

REVIEW

Pollution, a Relevant Exposome Factor in Skin Aging and the Role of Multi-benefit Photoprotection

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Exposoma;
Contaminación del aire;
Radiación solar;
Anticontaminación

Abstract Skin aging is a complex, continuous, multifactorial process resulting from cumulative morphological and functional changes in the skin over time.

This happens because of two processes: intrinsic and extrinsic skin aging. Intrinsic skin aging occurs naturally over time and reflects each person's genetic makeup, or heredity. Extrinsic skin aging, on the other hand, is due to exposomal factors, such as solar radiation, air pollution, tobacco or nutrition, being the first two the most important of all. Exposure to air pollutants, primarily gases such as ground-level ozone and particulate matter, can accelerate the process via four key mechanisms: reactive oxygen species generation, inflammation, skin microbiome disruption, and aryl hydrocarbon receptor activation. Regarding solar radiation, all wavelengths reaching the Earth's surface have an impact on the skin, having a synergistic effect with air pollution ("photo-pollution"). Here, we discuss this phenomenon and mitigation strategies, including sunscreens, cosmetics with film-forming plus antioxidant ingredients, and oral supplementation.

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La contaminación, un factor relevante del exposoma en el envejecimiento cutáneo y el papel de la fotoprotección multibeneficio

Resumen El envejecimiento cutáneo es un proceso complejo, continuo y multifactorial que resulta de los cambios morfológicos y funcionales acumulados en la piel a lo largo del tiempo. Esto sucede debido a 2 procesos: el envejecimiento cutáneo intrínseco y el extrínseco. El envejecimiento cutáneo intrínseco ocurre de forma natural con el tiempo, y refleja el trasfondo genético o herencia de cada persona. El envejecimiento cutáneo extrínseco, por otro lado, es causado por factores del exposoma, como la radiación solar, la contaminación atmosférica, el tabaco o la nutrición, siendo los 2 primeros los más importantes. La exposición a contaminantes

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atmosféricos, principalmente gases como el ozono troposférico y las partículas en suspensión, puede acelerar el proceso a través de 4 mecanismos clave: generación de especies reactivas de oxígeno, inflamación, alteración del microbioma cutáneo y activación del receptor de hidrocarburos arílicos. En cuanto a la radiación solar, todas las longitudes de onda que llegan a la superficie de la Tierra tienen un impacto en la piel, teniendo un efecto sinérgico con la contaminación atmosférica («fotocontaminación»). Aquí analizamos este fenómeno y las estrategias de mitigación, que incluyen protectores solares, cosméticos con ingredientes formadores de película, y antioxidantes y suplementos orales.

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Introduction

Aging is an unavoidable physiological phenomenon, characterized by a progressive decline in the biological function of cells and organs.¹ It is influenced by genetics (intrinsic aging) and the exposure to exposomal factors such as solar radiation, air pollution, nutrition, lifestyle, and others (extrinsic aging).² Molecular epidemiologist Christopher Wild coined the term “exposome” in 2005 to represent the totality of exposures an individual experiences from conception to death.³ Miller and Jones further refined it in 2014, proposing that the exposome should be considered as the total of environmental factors that interact with an individual throughout life, and the biological response to these factors.⁴

