20μM	-	UVB (100mJ/cm2)	MAPKs↓	ROS↓, MMP-1↓, MMP-8↓, MMP-13↓	[42]
	Magnesium Lithospermate B	-	-	Hs27 /2μM, 10 μM	Mice /2,8 (mg/kg/day)	UVB (30mJ/cm2)	TGF-β1↑	COL1A1↑, COL3A1↑	[43]
	Erythrodiol-3-acetate	-	-	Human dermal fibroblasts /0.01,0.1,1 μM	-	UVB (100mJ/cm2)	-	MMP-1↓	[44]
Herbal extracts	***Coriandrum sativum L.***	Aisa, North America	70% ethanol	NHDFs /1,10,100μg/mL	Mice /0.5%,1%	UVB (144mJ/cm2)	AP-1↓ /TGF-β1↑	MMP-1↓, ROS↓	[45]
	Eucalyptus globulus	Australia		NHDFs /1,10,100 μg/mL	Mice /1%,5%	UVB (144mJ/cm2)	TGF-β↑/ Smad	ROS↓, AP-1↓, MMP-3↓, IL-6↓	[46]
	Michelia alba	South-east Asia		Hs68/50, 100, 500,1000 μg/mL	-	UVB (80mJ/cm2)	MAPKs↓	MMPs↓	[47]
	Emblica officinalis fruit	India		HS68 /10,20,40μg/mL	-	UVB (10mJ/cm2)	-	MMP-1↓, ROS↓	[48]
	Perilla frutescens (L). Britt.	Aisa	50% ethanol.	HDFs /1, 5, 20 μg/mL	Mice /0.5%	UV (100mJ/cm2)	MAPKs↓	MMP-1, 3↓, ROS↓, AP-1↓	[49]
	Rubus idaeus L.	Aisa, Europe	Ethyl-alcohol	NHDFs /1,10,100μg/mL	-	UVB (144mJ/cm2)	MAPKs↓, NF-κβ↓ AP-1↓ /TGF-β1↑	MMP-1, 3↓, ROS↓, IL-6↓, IL-1β↓	[50]
	Helianthus annuus L Flower	North America	50% ethanol	NHDFs /1,10,100μg/mL	-	UVB (144mJ/cm2)	MAPKs↓ Nrf2↑, AP-1↓ /TGF-β1↑	MMP-1, 3↓, ROS↓, IL-6↓, COX-2↓, iNOS↓, TNF-α↓	[51]
	Ixora	India	Water or methanol	Hs68 /0,1,5,10,50 μg/mL	-	UVB (40mJ/cm2)	MAPKs↓	MMP-1, 3, 9↓, NO↓, COX-2↓	[52]
Polyherbal mixtures formulations	***Panax ginseng Meyer and Crataegus pinnatifida***	China, Russia, North Korea	Patent protocol	NHDFs /1,10,100 μg/mL	-	UVB (144mJ/cm2)	-	MMP −1↓	[53]
	Gelidium amansii and Cirsium japonicum	Russia, Korea, Japan	50% ethanol	Hs68 /10 μg/mL	Mice /500 mg / kg	UVB (50mJ/cm2)	-	MMP-1↓	[54]

https://doi.org/10.2147/CCID.S538326

Table 2 The Anti-Photoaging Effect of Active Ingredients and Extracts from Plants on Keratinocyte

Categories	Names	Location	Extract Method	Experiments/Dose		UV Types and Dosage	Mechanism		Ref.
				in vitro	in vivo		Pathways	Molecules	
Effective compounds	**Timosaponin A-III**	China, Japan, Korea	–	HaCaT /0.025, 0.05, 0.1 μM	–	UVB (20mJ/cm2)	–	MMP-1↓, IL-1β↓, IL-8 ↓, TNF-α↓, TIMP↑	[55]
	Ginsenoside	China, Korea, Japan	Ethyl-acetate	HaCaT /1, 5, 10, 50 μM	-	F1:UVB (60mJ/cm2) Rb2:UVB (70 mJ/cm2)	F1:Bcl-2↑	F1 (Brn-3a↓) Rb2 (ROS↓, MMP-2↓)	[38, 56]
	Macelignan	Aisa	100% ethanol	HaCaT /0.1,0.5,1μM	-	UVB (30mJ/cm2)	MAPKs↓, PI3K/Akt↓ AP-1↓	MMP-9↓, COX-2 ↓,	[57]
	Luteolin	-	-	HaCaT /5, 10 μM	Mice /10,40 nmol	UVB (10mJ/cm2)	MAPKs↓ AP-1↓	MMP-1↓,	[58]
	Asiatic acid	-	-	HaCaT/ 5 μM	-	UVA (80–320mJ/cm2)	-	ROS↓, MMP-2↓, P53↓	[59]
	Andrograph-olide sodium bisulfate	-	-	HaCaT /10,30,100 μM	-	UVB (90mJ/cm2)	NF-κB↓, Keap1/ Nrf2↑	ROS↓, p65↓, IL-1β↓, IL-6↓, TNF-α↓	[60]
Herbal extracts	***Cynanchum wilfordii Hemsley***	Aisa	Water	HaCaT /100,200,400 μg/mL	-	UVB (10mJ/cm2)	MAPKs↓, AP-1↓	MMP-1↓,	[61]
	Rhus javanica	Aisa	95% ethanol	HaCaT /25,50,100 μg/mL	Mice /40,200 (mg/kg/day)	UVB (40mJ/cm2)	MAPKs↓, AP-1↓	MMP-1↓, MMP-13 ↓, COX-2 ↓	[62]
	Labisia pumila	Malaysia	Water	HaCaT/ 0.00005% to 0.001%	-	UVB (25 or 52mJ/cm2)	-	TNF-α↓, COX-2↓, MMP-1, 9↓	[63]

Table 3 The Anti-Photoaging Effect of Active Ingredients and Extracts from Plants on Melanocytes, Endothelial Cells and Langerhans Cells

Categories	Names	Location	Extract Method	Experiments/Dose		UV Types and Dosage	Mechanism		Ref
				in vitro	in vivo		Pathways	Molecules	
Melanocytes	**Artemisia asiatica**	China, Japan, Korea	Ethanol	B16F10 /12.5,25,50,100 μg/mL	–	UVB (30 mJ/cm2)	MAPKs↓	ERK↑, MITF↓, tyrosinase↓, TRP-1↓, TRP-2↓	[64]
	Ganoderma lucidum polysaccharide	China	-	B16F10, PIG1 cells /40 μg/mL	Zebrafish /40 mg/mL	UVB (20 mJ/cm2 ×3d)	cAMP/ PKA↓, MAPKs↓	MITF↓, TYR↓, TYRP1↓, TYRP2↓, Rab27A↓, Myosin↓	[65]

(*Continued*)

Table 3 (Continued).

Categories	Names	Location	Extract Method	Experiments/Dose		UV Types and Dosage	Mechanism		Ref
				in vitro	in vivo		Pathways	Molecules	
		China	-	PIG1 cells /0,2.5,5,20 μg/mL	-	-	IL-6/STAT3↓ MAPKs↓	MITF↓, TYR↓, TYRP1↓, TYRP2↓, Rab27A↓, FSCN1↓, FGF2↓	[66]
	Valencene	Tropical, subtropical temperate regions	MeOH	B16F10 /10,30,90 μM	-	UVB (10 mJ/cm2)	Intracellular Ca2+ signaling↓	TRPV1↓, ORAI1↓	[67]
Endothelial cells	**Royal jelly**	-	-	HDMEC /10 μg/mL	-	UVB (10mJ/ cm²×3d)	-	miR-129-5p↓	[37]
	Rhus coriaria L. Fruit	Southern Europe	Ethanol	HMEC-1 /10 or 25 μg/mL	-	UVA (10, 15, 20 J/ cm2)	-	ROS↓, DNA damage↓	[68]
Langerhans cells	**Aloe barbadensis M.**	-	-	LC /10,100, 500μg/mL	Mice	UVB (2.4 KJ/m2)	-	Number and function of LC↑	[69]
	Green tea	Aisa	-	LC /2%, 3%, 4%, and 5%	-	UVR (25–40mJ/ cm2)	-	Number of LC↑, CD1a+↑	[70]

Effective Compounds

Gallic Acid

Gallic acid (GA), a phenolic compound, is found in various plants such as *Rhus verniciflua*, green tea, and *Ziziphus mauritiana* seeds.[71–73] GA has antioxidant properties and is known to promote healing and exhibit anticancer activity.[40,74,75] Hwang et al[40] found that in UVB-irradiated normal human dermal fibroblasts (NHDFs), GA reduced ROS activity, decreased MMP-1 and IL-6 levels, and upregulated TGF-β1, leading to increased collagen production and normal elastin levels. Another study showed that GA-treated NHDFs inhibited the activated forms of c-Fos and c-Jun, key components of the AP-1 transcription factor. By inhibiting AP-1 expression, GA reduces MMP-1 levels and stimulates TGF-β1 production, which is crucial in the MAPK signaling pathway. As a result, GA helps reduce skin dryness and wrinkle formation.

Magnolol

Magnolol is a phenolic compound derived from *Magnolia obovata* Thunb. It has tumor metastasis-inhibiting, anti-inflammatory, and antibacterial properties.[76,77] Tanaka et al[41] discovered that Magnolol prevents MMP-1 production in fibroblasts that overexpress p65 (a major NF-κB subunit). The mechanism appears to involve the inhibition of downstream NF-κB signaling without affecting IκB degradation. This suggests that Magnolol protects the skin from photoaging by suppressing NF-κB signaling.

Epigallocatechin Gallate

Epigallocatechin gallate (EGCG), a bioactive catechin from green tea, has been shown to mitigate solar oxidative damage and carcinogenesis.[78,79] Bae et al[42] found that EGCG inhibited ROS generation and MMP-1, MMP-8, and MMP-13 production in a dose-dependent manner after UVB exposure. This effect occurs through interference with the MAPK pathways, including the inhibition of JNK, p38 MAPK, and ERK1/2 phosphorylation in fibroblasts.

Magnesium Lithospermate B

Magnesium lithospermate B (MLB), a polyphenolic acid from *Danshen* (a traditional herb), has been reported to possess antioxidative, neuroprotective, and antidiabetic properties.[80–82] MLB has been shown to enhance the expression of collagen-modulating genes, such as COL1A1 and COL3A1, in fibroblasts after UVB radiation. These changes lead to increased expression of TGF-β1 and collagen, suggesting that MLB can prevent skin photoaging by regulating the ECM.[43]

Styrax japonica

Styrax japonica (Styracaceae), a deciduous tree found in East Asia, has been used to treat inflammatory diseases, coughs, and as a piscicidal agent.[44] Erythrodiol-3-acetate, a biologically active compound from *Styrax japonica*, has been shown to reduce MMP-1 expression and enhance type-1 procollagen expression in a dose-dependent manner in fibroblasts after UVB exposure. However, the precise molecular mechanisms behind these effects remain to be fully elucidated.[83]

Herbal Extract and Their Impact on Skin Health

Coriandrum sativum

Coriandrum sativum L. (CS), a member of the Apiaceae family, is a herbaceous plant known for its various biological benefits, including antidiabetic, hypolipidemic, and antimutagenic properties.[84–86] The ethanol extract of CS (CSE) has been shown to significantly reduce UVB-induced MMP-1 expression and promote collagen production in a dose-dependent manner. This effect is mediated by the reduction of reactive oxygen species (ROS) and the inhibition of AP-1 expression. Despite no significant changes in the total c-Jun and c-Fos levels, their activated forms were notably reduced after treatment with 10 and 100 μg/mL CSE. In animal studies, CSE helped mitigate collagen and elastin degradation by enhancing TGF-β1 activity.[45]

Eucalyptus globulus

Eucalyptus globulus is widely known for its antimicrobial, antibacterial, and antiviral properties.[87–89] Park et al[46] demonstrated that the ethanol extract of Eucalyptus globulus (EGE) inhibits the mobilization of AP-1, which leads to decreased ROS and MMP-1 levels in UVB-irradiated human dermal fibroblasts (NHDFs). EGE also stimulates the recovery of TGF-β1 and Smad2/3 levels, while reducing Smad7 expression in a dose-dependent manner. Furthermore, EGE reduces the production of MMP-3 and IL-6.

Michelia alba

Michelia alba, a member of the Magnoliaceae family, has traditionally been used to treat conditions such as prostatitis, bronchitis, and malaria.[90,91] Chiang et al[47] showed that the extract of Michelia alba (MAE) suppresses the expression of MMPs (MMP-1, MMP-3, and MMP-9) by inhibiting MAPK signaling pathways (primarily JNK and ERK) in UVB-exposed human foreskin fibroblasts (Hs68). This results in a concentration-dependent repair of collagen, though type I procollagen expression is slightly affected. Additionally, MAE inhibits elastase activity at a concentration of 500 μg/mL and increases hyaluronic acid content in Hs68 cells.

Emblica officinalis

Emblica officinalis, also known as Phyllanthus emblica L., is widely used for treating inflammatory diseases, diabetes, skin disorders, and for beauty care.[92] Adil et al[48] found that the extract of Emblica officinalis (EOE) exhibits potent ROS scavenging and antioxidant properties. It reduces MMP-1 levels in Hs68 cells and protects against UVB-induced cell death. EOE also helps restore collagen and hyaluronic acid levels. Furthermore, the functional tannins in EOE may regulate MMP expression through mechanisms involving the activation of AP-1 and NF-κB.

Perilla frutescens

Perilla frutescens (L) Britt., a member of the Lamiaceae family, has long been used to treat skin irritation and hypersensitivity.[49] Bae et al[49] demonstrated that the extract of Perilla (PLE) inhibits the expression of MMP-1 and MMP-3, while promoting the production of type I collagen in UV-irradiated human dermal fibroblasts. The mechanism involves blocking ROS generation, suppressing the activation of c-Fos and c-Jun, and inhibiting the phosphorylation of ERK, JNK, and p38, which leads to reduced AP-1 and MMP expression.

Red Raspberry

Red raspberry (Rubus idaeus L) is commonly consumed and gaining attention for its health benefits, including antioxidant, anti-inflammatory, and antimicrobial activities.[93] Its ethyl alcohol extract has been shown to repair the extracellular matrix (ECM) and reduce inflammation by inhibiting UVB-induced ROS production and MMP-1, MMP-3, IL-1β, and IL-6 expression. Additionally, the extract enhances the generation of type I procollagen through the suppression of MAPK and NF-κB pathways and the activation of the TGF-β/Smad pathway in UVB-irradiated NHDFs.[50]

Sunflower

Sunflower (Helianthus annuus L) is native to North America and its extract oil is commonly used in skin care products. It has been found to strengthen the skin barrier and alleviate symptoms of skin inflammation, such as dryness and itching.[94,95] Hwang et al[51] reported that Helianthus annuus flower extract (HAF) significantly inhibits UVB-induced ROS, MMP-1, and MMP-3 production, while enhancing the formation of type I procollagen in NHDFs. The anti-photoaging action of HAF is linked to the activation of Nrf2 nuclear translocation, increased TGF-β1 levels, and the inhibition of AP-1 and MAPK phosphorylation. Furthermore, HAF suppresses the secretion of VEGF and inflammatory cytokines such as IL-6, COX-2, and TNF-α after UVB exposure.

Ixora parviflora

Ixora parviflora, belonging to the Rubiaceae family, is rich in polyphenols and widely used in traditional medicine in India.[96,97] The extract of Ixora parviflora (IPE) exhibits ROS-scavenging activity.[98] Wen et al[52] demonstrated that IPE increases the expression of type I procollagen while decreasing the levels of MMP-1, MMP-3, and MMP-9 in UV-irradiated Hs68 cells. The underlying mechanism involves the inhibition of p38, ERK, and JNK phosphorylation and downregulation of Smad7 expression. Additionally, IPE inhibits nitric oxide (NO) formation and COX-2 expression.

Polyherbal Mixtures and Their Effects

Panax ginseng Meyer and Crataegus pinnatifida are both renowned for their anti-aging, anti-inflammatory, and antioxidative properties.[99–103] Hwang et al[53] produced a polyherbal mixture of Panax Ginseng and Crataegus pinnatifida in a 1:1 ratio. This combination strongly enhances type I procollagen synthesis and reduces MMP-1 expression in NHDFs. The combination was more effective for photoaging than the individual plants alone.

Gelidium amansii (GA), a red alga, and Cirsium japonicum (CJ), known for its pharmacological effects such as controlling hypertension and bleeding,[104,105] were used in a mixture (GCM) at a 4:1 ratio (w/w). Kim et al[54] found that this mixture, particularly when fermented, increased type I procollagen expression and downregulated MMP-1 expression in UVB-irradiated Hs68 cells. Fermented GCM was more effective than the unfermented mixture.

Medical Plants and Keratinocytes

Anemarrhena asphodeloides

Anemarrhena asphodeloides is traditionally used for its hypoglycemic, antipyretic, and antidepressive properties.[106,107] Timosaponin A-III, a major chemical component, inhibits MMP-1 production and upregulates tissue inhibitors of metalloproteinase (TIMP), promoting the premature senescence phenotype. Additionally, Timosaponin A-III attenuates the production of proinflammatory cytokines (IL-1β, IL-8, TNF-α) in human immortalized keratinocytes (HaCaT), suggesting its potential as a photoprotective agent after UVB exposure.[55]

Panax ginseng, widely used in Asian traditional medicine, contains ginsenosides, which have numerous health benefits including antioxidation and anti-inflammation.[56] Lee et al[38] reported that ginsenoside F1 can significantly prevent UVB-induced keratinocyte apoptosis by inhibiting Bcl-2 promoter activity in HaCaT cells. Furthermore, ginsenoside Rb2 reduces ROS generation and MMP-2 activity in UVB-irradiated HaCaT cells.[56]

Macelignan, derived from Myristica fragrans Houtt., has various medicinal properties such as antioxidation, anti-inflammation, and antitumor effects.[108–110] Anggakusuma et al[57] demonstrated that macelignan (0.1–1 μM) dose-dependently attenuates MMP-9 and COX-2 overexpression in HaCaT cells by downregulating MAPK and PI3K/Akt pathways. It inhibits p38, ERK, Akt, JNK, c-Jun phosphorylation, and the expression of c-Fos and PI3K.