The skin is the first defensive barrier of the body and is in continuous contact with the environment.⁵ It undergoes significant changes due to intrinsic and extrinsic aging.⁵ Intrinsic skin aging manifests as fine lines, xerosis, and laxity, whereas extrinsic aging skin shows as coarse wrinkles, irregular pigmentation, and lentigines.² The term photoaging has been used synonymously with extrinsic skin aging to indicate the high impact of solar radiation on the aging process.² However, many other factors contribute to extrinsic skin aging such as air pollution, climate, tobacco and nutrition.^{5,6} Added to their individual effects, these environmental factors also interact with each other in a complex manner.² Air pollution and solar radiation are the main factors responsible for skin aging. They accelerate skin aging mainly via oxidative stress. This occurs when there is an imbalance between the production of reactive oxygen species (ROS) and the body’s capacity to neutralize them, which ultimately results in ROS accumulation.^{6–8} The best defense vs age-related changes in the skin is protection vs exposomal factors.⁹ Urban life exposes people to heavy air pollution created by vehicles, industry, construction activities, and others. The main air pollutants that induce skin damage are particulate matter (PM) and gases such as ground-level ozone (O₃). The skin damage caused by these air pollutants is further augmented by solar radiation (“photo-pollution”). The combination of ultraviolet radiation (UVR) plus PM and ozone exposure has a synergistic effect increasing the oxidative stress and inflammation.¹⁰ Their harmful effects can be mitigated by fighting them: passively by creating a physical barrier on the skin surface to reduce contact with air pollutants (film-forming ingredients

and actively by counteracting oxidative stress (antioxidants) and absorbing, reflecting and/or scattering solar radiation (sun filters).^{11,12}

Therefore, the use of products that protect from solar radiation and air pollution has become important as a part of daily skincare.¹³ This review focuses on the effects of air pollution and solar radiation on skin aging and how we can counter them with cosmetics and oral supplements.

Methods

We conducted a literature narrative review. GoogleScholar and PubMed searches from November 1997 through June 2024 using the keywords “exposome”, “pollution”, “air pollution”, “solar radiation”, “sunlight”, “ultraviolet radiation”, “visible light”, “blue light”, “infrared radiation”, “sunscreens”, “photoprotection”, “oral photoprotection”, “oral supplements”, “skin aging”, “hyperpigmentation”, “skin cancer”, “cosmetics”, “dermocosmetics”, “film-forming” and “antioxidant”, were performed. The search strategy, restricted to English and Spanish language articles, included meta-analyses, observational studies, clinical trials, and review articles.

Results and discussion

Air pollution and solar radiation effects on skin aging

Skin exposome

The skin exposome includes external and internal factors, their interactions, and the response of the human body to these factors leading to biological and clinical signs of skin aging.⁶ However, studying the exposome in its entirety is challenging because it is highly variable and dynamic.¹⁴ The exposome factors of skin aging can be broadly categorized as solar radiation: UVR, visible light (VL), and infrared (IR) radiation; air pollution; tobacco; nutrition, cosmetics, and other lesser-known factors.⁶ Most factors of skin exposome are solar radiation and air pollution. Biological responses to exposome factors can cause physiological adaptations, such as metabolic changes, protein modifications, DNA mutations and others.¹⁵ Most skin disorders are highly influenced by exposure to exposome factors.¹⁴

Effects of air pollution on skin aging

Air pollutants that can cause extrinsic skin aging mostly include gases such as ground-level ozone, and PM, a complex mixture of solids and liquid particles suspended in the air. PM can be formed in two main ways: directly emitted from a source like vehicle exhaust, construction activities, wildfires (primary PM) or formed in the atmosphere via complex chemical reactions between gas precursors such as volatile organic compounds (VOCs) and the presence of solar radiation (secondary PM).^{16,17} PMs are categorized based on its diameter: PM_{2.5} (fine particles; diameter $\leq 2.5 \mu\text{m}$) and PM₁₀ (coarse particles; diameter $\leq 10 \mu\text{m}$). Air pollutants interact with the body, either via skin contact, inhalation or ingestion.¹⁸ Air pollutants affect the human skin through four different ways: generating ROS, triggering inflammation, impacting skin microbiota, and activating hydrocarbon receptor (AhR).¹⁹ The activation of AhR – a transcription factor – can lead to hyperpigmentation, wrinkles, skin cancer and worsening of several dermatoses such as acne, atopic dermatitis and psoriasis.¹¹

Exposure to ozone causes changes in the stratum corneum (SC), the outermost layer of the epidermis. It reduces SC-associated antioxidants, leading to an oxidative stress response. This oxidative stress response reaches the deeper layers of the skin, including the dermis, affecting collagen metabolism, leading to wrinkle formation.²⁰