Luteolin, a flavonoid found in many plants, including parsley, celery, and thyme, has anti-inflammatory and antioxidant effects.[111–114] Lim et al[58] have indicated that luteolin markedly inhibited JNK1 and p90RSK2 activity, but showed no inhibitory effect on JNK2 and ERK2, leading to blocking of MAPK pathway, further causing MMP-1 downregulation. And Luteolin also inhibited UVB-related AP-1 transcriptional activity, c-Fos promoter activation and c-Jun phosphorylation within human keratinocytes, thus protect skin from ECM degradation.

Asiatic acid, a pentacyclic triterpenoid found in *Centella asiatica*, has been reported for its therapeutic effects in dermatosis and leprosy.[115] Interestingly, ursolic acid, a compound with a similar structure to asiatic acid, also shows potential in enhancing skin collagen concentration. Additionally, it possesses anti-inflammatory, anti-invasive, and skin tumor-preventive properties.[116,117]

Research by Soo Lee et al[59] found that asiatic acid (5 μM) and ursolic acid (10 μM) can suppress MMP-2 activity. They also notably reduce ROS generation and lipid peroxidation in UVA-irradiated HaCaT cells. Furthermore, asiatic acid inhibits the expression of P53 in HaCaT cells, which may help mitigate dermal cell damage induced by UVA irradiation during photoaging.

Andrographolide sodium bisulfate (ASB), an active component of *Andrographis paniculata*, is known for its anti-inflammatory and antipyretic-analgesic effects.[118] ASB has been shown to inhibit oxidative damage and NF-κB-mediated inflammation, particularly in diabetes mellitus.[119] According to WANG et al,[60] ASB significantly reduces ROS generation and stimulates Nrf2 production in UV-treated HaCaT cells. Additionally, ASB downregulates the p65 expression, thus reducing the production of pro-inflammatory cytokines such as IL-1β, IL-6, and TNF-α. These findings suggest that ASB alleviates UV-induced photodamage by activating the Keap1/Nrf2 pathway while inhibiting the NF-κB pathway in HaCaT cells.

Herbal Extracts for Skin Health

Cynanchum wilfordii, commonly known for alleviating rheumatoid arthritis and vascular conditions,[120–122] has been investigated for its anti-photoaging properties. Jang et al[61] separated a high-molecular-weight fraction (HMF) from this plant and observed its effects on human keratinocytes. HMF was shown to reduce MMP-1 expression and c-Jun phosphorylation in a dose-dependent manner. It also attenuates the phosphorylation of JNK, ERK, and p38 in UVB-treated keratinocytes.

Rhus javanica, a member of the Anacardiaceae family, is recognized for its antimicrobial, antioxidant, and anti-tumor activities.[123–125] The extract of *Rhus javanica* (RJE) has been found to inhibit UVB-induced phosphorylation of EGFR, MAPKs, and Akt, thereby lowering AP-1 activity and decreasing MMP-1 and COX-2 expression in HaCaT cells. Furthermore, RJE reduces wrinkles and suppresses COX-2 and MMP-13 expression in UVB-treated hairless mice, highlighting its potential in preventing UV-induced dermatitis and photoaging.[62]

Labisia pumila, a popular herbal remedy in Malaysia, is widely used for maintaining female reproductive health and treating postpartum complications.[126,127] Choi et al[63] demonstrated that treating HaCaT cells with Labisia pumila extract significantly reduced the secretion of TNF-α and COX-2 expression in a dose-dependent manner. Moreover, the extract attenuated MMP-1 and MMP-9 expression while increasing type I procollagen expression, promoting extracellular matrix (ECM) synthesis.

Herbal Effects on Melanocytes

Artemisia asiatica

Artemisia asiatica, a well-known herb in Asia for treating inflammation and infections,[128,129] has demonstrated notable anti-inflammatory properties.[130] Jeong et al[64] found that Artemisia asiatica ethanol extract (Aa-EE) significantly reduced melanin production in B16F10 melanoma cells, which were treated with α-Melanocyte-stimulating hormone (α-MSH). The mechanism of action involves enhancing ERK phosphorylation, which indirectly degrades MITF and directly reduces MITF expression, leading to decreased expression of melanin-related genes, TYR and TYRP-1.

Ganoderma lucidum Polysaccharide

Ganoderma lucidum, a prominent Chinese herb, is known for its anti-aging, immune-regulatory, and gut health-promoting properties.[131–133] Its polysaccharide (GLP) has shown potential in inhibiting UVB-induced skin pigmentation by suppressing the cAMP/PKA and MAPK pathways. This results in reduced expression of melanogenesis-related genes such as MITF, TYR, and TYRP1 in UVB-treated B16F10 cells and immortalized human melanocytes (PIG1). Additionally, Jiang Ling et al[66] found that GLP inhibits the paracrine effects of keratinocytes and fibroblasts via the IL-6/STAT3 pathway, further decreasing melanin production in melanocytes.

Valencene from Cyperus rotundus

Cyperus rotundus, known for its antioxidant and antiproliferative properties,[134,135] contains valencene, which has been shown to suppress melanogenesis. Valencene reduces currents from TRPV1 and ORAI1 channels in B16F10 melanoma cells at concentrations of 90 μM, leading to a decrease in melanin synthesis, independent of direct inhibition of tyrosinase activity Endothelial Cell Protection.[67]

The Effect of Medical Plants on Endothelial Cells

Royal Jelly

Royal jelly, produced by honeybees, is widely known for its anti-aging and anti-hypertensive effects.[136] A study by Kawano et al[37] found that royal jelly can counteract UV-induced downregulation of miR-129-5p, a microRNA related to photoaging, thus enhancing the number of dermal microvascular endothelial cells (HDMECs) and promoting endothelial health.

Rhus coriaria L. Fruit

Rhus coriaria fruit, native to the Mediterranean region, is used for various therapeutic purposes, including treating gastrointestinal issues and arthritis.[137] Nozza et al[68] showed that macerated extracts of *Rhus coriaria* significantly reduced ROS production and oxidative damage in microvascular endothelial cells (HMEC-1) following UVA exposure. Higher concentrations of the extract also counteracted DNA damage in these cells.

The Effect of Medical Plants on Langerhans Cells

Aloe barbadensis M

Aloe barbadensis M., commonly known as Aloe vera, has been extensively used as a health-promoting food additive, cosmetic ingredient, and medicinal herb. It has been reported to exhibit a preventive effect against UVB-induced suppression of skin hypersensitivity.[138] Lee et al[69] discovered that low molecular mass immunomodulators (G1C2F1), found in Aloe vera, can counteract immune suppression in UVB-irradiated Langerhans cells (LCs) within the skin. These immunomodulators help restore the functionality of LCs, which is typically impaired by UVB irradiation. Furthermore, topical application of Aloe vera extract has been shown to preserve both the quantity and morphology of LCs in the skin following UV radiation exposure.

Green Tea

Green tea has been extensively studied for its antimutagenic properties and potential for cancer chemoprevention, owing to its rich content of polyphenolic compounds and antioxidants.[139] Li et al[70] reported that a 3% green tea extract (GTE) provides partial protection against the downregulation of LCs following UV radiation (UVR). Specifically, treatment with GTE significantly inhibited the expression of matrix metalloproteinases MMP-2 and MMP-9, as well as the depletion of CD1a+ LCs. The precise mechanisms underlying these effects remain to be elucidated and warrant further investigation.

Discussion and Conclusion

This paper reviews the current research on the role of medicinal herbs in the prevention and treatment of photoaging. The application of Chinese herbal medicine has garnered increasing attention in recent years, particularly for its potential in combating skin aging caused by ultraviolet (UV) radiation. Herbal medicines primarily exert their effects through the

active components found in their extracts, which can be utilized either individually or in combination with other extracts for synergistic benefits.

Many plant-derived antioxidants, such as flavonoids and phenolic acids, are biosynthesized via the phenylpropanoid pathway—a major secondary metabolic route initiated by the deamination of phenylalanine through phenylalanine ammonia-lyase (PAL) activity. These metabolites play key roles in photoprotection by scavenging ROS and modulating inflammatory pathways. Medicinal plants can effectively inhibit skin photoaging in various skin cell types through distinct signaling pathways. For instance, in the context of photoaging, the MAPK pathway can be suppressed by extracts of EGCG, *Michelia alba, Perilla frutescens* (L). Britt, and *Rubus idaeus* L. in fibroblasts, while suppression of the same pathway has been observed with extracts of *Macelignan, Luteolin, Rhus javanica*, and *Cynanchum wilfordii* Hemsley in keratinocytes. Furthermore, *Magnolol* and *Rubus idaeus* L. extract have been shown to inhibit the NF-κB pathway in fibroblasts. Additionally, *Artemisia asiatica* and *Ganoderma lucidum* polysaccharide extracts provide protection to melanocytes by modulating the MAPK pathway, while *Rhus coriaria* L. fruit extract mitigates photodamage in endothelial cells by reducing reactive oxygen species (ROS). Both Aloe barbadensis M. and green tea extracts have been found to enhance the number of LCs during photoaging.

The therapeutic potential of these herbal extracts is evident, yet several challenges need to be addressed:

Unclear Molecular Mechanisms: A substantial portion of current research focuses on the effects of herbal extracts on ROS, matrix metalloproteinases (MMPs), and collagen synthesis. However, the detailed molecular mechanisms remain largely unexplored, necessitating further investigation to clarify these pathways.