Repeated exposure of the skin to PM even at non-toxic concentrations increases the production of ROS, which in turn leads to the secretion of cytokines and cellular dysfunction. This can break down the lipid barrier of the epidermis, decrease the number of cutaneous microbial species, trigger inflammatory skin diseases, and induce melanogenesis via AhR activation.²¹ A study showed that one-unit increase in PMs was associated with a 20% increase in lentigines on the forehead and cheeks and in wrinkles of the nasolabial fold. The risk of lentigines was higher after increased exposure to PM_{2.5} than after increased exposure to PM₁₀.²² PM_{2.5} are highly lipophilic and penetrate the skin easily vs the PM₁₀ particles that stay at the surface of the skin.²³ Regarding skin cancer, an epidemiological study shows that an increment of $10 \mu\text{g}/\text{m}^3$ PM₁₀ in the air increases 52% relative risk of developing non-melanoma skin cancers.²⁴ AhR may play a role in inducing skin cancer; AhR-deficient mice exposed to PM did not develop keratinocyte carcinomas whereas those AhR positive developed keratinocyte carcinomas via CYP1A1 expression.²⁵

In addition to PM, other air pollutants have been shown to have an impact on skin health.²⁶ Studies indicate that high levels of environmental factors including polycyclic aromatic hydrocarbons (PAHs), VOCs, heavy metals, and gases such as CO, NO_x, SO, and O₃, can impair the skin's barrier function. Both the concentration and duration of exposure are significant. These pollutants are linked to various skin issues, including aging, inflammatory diseases, acne, hair loss, and skin cancers, with responses varying by pollutant characteristics.²⁷

The relationship between pollution and skin aging was evaluated in a cohort of 389 patients aged 30–74 who planned to undergo laser treatment. Researchers used the VISIA Complexion Analysis System to assess skin conditions alongside levels of various air pollutants, including CO, non-methane hydrocarbons (NMHC), nitrogen oxides (NO, NO₂,

NO_x), PM_{2.5} and PM₁₀, O₃, and SO₂. Findings revealed a strong correlation between NMHC exposure and issues such as skin texture, enlarged pores, and brown spots. Conversely, exposure to ozone was associated with better skin texture and pore scores. Notably, brown spots were negatively associated with several pollutants, particularly in individuals older than 45 years.²⁸

Moreover, we already know that increased NO₂ concentrations characterize traffic-related air pollution. Although the effects of NO₂ on skin health have not been previously explored, the relationship between environmentally induced lung and skin aging prompted an assessment of NO₂ exposure and lentigo development.²⁹ Data from the extended SALIA population and an independent Han Chinese cohort were analyzed. Results indicated a significant correlation between NO₂ exposure and increased lentigines on the cheeks in both cohorts. Specifically, a $10 \mu\text{g}/\text{m}^3$ increase in NO₂ correlated with a 25% increase in lentigines in SALIA participants and a 24% increase in Chinese women >50 years.²⁶

The effects of solar radiation on aging

Solar radiation is the major external factor related to skin aging. Up to 80% of skin aging is caused by sun exposure.³⁰ Sun-exposed areas such as the face and neck appear prematurely aged vs unexposed skin. Clinical features of aging due to sun exposure include wrinkles, dullness, changes in pigmentation, laxity, roughness, and telangiectasia.³¹ Solar radiation that raises earth's surface includes electromagnetic rays of different wavelengths. These include UVB (UVB and UVA), VL, and IR. They arrive at the earth's surface in different proportions and penetrate the skin to different levels. Skin aging occurs due to daily exposure to non-extreme, low doses of these solar waves. The most energetic radiation – UVB (290–315 nm) – only penetrates up to the epidermis. It mainly causes erythema, photo-immunosuppression and cutaneous cancer. Additionally, it increases the production of epidermal matrix metalloproteinase (MMP)-1, which disseminates into the dermis where it breaks down collagen, contributing to skin aging.² UVA (315–400 nm) is the main radiation responsible for photoaging. It penetrates up to the dermis and has direct effects on dermal fibroblasts.^{32,33} Both UVB and UVA contribute to acquired skin pigmentation and skin cancer.³¹ Regarding skin cancer, while UVB causes direct damage to DNA, UVA causes indirect damage to DNA by forming ROS.³⁴ UVR is a recognized carcinogen, responsible for more than 50% of all tumors. It causes almost 65% of malignant melanomas and 90% of keratinocyte carcinomas.³¹ VL (400–700 nm) is the wavelength that can be detected by the human eye. One of the VL wavelengths with more biologic effects is the blue-violet light (BL) also known as high-energy visible light ($\approx 400\text{--}500 \text{ nm}$). It produces ROS and induces MMP-1 expression contributing to skin aging.^{6,35} OPN3 receptors detect BL and increase the synthesis of tyrosinase and dopachrome tautomerase involved in melanogenesis and hyperpigmentation.