Limited Scope of Research: Most in vitro experiments have been conducted on UV-induced fibroblasts and keratinocytes, with relatively few studies focusing on other skin cell types, such as melanocytes, vascular endothelial cells, immune cells, and adipocytes. In vivo studies typically examine the effects of medicinal plants through oral or topical administration; however, combined oral and topical administration has not been extensively studied, despite its potential to produce different results. Moreover, much of the research conducted thus far has involved aged rat skin. Further investigations are necessary to determine whether medicinal herbs can also protect against photoaging in artificial skin models or ex vivo skin tissue models.[140]

Impact of UVA and Infrared Radiation: While UVB is the most biologically damaging form of UV radiation, UVA and infrared radiation also contribute significantly to skin photoaging, particularly in deeper layers of the skin. However, there are relatively few studies examining the effects of UVA or infrared radiation. Specifically, the biological effects and mechanisms induced by infrared radiation (IRA) should be distinguished from UVR responses in human skin cells. Further research is required to evaluate effective treatments for IRA-induced photoaging.

Safety and Toxicity Considerations: Although herbal treatments are generally considered safe with few side effects, their long-term use in cosmetics raises concerns about potential adverse effects. While the side effects of most herbal medicines are not well-documented, existing clinical trials on these products often provide one-sided opinions. It is crucial to identify the potential side effects of these herbal treatments and explore alternatives to mitigate any risks associated with their use.

Some studies have evaluated the SPF value of plant-based formulations, reporting that polyphenol-rich extracts, such as from green tea, Phyllanthus emblica, and patchouli oil, provide measurable UV protection when applied topically, with SPF values ranging from 5 to 15, depending on concentration and formulation base.[141–151]

To further enhance the clarity and comprehensiveness of this review, additional seminal and recent studies have been incorporated to support key mechanistic insights. Early investigations established the structural and enzymatic damage caused by UV radiation, including epidermal destruction and elastase activation, which laid the foundation for our understanding of photoaging pathophysiology.[141–143] The roles of oxidative stress, extracellular matrix degradation, and cytokine-driven inflammation in both chronological and photoinduced aging have been extensively documented.[144–146] Furthermore, paracrine signaling between keratinocytes and fibroblasts has been shown to contribute significantly to wrinkle formation through the stimulation of elastase and matrix metalloproteinases.[146] In recent years, several medicinal plant-derived formulations—such as patchouli oil, *Caralluma adscendens*, and *Ipomoea carnea*—have demonstrated protective effects against UVB-induced damage in animal models, primarily via antioxidant and anti-inflammatory mechanisms.[147,148] Bioactive flavonoids like artocarpin have also been shown to attenuate UV-induced skin injury by

suppressing oxidative stress pathways.[149] Moreover, some natural compounds have been evaluated for their sun protection factor (SPF), supporting their potential integration into topical photoprotective formulations.[150,151] Together, these studies reinforce the therapeutic relevance of medicinal plants in combating UV-induced skin aging and further validate the mechanistic pathways summarized in this review.

In conclusion, medicinal plants represent a promising approach to the inhibition of skin photoaging and offer significant cosmetic and therapeutic benefits. Extensive preclinical evidence indicates that various herbal extracts exert anti-photoaging effects through multiple molecular pathways. These include inhibition of the MAPK/AP-1 signaling cascade to suppress matrix metalloproteinase expression, activation of the Nrf2/Keap1 pathway to enhance antioxidant defense, downregulation of NF-κB to mitigate inflammation, and stimulation of the TGF-β/Smad pathway to promote collagen synthesis and extracellular matrix repair. These mechanisms operate across different skin cell types—including fibroblasts, keratinocytes, melanocytes, endothelial cells, and Langerhans cells—highlighting the broad biological relevance of medicinal plants in skin photoprotection. However, further research is required to elucidate the full spectrum of molecular mechanisms, expand investigations to additional cell types and in vivo systems, and address concerns related to safety, long-term use, and formulation standardization. The continued exploration of these herbal remedies holds great potential for advancing skin care and dermatological treatments. Future studies should prioritize the development of standardized herbal formulations and focus on high-quality randomized clinical trials with adequate sample sizes, blinding, and long-term follow-up to validate efficacy and safety. At present, most clinical studies lack methodological rigor, thereby limiting the generalizability of their findings.

Data Sharing Statement

The datasets used and/or analysed during the current study available from the corresponding author on reasonable request.

Compliance with Ethics Guidelines

This manuscript is a review article and does not involve a research protocol requiring approval by the relevant institutional review board or ethics committee.

Funding

There is no funding to report.

Disclosure

The author declares that she has no competing interests.

References

1. Baik KK, Song WY, Song DK, et al. Protective effects of sesame glycoproteins on ultraviolet-induced skin aging: in vitro and in vivo studies. *Pharmaceuticals*. 2024;17(10):1306. doi:10.3390/ph17101306
2. Zhang M, Lin Y, Han Z, et al. Exploring mechanisms of skin aging: insights for clinical treatment. *Front Immunol*. 2024;15:1421858. doi:10.3389/fimmu.2024.1421858
3. Milutinov J, Pavlović N, Ćirin D, Atanacković Krstonošić M, Krstonošić V. The potential of natural compounds in UV protection products. *Molecules*. 2024;29(22):5409. doi:10.3390/molecules29225409
4. Pellacani G, Argenziano G. New insights from non-invasive imaging: from prospection of skin photodamages to training with mobile application. *J Eur Acad Dermatol Venereol*. 2022;36(Suppl 6):38–50. doi:10.1111/jdv.18197
5. Fisher GJ, Datta S, Wang Z, et al. C-Jun-dependent inhibition of cutaneous procollagen transcription following ultraviolet irradiation is reversed by all-trans retinoic acid. *J Clin Invest*. 2000;106(5):663–670. doi:10.1172/JCI9362
6. Daré RG, Oliveira MM, Truiti MCT, Nakamura CV, Ximenes VF, Lautenschlager SOS. Abilities of protocatechuic acid and its alkyl esters, ethyl and heptyl protocatechuates, to counteract UVB-induced oxidative injuries and photoaging in fibroblasts L929 cell line. *J Photochem Photobiol B*. 2020;203:111771. doi:10.1016/j.jphotobiol.2019.111771
7. Sun J, Jiang Y, Fu J, et al. Beneficial effects of epigallocatechin gallate in preventing skin photoaging: a review. *Molecules*. 2024;29(22):5226. doi:10.3390/molecules29225226
8. Duque L, Bravo K, Osorio E. A holistic anti-aging approach applied in selected cultivated medicinal plants: a view of photoprotection of the skin by different mechanisms. *Ind Crop Prod*. 2017;97:431–439. doi:10.1016/j.indcrop.2016.12.059

9. Mukherjee PK, Maity N, Nema NK, Sarkar BK. Bioactive compounds from natural resources against skin aging. *Phytomedicine*. 2011;19(1):64–73. doi:10.1016/j.phymed.2011.10.003
10. Masaki H. Role of antioxidants in the skin: anti-aging effects. *J Dermatol Sci*. 2010;58(2):85–90. doi:10.1016/j.jdermsci.2010.03.003
11. Putthong C, Panmanee T, Charoensit P, Ross S, Tongpoolsomjit K, Viyoch J. Efficacy of natural β-carotene chewable tablets derived from banana (Musa AA) pulp in reducing UV-induced skin erythema. *Nutrients*. 2024;17(1):65. doi:10.3390/nu17010065
12. Deliconstantinos G, Villiotou V, Stavrides JC. Increase of particulate nitric oxide synthase activity and peroxynitrite synthesis in UVB-irradiated keratinocyte membranes. *Biochem J*. 1996;320(Pt 3):997–1003. doi:10.1042/bj3200997
13. Mattson MP. Nitro-PDI incites toxic waste accumulation. *Nat Neurosci*. 2006;9(7):865–867. doi:10.1038/nn0706-865
14. Kammeyer A, Luiten RM. Oxidation events and skin aging. *Ageing Res Rev*. 2015;21:16–29. doi:10.1016/j.arr.2015.01.001
15. Ballard JW, Dean MD. The mitochondrial genome: mutation, selection and recombination. *Curr Opin Genet Dev*. 2001;11(6):667–672. doi:10.1016/S0959-437X(00)00251-3
16. Fisher GJ, Kang S, Varani J, et al. Mechanisms of photoaging and chronological skin aging. *Arch Dermatol*. 2002;138(11):1462–1470. doi:10.1001/archderm.138.11.1462
17. Bosch R, Philips N, Suárez-Pérez JA, et al. Mechanisms of photoaging and cutaneous photocarcinogenesis, and photoprotective strategies with phytochemicals. *Antioxidants*. 2015;4(2):248–268. doi:10.3390/antiox4020248
18. Thring TS, Hili P, Naughton DP. Anti-collagenase, anti-elastase and anti-oxidant activities of extracts from 21 plants. *BMC Complement Altern Med*. 2009;9(1):27. doi:10.1186/1472-6882-9-27
19. Gupta A, Kaur CD, Jangdey M, Saraf S. Matrix metalloproteinase enzymes and their naturally derived inhibitors: novel targets in photo-carcinoma therapy. *Ageing Res Rev*. 2014;13:65–74. doi:10.1016/j.arr.2013.12.001
20. Talwar HS, Griffiths CE, Fisher GJ, Hamilton TA, Voorhees JJ. Reduced type I and type III procollagens in photodamaged adult human skin. *J Invest Dermatol*. 1995;105(2):285–290. doi:10.1111/1523-1747.ep12318471
21. Quan T, He T, Kang S, Voorhees JJ, Fisher GJ. Solar ultraviolet irradiation reduces collagen in photoaged human skin by blocking transforming growth factor-beta type II receptor/smad signaling. *Am J Pathol*. 2004;165(3):741–751. doi:10.1016/S0002-9440(10)63337-8
22. Kang S, Fisher GJ, Voorhees JJ. Photoaging: pathogenesis, prevention, and treatment. *Clin Geriatr Med*. 2001;17(4):643–59, v–vi. doi:10.1016/S0749-0690(05)70091-4
23. Umezawa K, Chaicharoenpong C. Molecular design and biological activities of NF-kappaB inhibitors. *Mol Cells*. 2002;14(2):163–167. doi:10.1016/S1016-8478(23)15088-6
24. Okamoto T, Sakurada S, Yang JP, Merin JP. Regulation of NF-kappa B and disease control: identification of a novel serine kinase and thioredoxin as effectors for signal transduction pathway for NF-kappa B activation. *Curr Top Cell Regul*. 1997;35:149–161.
25. Bond M, Baker AH, Newby AC. Nuclear factor kappaB activity is essential for matrix metalloproteinase-1 and −3 upregulation in rabbit dermal fibroblasts. *Biochem Biophys Res Commun*. 1999;264(2):561–567. doi:10.1006/bbrc.1999.1551
26. Sabourin CL, Kusewitt DF, Applegate LA, Budge CL, Ley RD. Expression of fibroblast growth factors in ultraviolet radiation-induced corneal tumors and corneal tumor cell lines from monodelphis domestica. *Mol, Carcinog*. 1993;7(3):197–205. doi:10.1002/mc.2940070310
27. Bachelor MA, Cooper SJ, Sikorski ET, Bowden GT. Inhibition of p38 mitogen-activated protein kinase and phosphatidylinositol 3-kinase decreases UVB-induced activator protein-1 and cyclooxygenase-2 in a SKH-1 hairless mouse model. *Mol Cancer Res*. 2005;3(2):90–99. doi:10.1158/1541-7786.MCR-04-0065
28. Simon JC, Tigelaar RE, Bergstresser PR, Edelbaum D, Pd C. Ultraviolet B radiation converts Langerhans cells from immunogenic to tolerogenic antigen-presenting cells. Induction of specific clonal anergy in CD4+ T helper 1 cells. *J Immunol*. 1991;146(2):485–491. doi:10.4049/jimmunol.146.2.485
29. Denfeld RW, Hara H, Tesmann JP, Martin S, Simon JC. UVB-irradiated dendritic cells are impaired in their APC function and tolerize primed Th1 cells but not naive CD4+ T cells. *J Leukoc Biol*. 2001;69(4):548–554. doi:10.1189/jlb.69.4.548
30. Duthie MS, Kimber I, Dearman RJ, Norval M. Differential effects of UVA1 and UVB radiation on Langerhans cell migration in mice. *J Photochem Photobiol B*. 2000;57(2–3):123–131. doi:10.1016/S1011-1344(00)00087-7
31. Dynoodt P, Mestdagh P, Van Peer G, et al. Identification of miR-145 as a key regulator of the pigmentary process. *J Invest Dermatol*. 2013;133(1):201–209. doi:10.1038/jid.2012.266
32. Park HY, Kosmadaki M, Yaar M, Gilchrest BA. Cellular mechanisms regulating human melanogenesis. *Cell Mol Life Sci*. 2009;66(9):1493–1506. doi:10.1007/s00018-009-8703-8
33. Drira R, Sakamoto K. Sakuranetin induces melanogenesis in B16BL6 melanoma cells through inhibition of ERK and PI3K/AKT signaling pathways. *Phytother Res*. 2016;30(6):997–1002. doi:10.1002/ptr.5606
34. Peng HY, Lin CC, Wang HY, Shih Y, Chou ST. The melanogenesis alteration effects of Achillea millefolium L. essential oil and linalyl acetate: involvement of oxidative stress and the JNK and ERK signaling pathways in melanoma cells. *PLoS One*. 2014;9(4):e95186. doi:10.1371/journal.pone.0095186
35. Kelly RI, Pearse R, Bull RH, Leveque JL, de Rigal J, Mortimer PS. The effects of aging on the cutaneous microvasculature. *J Am Acad Dermatol*. 1995;33(5 Pt 1):749–756. doi:10.1016/0190-9622(95)91812-4
36. Chung JH, Eun HC. Angiogenesis in skin aging and photoaging. *J Dermatol*. 2007;34(9):593–600. doi:10.1111/j.1346-8138.2007.00341.x
37. Kawano Y, Makino K, Jinnin M, et al. Royal jelly regulates the proliferation of human dermal microvascular endothelial cells through the down-regulation of a photoaging-related microRNA. *Drug Discov Ther*. 2019;13(5):268–273. doi:10.5582/ddt.2019.01070
38. Kulms D, Schwarz T. Molecular mechanisms of UV-induced apoptosis. *Photodermatol Photoimmunol Photomed*. 2000;16(5):195–201. doi:10.1034/j.1600-0781.2000.160501.x
39. Widgerow AD, Grekin SK. Effecting skin renewal: a multifaceted approach. *J Cosmet Dermatol*. 2011;10(2):126–130. doi:10.1111/j.1473-2165.2011.00555.x
40. Hwang E, Park SY, Lee HJ, Lee TY, Sun ZW, Yi TH. Gallic acid regulates skin photoaging in UVB-exposed fibroblast and hairless mice. *Phytother Res*. 2014;28(12):1778–1788. doi:10.1002/ptr.5198
41. Tanaka K, Hasegawa J, Asamitsu K, Okamoto T. Magnolia ovovata extract and its active component magnolol prevent skin photoaging via inhibition of nuclear factor kappaB. *Eur J Pharmacol*. 2007;565(1–3):212–219. doi:10.1016/j.ejphar.2007.01.095