The impact of BL is higher in subjects with darker skin tones (ITA $<28^\circ$); hence, protection vs BL is specially recommended in them.^{36,37} IR (700 nm–1 mm) damages the skin; IR-A penetrates deeply, generating mitochondrial ROS,

249 increasing MMP-1, reducing collagen production, promoting
250 angiogenesis, and raising mast cell numbers.³⁸

251 Synergies between solar radiation and air pollution

252 Humans are not exclusively exposed to a single exposome
253 factor. While many studies examine the impact of solar radi-
254 ation or air pollution on the skin of aging individually, few
255 assess their combined effect.³⁰ In this regard, solar radiation
256 converts volatile organic compounds (VOC), found in the air,
257 into secondary organic aerosols that contribute to PM forma-
258 tion, particularly PM_{2.5}.¹⁹ Additionally, the combined effect
259 of UVA radiation and ozone can synergistically induce more
260 oxidative stress in human skin.³⁹

261 In preclinical studies, the combination of UVA and diesel
262 PMs (DPM) induced significant cytotoxic and genotoxic dam-
263 age through the photoactive production of singlet oxygen,
264 which was not observed with either UVA or DPM extracts
265 alone.⁴⁰ Furthermore, other *in vitro* data have demon-
266 strated that PAHs, found in PM, in combination with UVA
267 significantly increases skin damage and aging in the skin.⁴¹
268 UVA along with air pollutants considerably increases the
269 risk of skin cancer.³⁴ Clinically, an analysis of 799 Caucasian
270 women in Germany suggested that facial lentiginosities are the
271 consequence of an interplay between UVR and PMs.⁴² A
272 recent study compared a similar population from two Chi-
273 nese cities located at the same latitude (thus receiving
274 approximately the same sun exposure) but exposed to dif-
275 ferent atmospheric pollution levels. It was found that people
276 living in a higher polluted environment for many years had
277 a higher prevalence of hyperpigmented lesions as well as
278 higher severity of wrinkles.⁴³

279 However, pollution could have in some circumstances a
280 photoprotective effect reducing UV irradiance due to the
281 presence of O₃, NO₂, SO₂ and scattering particulates in
282 the troposphere.⁴⁴ More research is needed to elucidate
283 the mechanisms of UV and pollution interaction.⁴⁵ How-
284 ever, all existing evidence leads to the conclusion that the
285 combination of solar radiation plus air pollution can have
286 synergistic effects increasing their detrimental cutaneous
287 effects. Hence, combined anti-pollution and photoprotec-
288 tive strategies should be implemented.

289 Anti-pollution ingredients

290 The first step to prevent the negative impact of air pol-
291 lutants to the skin is to prevent their deposition and
292 penetration, and the next step is to revert their effects once
293 they have penetrated the skin.⁴⁶ The main approaches to
294 counteract cutaneous negative effects of air pollution are to
295 reinforce the skin's barrier function and increase its antiox-
296 idant stores.²¹ Several anti-pollution ingredients have been
297 proven to be effective in preventing and counteracting the
298 skin damage caused by air pollutants. Some of these actives
299 are added to sunscreens, topical cosmetics, while others can
300 be taken as oral supplements.