42. Bae JY, Choi JS, Choi YJ, et al. (-)Epigallocatechin gallate hampers collagen destruction and collagenase activation in ultraviolet-B-irradiated human dermal fibroblasts: involvement of mitogen-activated protein kinase. *Food Chem Toxicol*. 2008;46(4):1298–1307. doi:10.1016/j.fct.2007.09.112
43. Jung YR, Lee EK, Kim DH, et al. Upregulation of collagen expression via PPARβ/δ activation in aged skin by magnesium lithospermate B from Salvia miltiorrhiza. *J Nat Prod*. 2015;78(8):2110–2115. doi:10.1021/acs.jnatprod.5b00348
44. Yoshikawa K, Hirai H, Tanaka M, Arihara S. Antisweet natural products. XV. structures of jegosaponins A-D from styrax japonica Sieb. et Zucc. *Chem Pharm Bull*. 2000;48(7):1093–1096. doi:10.1248/cpb.48.1093
45. Hwang E, Lee DG, Park SH, Oh MS, Kim SY. Coriander leaf extract exerts antioxidant activity and protects against UVB-induced photoaging of skin by regulation of procollagen type I and MMP-1 expression. *J Med Food*. 2014;17(9):985–995. doi:10.1089/jmf.2013.2999
46. Park B, Hwang E, Seo SA, Cho JG, Yang JE, Yi TH. Eucalyptus globulus extract protects against UVB-induced photoaging by enhancing collagen synthesis via regulation of TGF-β/smad signals and attenuation of AP-1. *Arch Biochem Biophys*. 2018;637:31–39. doi:10.1016/j.abb.2017.11.007
47. Chiang HM, Chen HC, Lin TJ, Shih IC, Wen KC. Michelia alba extract attenuates UVB-induced expression of matrix metalloproteinases via MAP kinase pathway in human dermal fibroblasts. *Food Chem Toxicol*. 2012;50(12):4260–4269. doi:10.1016/j.fct.2012.08.018
48. Adil MD, Kaiser P, Satti NK, Zargar AM, Vishwakarma RA, Tasduq SA. Effect of emblica officinalis (fruit) against UVB-induced photo-aging in human skin fibroblasts. *J Ethnopharmacol*. 2010;132(1):109–114. doi:10.1016/j.jep.2010.07.047
49. Bae JS, Han M, Shin HS, et al. Perilla frutescens leaves extract ameliorates ultraviolet radiation-induced extracellular matrix damage in human dermal fibroblasts and hairless mice skin. *J Ethnopharmacol*. 2017;195:334–342. doi:10.1016/j.jep.2016.11.039
50. Gao W, Wang YS, Hwang E, et al. Rubus idaeus L. (red raspberry) blocks UVB-induced MMP production and promotes type I procollagen synthesis via inhibition of MAPK/AP-1, NF-κβ and stimulation of TGF-β/Smad, Nrf2 in normal human dermal fibroblasts. *J Photochem Photobiol B*. 2018;185:241–253. doi:10.1016/j.jphotobiol.2018.06.007
51. Hwang E, Gao W, Xiao YK, Ngo HTT, Yi TH. Helianthus annuus L. flower prevents UVB-induced photodamage in human dermal fibroblasts by regulating the MAPK/AP-1, NFAT, and Nrf2 signaling pathways. *J Cell Biochem*. 2019;120(1):601–612. doi:10.1002/jcb.27417
52. Wen KC, Fan PC, Tsai SY, Shih IC, Chiang HM. Ixora parviflora protects against UVB-induced photoaging by inhibiting the expression of MMPs, MAP kinases, and COX-2 and by promoting type i procollagen synthesis. *Evid Based Complement Alternat Med*. 2012;2012:417346. doi:10.1155/2012/417346
53. Hwang E, Park SY, Yin CS, Kim HT, Kim YM, Yi TH. Antiaging effects of the mixture of Panax ginseng and Crataegus pinnatifida in human dermal fibroblasts and healthy human skin. *J Ginseng Res*. 2017;41(1):69–77. doi:10.1016/j.jgr.2016.01.001
54. Kim HM, Lee DE, Park SD, et al. Preventive effect of fermented Gelidium amansii and Cirsium japonicum extract mixture against UVB-induced skin photoaging in hairless mice. *Food Sci Biotechnol*. 2014;23(2):623–631. doi:10.1007/s10068-014-0085-5
55. Im AR, Seo YK, Cho SH, Kh O, Kim KM, Chae S. Clinical evaluation of the safety and efficacy of a timosaponin A-III-based antiwrinkle agent against skin aging. *J Cosmet Dermatol*. 2020;19(2):423–436. doi:10.1111/jocd.13035
56. Oh SJ, Kim K, Lim CJ. Suppressive properties of ginsenoside Rb2, a protopanaxadiol-type ginseng saponin, on reactive oxygen species and matrix metalloproteinase-2 in UV-B-irradiated human dermal keratinocytes. *Biosci Biotechnol Biochem*. 2015;79(7):1075–1081. doi:10.1080/09168451.2015.1020752
57. Yanti A, Hwang JK, Hwang J-K. Effects of macelignan isolated from myristica fragrans houtt. on UVB-induced matrix metalloproteinase-9 and cyclooxygenase-2 in HaCaT cells. *J Dermatol Sci*. 2010;57(2):114–122. doi:10.1016/j.jdermsci.2009.10.005
58. Lim SH, Jung SK, Byun S, et al. Luteolin suppresses UVB-induced photoageing by targeting JNK1 and p90 RSK2. *J Cell Mol Med*. 2013;17(5):672–680. doi:10.1111/jcmm.12050
59. Soo Lee Y, Jin DQ, Beak SM, Lee ES, Kim JA. Inhibition of ultraviolet-A-modulated signaling pathways by asiatic acid and ursolic acid in HaCaT human keratinocytes. *Eur J Pharmacol*. 2003;476(3):173–178. doi:10.1016/S0014-2999(03)02177-0
60. Wang ML, Zhong QY, Lin BQ, et al. Andrographolide sodium bisulfate attenuates UV-induced photo-damage by activating the keap1/Nrf2 pathway and downregulating the NF-κB pathway in HaCaT keratinocytes. *Int J Mol Med*. 2020;45(2):343–352. doi:10.3892/ijmm.2019.4415
61. Jang M, Cho C, Han A, et al. MMP-1 suppressing activity of high-molecular weight fraction isolated from the roots of Cynanchum wilfordii Hemsley. *J Funct Foods*. 2018;40:329–335. doi:10.1016/j.jff.2017.11.023
62. Ha SJ, Lee J, Kim H, et al. Preventive effect of Rhus javanica extract on UVB-induced skin inflammation and photoaging. *J Funct Foods*. 2016;27:589–599. doi:10.1016/j.jff.2016.10.011
63. Choi HK, Kim DH, Kim JW, Ngadiran S, Sarmidi MR, Park CS. Labisia pumila extract protects skin cells from photoaging caused by UVB irradiation. *J Biosci Bioeng*. 2010;109(3):291–296. doi:10.1016/j.jbiosc.2009.08.478
64. Jeong D, Lee J, Jeong SG, et al. Artemisia asiatica ethanol extract exhibits anti-photoaging activity. *J Ethnopharmacol*. 2018;220:57–66. doi:10.1016/j.jep.2018.03.037
65. Hu S, Huang J, Pei S, et al. Ganoderma lucidum polysaccharide inhibits UVB-induced melanogenesis by antagonizing cAMP/PKA and ROS/MAPK signaling pathways. *J Cell Physiol*. 2019;234(5):7330–7340. doi:10.1002/jcp.27492
66. Jiang L, Huang J, Lu J, et al. Ganoderma lucidum polysaccharide reduces melanogenesis by inhibiting the paracrine effects of keratinocytes and fibroblasts via IL-6/STAT3/FGF2 pathway. *J Cell Physiol*. 2019;234(12):22799–22808. doi:10.1002/jcp.28844
67. Nam JH, Nam DY, Lee DU. Valencene from the rhizomes of Cyperus rotundus inhibits skin photoaging-related ion channels and UV-induced melanogenesis in B16F10 melanoma cells. *J Nat Prod*. 2016;79(4):1091–1096. doi:10.1021/acs.jnatprod.5b01127
68. Nozza E, Melzi G, Marabini L, et al. Fruit extract prevents UV-A-induced genotoxicity and oxidative injury in human microvascular endothelial cells. *Antioxidants*. 2020;9(4):292. doi:10.3390/antiox9040292
69. Lee CK, Han SS, Mo YK, et al. Prevention of ultraviolet radiation-induced suppression of accessory cell function of Langerhans cells by aloe vera gel components. *Immunopharmacology*. 1997;37(2–3):153–162. doi:10.1016/S0162-3109(97)00042-8
70. Li YH, Wu Y, Wei HC, et al. Protective effects of green tea extracts on photoaging and photommunosuppression. *Skin Res Technol*. 2009;15(3):338–345. doi:10.1111/j.1600-0846.2009.00370.x
71. Liu CS, Nam TG, Han MW, et al. Protective effect of detoxified Rhus verniciflua stokes on human keratinocytes and dermal fibroblasts against oxidative stress and identification of the bioactive phenolics. *Biosci Biotechnol Biochem*. 2013;77(8):1682–1688. doi:10.1271/bbb.130236