301 Anti-pollution cosmetic ingredients

302 Cosmetic ingredients can help protect the skin from air pol-
303 lution mainly by forming a protective film (film-forming),
304 neutralizing ROS (antioxidants) and decreasing AhR expres-

sion. Some active ingredients with scientific evidence are
listed below:

- 305 1. *Film-forming agents*: One novel film-forming agent is
306 *Alteromonas* ferment extract, an exopolysaccharide
307 derived from marine microorganisms. It creates a pro-
308 tective film on the skin, reducing the adherence of PM_{2.5}
309 and improving skin barrier function. A study showed
310 that after 56 days of daily use, *Alteromonas* ferment
311 extract significantly improved skin hydration, firmness,
312 and elasticity.⁴⁷ Other film-forming agents are extracts
313 from *Kappaphycus alvarezii* and *Caesalpinia spinosa*.^{48,49}
- 314 2. *Antioxidant ingredients*: Several categories of ingre-
315 dients can act as antioxidants including vitamins,
316 plant-derived compounds, and microorganism-derived
317 substances.
318 2.1 *Vitamins*: Topical vitamin C is a potent antioxi-
319 dant that increases collagen synthesis and reduces
320 the expression of matrix metalloproteinases (MMPs),
321 increasing collagen levels.⁵⁰ It can reduce pig-
322 mentation by inhibiting tyrosinase activity, pre-
323 vent UV-induced erythema, and maintain skin
324 hydration.⁵⁰ Vitamin E is another important antioxi-
325 dant that decreases MMP-1 levels and reinforces
326 skin barrier.⁵⁰ Topical niacinamide (vitamin B3)
327 has been shown to improve the appearance of
328 aging facial skin by reducing hyperpigmentation,
329 pore size, and redness.^{51,52} It also offers ben-
330 efits in preventing photo-immunosuppression and
331 photo-carcinogenesis. Niacinamide improves skin
332 barrier function, decreases sebum production, and
333 is well tolerated.^{51,52} An *in vitro* study showed
334 that continuous exposure for 3 days to 30 pmol/cm²
335 of benzo(a)pyrene (BaP) and squalene monohy-
336 droperoxides, as surrogate environmental stressors,
337 led to significant reductions in cell viability,
338 increased inflammation, and rise in pigmentation in
339 reconstructed human skin equivalents. Niacinamide
340 pretreatment (5 mmol/L) effectively reduced these
341 effects, likely by supporting NAD⁺-dependent detox-
342 ification pathways.⁵³ Medical literature does not
343 contain studies specifically examining the protec-
344 tive effects of the other vitamins individually vs
345 pollution-related skin aging. However, their capac-
346 ity to mitigate oxidative stress – regardless of the
347 source – suggests that such effects may be inferred.
348 Future research should focus on this area of investi-
349 gation.
- 350 2.2 *Plant-derived compounds*: Ginger oil from
351 *Zingiber officinale* possesses antioxidant and
352 anti-inflammatory properties. It has been shown
353 to reduce UVB-induced erythema and wrinkle
354 formation.⁵⁴ In preclinical studies, *Zingiber mon-
355 tanum* has proven capable of inhibiting ROS
356 formation, increase type I procollagen synthesis and
357 decrease MMP (1, 3 and 9) expression and elastase
358 activity.⁵⁵ Another plant-derived antioxidant active
359 is *Polypodium leucotomos* (PL). An aqueous PL
360 leaf extract reduced oxidative stress and inhibited
361 melanogenic pathway activation in keratinocyte
362 and melanocyte cells exposed to BaP pollutants and
363 UVA.⁵⁶ In a study of cultured keratinocytes exposed
364
365