72. Botten D, Fugallo G, Fraternali F, Molteni C. A computational exploration of the interactions of the green tea polyphenol (-)-Epigallocatechin 3-Gallate with cardiac muscle troponin C. *PLoS One*. 2013;8(7):e70556. doi:10.1371/journal.pone.0070556
73. San AM, Thongpraditchote S, Sithisarn P, Gritsanapan W. Total phenolics and total flavonoids contents and hypnotic effect in mice of ziziphus mauritiana lam. seed extract. *Evid Based Complement Alternat Med*. 2013;2013:835854. doi:10.1155/2013/835854
74. Fiuza SM, Gomes C, Teixeira LJ, et al. Phenolic acid derivatives with potential anticancer properties--a structure-activity relationship study. part 1: methyl, propyl and octyl esters of caffeic and gallic acids. *Bioorg Med Chem*. 2004;12(13):3581–3589. doi:10.1016/j.bmc.2004.04.026
75. Russell LH, Mazzio E, Badisa RB, et al. Autoxidation of gallic acid induces ROS-dependent death in human prostate cancer LNCaP cells. *Anticancer Res*. 2012;32(5):1595–1602.
76. Ikeda K, Sakai Y, Nagase H. Inhibitory effect of magnolol on tumour metastasis in mice. *Phytother Res*. 2003;17(8):933–937. doi:10.1002/ptr.1264
77. Park J, Lee J, Jung E, et al. In vitro antibacterial and anti-inflammatory effects of honokiol and magnolol against Propionibacterium sp. *Eur J Pharmacol*. 2004;496(1–3):189–195. doi:10.1016/j.ejphar.2004.05.047
78. Katiyar SK, Afaq F, Perez A, Mukhtar H. Green tea polyphenol (-)-epigallocatechin-3-gallate treatment of human skin inhibits ultraviolet radiation-induced oxidative stress. *Carcinogenesis*. 2001;22(2):287–294. doi:10.1093/carcin/22.2.287
79. Tobi SE, Gilbert M, Paul N, McMillan TJ. The green tea polyphenol, epigallocatechin-3-gallate, protects against the oxidative cellular and genotoxic damage of UVA radiation. *Int, J, Cancer*. 2002;102(5):439–444. doi:10.1002/ijc.10730
80. Jin CJ, Yu SH, Wang XM, et al. The effect of lithospermic acid, an antioxidant, on development of diabetic retinopathy in spontaneously obese diabetic rats. *PLoS One*. 2014;9(6):e98232. doi:10.1371/journal.pone.0098232
81. Tzen JT, Jinn TR, Chen YC, et al. Magnesium lithospermate B possesses inhibitory activity on Na+, K+-ATPase and neuroprotective effects against ischemic stroke. *Acta Pharmacol Sin*. 2007;28(5):609–615. doi:10.1111/j.1745-7254.2007.00544.x
82. Liu X, Chen R, Shang Y, Jiao B, Huang C. Superoxide radicals scavenging and xanthine oxidase inhibitory activity of magnesium lithospermate B from Salvia miltiorrhiza. *J Enzyme Inhib Med Chem*. 2009;24(3):663–668. doi:10.1080/14756360802323829
83. Moon HI, Lee J, Chung JH. The effect of erythrodiol-3-acetate on the expressions of matrix metalloproteinase-1 and type-1 procollagen caused by ultraviolet irradiated cultured primary old aged human skin fibroblasts. *Phytomedicine*. 2006;13(9–10):707–711. doi:10.1016/j.phymed.2005.10.001
84. Gray AM, Flatt PR. Insulin-releasing and insulin-like activity of the traditional anti-diabetic plant Coriandrum sativum (coriander). *Br J Nutr*. 1999;81(3):203–209. doi:10.1017/S0007114599000392
85. Lal AA, Kumar T, Murthy PB, Pillai KS. Hypolipidemic effect of Coriandrum sativum L. in triton-induced hyperlipidemic rats. *Indian J Exp Biol*. 2004;42(9):909–912.
86. Cortés-Eslava J, Gómez-Arroyo S, Villalobos-Pietrini R, Espinosa-Aguirre JJ. Antimutagenicity of coriander (Coriandrum sativum) juice on the mutagenesis produced by plant metabolites of aromatic amines. *Toxicol Lett*. 2004;153(2):283–292. doi:10.1016/j.toxlet.2004.05.011
87. Pawar PL, Nabar BM. Effect of plant extracts formulated in different ointment bases on MDR strains. *Indian J Pharm Sci*. 2010;72(3):397–401. doi:10.4103/0250-474X.70494
88. Takasaki M, Konoshima T, Fujitani K, et al. Inhibitors of skin-tumor promotion. VIII. Inhibitory effects of euglobals and their related compounds on Epstein-Barr virus activation. (1). *Chem Pharm Bull*. 1990;38(10):2737–2739. doi:10.1248/cpb.38.2737
89. Maccioni AM, Anchisi C, Sanna A, Sardu C, Dessì S. Preservative systems containing essential oils in cosmetic products. *Int J Cosmet Sci*. 2002;24(1):53–59. doi:10.1046/j.0412-5463.2001.00113.x
90. Shang C, Yaoming H, Deng C, Hu K. Rapid determination of volatile constituents of Michelia alba flowers by gas chromatography-mass spectrometry with solid-phase microextraction. *J Chromatogr A*. 2002;942(1–2):283–288. doi:10.1016/S0021-9673(01)01382-6
91. Asaruddin MR, Honda G, Tsubouchi A, Nakajima-Shimada J, Aoki T, Kiuchi F. Trypanocidal constituents from Michelia alba. *Nat Med*. 2003;57(2):61–63.
92. Tasduq SA, Kaisar P, Gupta DK, et al. Protective effect of a 50% hydroalcoholic fruit extract of Emblica officinalis against anti-tuberculosis drugs induced liver toxicity. *Phytother Res*. 2005;19(3):193–197. doi:10.1002/ptr.1631
93. Vuorela S, Kreander K, Karonen M, et al. Preclinical evaluation of rapeseed, raspberry, and pine bark phenolics for health related effects. *J Agric Food Chem*. 2005;53(15):5922–5931. doi:10.1021/jf050554r
94. Danby SG, AlEnezi T, Sultan A, et al. Effect of olive and sunflower seed oil on the adult skin barrier: implications for neonatal skin care. *Pediatr Dermatol*. 2013;30(1):42–50. doi:10.1111/j.1525-1470.2012.01865.x
95. Msika P, De Belilovsky C, Piccardi N, Chebassier N, Baudouin C, Chadoutaud B. New emollient with topical corticosteroid-sparing effect in treatment of childhood atopic dermatitis: SCORAD and quality of life improvement. *Pediatr Dermatol*. 2008;25(6):606–612. doi:10.1111/j.1525-1470.2008.00783.x
96. Soobrattee MA, Bahorun T, Neergheen VS, Googoolye K, Aruoma OI. Assessment of the content of phenolics and antioxidant actions of the Rubiaceae, Ebenaceae, Celastraceae, Erythroxylaceae and Sterculaceae families of Mauritian endemic plants. *Toxicol in vitro*. 2008;22(1):45–56. doi:10.1016/j.tiv.2007.07.012
97. Torey A, Sasidharan S, Latha LY, Sudhakaran S, Ramanathan S. Antioxidant activity and total phenolic content of methanol extracts of Ixora coccinea. *Pharm Biol*. 2010;48(10):1119–1123. doi:10.3109/13880200903490505
98. Wen KC, Chiu HH, Fan PC, et al. Antioxidant activity of Ixora parviflora in a cell/cell-free system and in UV-exposed human fibroblasts. *Molecules*. 2011;16(7):5735–5752. doi:10.3390/molecules16075735
99. Park HJ, Kim DH, Park SJ, Kim JM, Ryu JH. Ginseng in traditional herbal prescriptions. *J Ginseng Res*. 2012;36(3):225–241. doi:10.5142/jgr.2012.36.3.225
100. Hong CE, Lyu SY. Anti-inflammatory and anti-oxidative effects of Korean red ginseng extract in human keratinocytes. *Immune Netw*. 2011;11(1):42–49. doi:10.4110/in.2011.11.1.42
101. Kang TH, Park HM, Kim YB, et al. Effects of red ginseng extract on UVB irradiation-induced skin aging in hairless mice. *J Ethnopharmacol*. 2009;123(3):446–451. doi:10.1016/j.jep.2009.03.022
102. Kao ES, Wang CJ, Lin WL, Chu CY, Tseng TH. Effects of polyphenols derived from fruit of Crataegus pinnatifida on cell transformation, dermal edema and skin tumor formation by phorbol ester application. *Food Chem Toxicol*. 2007;45(10):1795–1804. doi:10.1016/j.fct.2007.03.016