to PM2.5, PL reduced inflammation and generation of new melanocytes.⁵⁷

2.3 *Microorganism-derived compounds*: Ectoine, a metabolite produced by a halophilic bacterium, reinforces the skin barrier and suppresses inflammatory processes.⁵⁸ In UVA-irradiated keratinocytes, it suppresses α -MSH-stimulated melanogenesis and activates the antioxidant Nrf2 pathways having a skin-whitening effect.⁵⁹ *Porphyridium cruentum*, a red microalga, produces a high amount of sulfated polysaccharides (SPs) that interfere with ROS formation and inhibit elastase activity, preserving elastin and collagen.⁶⁰ *K. alvarezii*, another seaweed extract, has demonstrated antioxidant, photoprotective, and anti-inflammatory properties.⁶¹

2.4 *Other anti-pollution ingredients*: Melatonin acts as an antioxidant by scavenging ROS (direct antioxidant) and increasing the levels of endogenous antioxidant enzymes (indirect antioxidant)⁶²; additionally, it has been demonstrated that topical melatonin 12.5% protect vs UVB-induced erythema.⁶³ Coenzyme Q10 (Q10) is an important endogenous coenzyme whose levels decrease with age and exposure factors exposure. Topical Q10 can penetrate the skin and exert antioxidant effects, benefiting both elderly individuals with Q10 deficiency and people of all ages seeking protection due to oxidative stress.⁶⁴ Moreover, Q10 decreases PM-induced inflammation by enhancing the cellular oxidative status, suppressing pro-inflammatory NF- κ B, and improving the levels of the antioxidant and anti-inflammatory regulators Nrf2 and SIRT1 in human dermal fibroblasts.⁶⁵

3. *Aryl-receptor inhibitors*: Another way to counteract the effect of pollution on the skin is through the blockade of the AhR. *Deschampsia antarctica* is a polyextremophile Gramineae native to Antarctica whose aqueous extract has shown to counteract the pollutant-induced AhR activation in the keratinocytes.⁶⁶ A night cream containing melatonin, carnosine and *Helichrysum italicum* extract reduced the expression levels of AhR by 96% in human skin explants exposed to a mixture of PAH and heavy metals for 1.5 h vs the control group.⁶⁷

Oral supplements that can counteract the effects of air pollution

In addition to cosmetics, oral supplements can also mitigate the harmful effects of exposome. Carotenoids reduce UVA-induced oxidative stress and pigmentation, and UVB-induced erythema.⁷ However, humans cannot synthesize carotenoids on their own and dietary supplementation is necessary.^{55,68,69} Ferulic acid found in tissues of plants has antioxidant and anti-inflammatory effects on the skin.⁶⁹ Green tea is rich in polyphenols which scavenge ROS, enhance immunity and decrease MMPs.⁷⁰ Another useful active ingredient is the extract of the Mesoamerican fern *P. leucotomos* (PL). It has been demonstrated that oral treatment with PL extracts reduced the harmful effects of UVB irradiation, including the appearance of sunburn cells, DNA damage, and inflammation.⁷¹ It also protects vs VL, particularly from

hyperpigmentation.^{72–74} In a study with 22 individuals irradiated with UVB, UVA, and VL, oral PL extract showed suppressive effects on UVB-induced erythema within 2 h of administration.⁷⁵

The mechanism of action and administration route of different anti-pollution ingredients are shown in Table 1.

Multi-benefit photoprotection formulations

Sunscreen is a great tool to prevent and reduce premature skin aging. Given the increasing list of actives that may help counteract the effects of air pollution on the skin, the use of sunscreens enriched with such ingredients are becoming an appealing and useful strategy. Few studies show multi-benefit sunscreens with anti-pollution actives effectively counter solar and pollution damage. Narda et al. evaluated a water-based SPF50 facial anti-pollution and anti-aging sunscreen (WSC) containing hyaluronic acid, palmitoyl tripeptide-38, and pentapeptide-34 trifluoroacetate.¹³ Pre-clinically, WSC protected vs sunburn cell formation and significantly reduced the adherence of PM to the skin (15.2% less than control; $p < 0.01$). Skin treated with WSC showed reduced levels of pro-inflammatory and oxidative stress markers. Clinically, daily use of the product during 28 days significantly decreased the area of brown spots, and count ($p < 0.01$) and increased firmness by 14.1% ($p < 0.01$).¹³ A topical serum of 15% ascorbic acid, 0.5% ferulic acid, and 1% tocopherol prevented skin barrier damage, lipid peroxidation an inflammation induced by PM+UVR in 15 subjects confirming its utility to prevent pollution-induced skin damage.⁷⁶