103. Huang XX, Liu QB, Wu J, et al. Antioxidant and tyrosinase inhibitory effects of neolignan glycosides from Crataegus pinnatifida seeds. *Planta Med*. 2014;80(18):1732–1738. doi:10.1055/s-0034-1383253
104. Hannan A, Kang JY, Hong YK, et al. The marine alga gelidium amansii promotes the development and complexity of neuronal cytoarchitecture. *Phytother Res*. 2013;27(1):21–29. doi:10.1002/ptr.4684
105. Yin J, Heo SI, Wang MH. Antioxidant and antidiabetic activities of extracts from Cirsium japonicum roots. *Nutr Res Pract*. 2008;2(4):247–251. doi:10.4162/nrp.2008.2.4.247
106. Miura T, Ichiki H, Iwamoto N, et al. Antidiabetic activity of the rhizoma of Anemarrhena asphodeloides and active components, mangiferin and its glucoside. *Biol Pharm Bull*. 2001;24(9):1009–1011. doi:10.1248/bpb.24.1009
107. Wang Y, Dan Y, Yang D, et al. The genus anemarrhena bunge: a review on ethnopharmacology, phytochemistry and pharmacology. *J Ethnopharmacol*. 2014;153(1):42–60. doi:10.1016/j.jep.2014.02.013
108. Jin DQ, Lim CS, Hwang JK, Ha I, Han JS. Anti-oxidant and anti-inflammatory activities of macelignan in murine hippocampal cell line and primary culture of rat microglial cells. *Biochem Biophys Res Commun*. 2005;331(4):1264–1269. doi:10.1016/j.bbrc.2005.04.036
109. Chung JY, Choo JH, Lee MH, Hwang JK. Anticariogenic activity of macelignan isolated from myristica fragrans (nutmeg) against streptococcus mutans. *Phytomedicine*. 2006;13(4):261–266. doi:10.1016/j.phymed.2004.04.007
110. Sohn JH, Han KL, Choo JH, Hwang JK. Macelignan protects HepG2 cells against tert-butylhydroperoxide-induced oxidative damage. *Biofactors*. 2007;29(1):1–10. doi:10.1002/biof.5520290101
111. Miean KH, Mohamed S. Flavonoid (myricetin, quercetin, kaempferol, luteolin, and apigenin) content of edible tropical plants. *J Agric Food Chem*. 2001;49(6):3106–3112. doi:10.1021/jf000892m
112. Romanová D, Vachálková A, Cipák L, Ovesná Z, Rauko P. Study of antioxidant effect of apigenin, luteolin and quercetin by DNA protective method. *Neoplasma*. 2001;48(2):104–107.
113. Yang SF, Yang WE, Chang HR, Chu SC, Hsieh YS. Luteolin induces apoptosis in oral squamous cancer cells. *J Dent Res*. 2008;87(4):401–406. doi:10.1177/154405910808700413
114. Seelinger G, Merfort I, Schempp CM. Anti-oxidant, anti-inflammatory and anti-allergic activities of luteolin. *Planta Med*. 2008;74 (14):1667–1677. doi:10.1055/s-0028-1088314
115. Shukla A, Rasik AM, Jain GK, Shankar R, Kulshrestha DK, Dhawan BN. In vitro and in vivo wound healing activity of asiaticoside isolated from Centella asiatica. *J Ethnopharmacol*. 1999;65(1):1–11. doi:10.1016/S0378-8741(98)00141-X
116. Huang MT, Ho CT, Wang ZY, et al. Inhibition of skin tumorigenesis by rosemary and its constituents carnosol and ursolic acid. *Cancer Res*. 1994;54(3):701–708.
117. Cha HJ, Bae SK, Lee HY, et al. Anti-invasive activity of ursolic acid correlates with the reduced expression of matrix metalloproteinase-9 (MMP-9) in HT1080 human fibrosarcoma cells. *Cancer Res*. 1996;56(10):2281–2284.
118. Lu H, Zhang XY, Wang YQ, Zheng XL, Yin-Zhao Xing WM, Zhang Q. Andrographolide sodium bisulfate-induced apoptosis and autophagy in human proximal tubular endothelial cells is a ROS-mediated pathway. *Environ Toxicol Pharmacol*. 2014;37(2):718–728. doi:10.1016/j.etap.2014.01.019
119. Liang E, Liu X, Du Z, Yang R, Zhao Y. Andrographolide ameliorates diabetic cardiomyopathy in mice by blockage of oxidative damage and NF- κ B-Mediated inflammation. *Oxid Med Cell Longev*. 2018;2018(1):9086747. doi:10.1155/2018/9086747
120. Lee HS, Choi JH, Kim YE, Kim IH, Kim BM, Lee CH. Effects of the cynanchum wilfordii ethanol extract on the serum lipid profile in hypercholesterolemic rats. *Prev Nutr Food Sci*. 2013;18(3):157–162. doi:10.3746/pnf.2013.18.3.157
121. Choi DH, Lee YJ, Oh HC, et al. Improved endothelial dysfunction by cynanchum wilfordii in apolipoprotein E(-/-) mice fed a high fat/cholesterol diet. *J Med Food*. 2012;15(2):169–179. doi:10.1089/jmf.2010.1222
122. Shan L, Liu RH, Shen YH, et al. Gastroprotective effect of a traditional Chinese herbal drug "Baishouwu" on experimental gastric lesions in rats. *J Ethnopharmacol*. 2006;107(3):389–394. doi:10.1016/j.jep.2006.03.022
123. Lee K, Kim J, Lee BJ, Park JW, Leem KH, Bu Y. Protective effects of Galla Rhois, the excrescence produced by the sumac aphid, schlechtendalia chinensis, on transient focal cerebral Ischemia in the rat. *J Insect Sci*. 2012;12(10):10. doi:10.1673/031.012.0110
124. Choi JG, Kang OH, Lee YS, et al. Antibacterial activity of methyl gallate isolated from Galla Rhois or carvacrol combined with nalidixic acid against nalidixic acid resistant bacteria. *Molecules*. 2009;14(5):1773–1780. doi:10.3390/molecules14051773
125. Ahn YJ, Lee HS, Oh HS, Kim HT, Lee YH. Antifungal activity and mode of action of Galla rhois-derived phenolics against phytopathogenic fungi. *Pestic Biochem Physiol*. 2005;81(2):105–112. doi:10.1016/j.pestbp.2004.10.003
126. Zaizuhana S, Puteri J, Noor MB, et al. The in vivo rodent micronucleus assay of Kacip Fatimah (Labisia pumila) extract. *Trop Biomed*. 2006;23 (2):214–219.
127. Wan Ezumi MF, Siti Amrah S, Suhaimi AWM, Mohsin SSJ. Evaluation of the female reproductive toxicity of aqueous extract of labisia pumila var. alata in rats. *Indian J Pharmacol*. 2007;39(1):30–32. doi:10.4103/0253-7613.30760
128. Park SW, Oh TY, Kim YS, et al. Artemisia asiatica extracts protect against ethanol-induced injury in gastric mucosa of rats. *J Gastroenterol Hepatol*. 2008;23(6):976–984. doi:10.1111/j.1440-1746.2008.05333.x
129. Hajdú Z, Hohmann J, Forgo P, Máthé I, Molnár J, Zupkó I. Antiproliferative activity of Artemisia asiatica extract and its constituents on human tumor cell lines. *Planta Med*. 2014;80(18):1692–1697. doi:10.1055/s-0034-1383146
130. Jeong D, Yi YS, Sung GH, et al. Anti-inflammatory activities and mechanisms of Artemisia asiatica ethanol extract. *J Ethnopharmacol*. 2014;152(3):487–496. doi:10.1016/j.jep.2014.01.030
131. Tie L, Yang HQ, An Y, et al. Ganoderma lucidum polysaccharide accelerates refractory wound healing by inhibition of mitochondrial oxidative stress in type 1 diabetes. *Cell Physiol Biochem*. 2012;29(3–4):583–594. doi:10.1159/000338512
132. Chan WK, Cheung CC, Law HK, Lau YL, Chan GC. Ganoderma lucidum polysaccharides can induce human monocytic leukemia cells into dendritic cells with immuno-stimulatory function. *J Hematol Oncol*. 2008;1:9. doi:10.1186/1756-8722-1-9
133. Chang CJ, Lin CS, Lu CC, et al. Ganoderma lucidum reduces obesity in mice by modulating the composition of the gut microbiota. *Nat Commun*. 2015;6(1):7489. doi:10.1038/ncomms8489
134. Ardestani A, Yazdanparast R. Cyperus rotundus suppresses AGE formation and protein oxidation in a model of fructose-mediated protein glycoxidation. *Int J Biol Macromol*. 2007;41(5):572–578. doi:10.1016/j.ijbiomac.2007.07.014

135. Kilani-Jaziri S, Neffati A, Limem I, et al. Relationship correlation of antioxidant and antiproliferative capacity of Cyperus rotundus products towards K562 erythroleukemia cells. *Chem Biol Interact*. 2009;181(1):85–94. doi:10.1016/j.cbi.2009.04.014
136. Niu K, Guo H, Guo Y, et al. Royal jelly prevents the progression of sarcopenia in aged mice in vivo and in vitro. *J Gerontol a Biol Sci Med Sci*. 2013;68(12):1482–1492. doi:10.1093/gerona/glt041
137. Asgarpanah J, Saati S. An overview on phytochemical and pharmacological properties of Rhus coriaria L. *Res J Pharmacogn*. 2014;1(3):47–54.
138. Strickland FM, Pelley RP, Kripke ML. Prevention of ultraviolet radiation-induced suppression of contact and delayed hypersensitivity by Aloe barbadensis gel extract. *J Invest Dermatol*. 1994;102(2):197–204. doi:10.1111/1523-1747.ep12371762
139. Yang CS, Maliakal P, Meng X. Inhibition of carcinogenesis by tea. *Annu Rev Pharmacol Toxicol*. 2002;42(1):25–54. doi:10.1146/annurev.pharmtox.42.082101.154309
140. Hwang E, Kim SH, Lee S, et al. A comparative study of baby immature and adult shoots of Aloe vera on UVB-induced skin photoaging in vitro. *Phytother Res*. 2013;27(12):1874–1882. doi:10.1002/ptr.4943
141. Kligman AM. Early destructive effect of sunlight on human skin. *JAMA*. 1969;210(13):2377–2380. doi:10.1001/jama.1969.03160390039008
142. Hruza LL, Pentland AP. Mechanisms of UV-induced inflammation. *J Invest Dermatol*. 1993;100(Suppl):S35–S41. doi:10.1038/jid.1993.21
143. Tsukahara K, Moriwaki S, Hotta M, et al. The effect of sunscreen on skin elastase activity induced by ultraviolet-A irradiation. *Biol Pharm Bull*. 2005;28(12):2302–2307. doi:10.1248/bpb.28.2302
144. Peres PS, Terra VA, Guarnier FA, Cecchini R, Cecchini AL. Photoaging and chronological aging profile: understanding oxidation of the skin. *J Photochem Photobiol B*. 2011;103(2):93–97. doi:10.1016/j.jphotobiol.2011.01.019
145. Svobodova A, Walterova D, Vostalova J. Ultraviolet light induced alteration to the skin. *Biomed Pap*. 2006;150(1):25–35. doi:10.5507/bp.2006.003
146. Imokawa G. Mechanism of UVB-induced wrinkling of the skin: paracrine cytokine linkage between keratinocytes and fibroblasts leading to the stimulation of elastase. *J Invest Dermatol Symp Proc*. 2009;14(1):36–43. doi:10.1038/jidsymp.2009.11
147. Lin RF, Feng XX, Li CW, et al. Prevention of UV radiation-induced cutaneous photoaging in mice by topical administration of patchouli oil. *J Ethnopharmacol*. 2014;154(2):408–418. doi:10.1016/j.jep.2014.04.020
148. Sundar M, Suresh S, Lingakumar K. Influence of caralluma adscendens var. attenuata cold cream on UV-B damaged skin epidermal cells: a novel approach. *Biotech*. 2021;11(6):1–16.
149. Lee CW, Ko HH, Lin CC, Chai CY, Chen WT, Yen FL. Artocarpin attenuates ultraviolet B-induced skin damage in hairless mice by antioxidant and anti-inflammatory effect. *Food Chem Toxicol*. 2013;60:123–129. doi:10.1016/j.fct.2013.07.029
150. He H, Li A, Li S, Tang J, Li L, Xiong L. Natural components in sunscreens: topical formulations with sun protection factor (SPF). *Biomed Pharmacother*. 2020;134:111161. doi:10.1016/j.biopha.2020.111161
151. Chanchal D, Swarnlata S. Herbal photoprotective formulations and their evaluation. *Open Nat Prod J*. 2009;2(1):71–76. doi:10.2174/1874848100902010071