Oral photoprotection may help mitigate combined pollution and solar effects, but evidence is limited. Marini et al., investigated the efficacy of an oral supplement containing a combination of actives with mainly antioxidant properties (*P. leucotomos*, green tea, lycopene, lutein, and vitamin C, among others) in protecting the skin from sun damage (UVA/UVB) and air pollution particles.⁷⁷ Minimal erythema dose and minimal persistent pigmentation dose were evaluated before and after supplementation in an *in vivo* double-blind placebo controlled study. Oral supplementation provided significant protection vs UVB-induced sunburn and UVA-induced pigmentation in air pollutant exposed skin. The observed reduction in air pollution-induced skin tanning responses is likely due to the antioxidant activity of the actives.⁷⁷ A double-blind randomized, parallel group trial conducted among 100 outdoors workers living in a polluted city showed that the intake of an oral supplement composed by four herbal extracts (*Olea europaea* leaf, *Lippia citriodora*, *Rosmarinus officinalis*, and *Sophora japonica*) reduced wrinkled depth, increased elasticity and firmness, improved skin moisturization and transepidermal water loss and reduced dark spots as early as 2 weeks after being used.⁷⁸

These results suggest that multi-benefit photoprotection formulas, both topical and oral, might contribute to mitigate the negative effects of combined effects of air pollution and solar radiation.

A summary of air pollution and solar radiation cutaneous effects and non-pharmacologic strategies to counteract them is shown in Fig. 1.

Table 1 Mechanism of action and administration route of different anti-pollution ingredients.

Anti-pollution ingredient	Mechanism of action	Route of administration
<i>Alteromonas ferment</i> extract ⁴⁷	<ul style="list-style-type: none"> • Film-forming. Reduces adherence of PM2.5 to the skin • Improves skin barrier function 	Topical
Ginger oil (<i>Zingiber</i> spp.) ⁵⁴	<ul style="list-style-type: none"> • Antioxidant • Anti-inflammatory 	Topical
<i>Zingiber montanum</i> ⁵⁵	<ul style="list-style-type: none"> • Antioxidant • Decreases the activity of elastase • Decreases MMP expression • Increases type I procollagen synthesis 	Topical
Ectoine ⁵²	<ul style="list-style-type: none"> • Antioxidant • Reinforce skin barrier • Protects from UV radiation and environmental hazards • Suppresses inflammatory processes: α-MSH-stimulated melanogenesis • Activates antioxidant Nrf2 pathways 	Topical
<i>Porphyridium cruentum</i> ⁶⁰	<ul style="list-style-type: none"> • Antioxidant • Reinforce skin barrier • Inhibit elastase 	Topical
<i>Kappaphycus alvarezii</i> ⁶¹	<ul style="list-style-type: none"> • Antioxidant • Photoprotective • Anti-inflammatory 	Topical
Coenzyme Q10 ⁶⁴	<ul style="list-style-type: none"> • Antioxidant • Anti-inflammatory 	Topical
<i>Deschampsia antarctica</i> ⁶⁶	<ul style="list-style-type: none"> • AhR inhibitor 	Topical
<i>Helichrysum italicum</i> ⁶⁷	<ul style="list-style-type: none"> • AhR inhibitor 	Topical
Carotenoids ⁷	<ul style="list-style-type: none"> • Antioxidant (reduces UVA-induced oxidative stress, pigmentation and UVB-induced erythema) 	Oral
Ferulic acid ⁶⁹	<ul style="list-style-type: none"> • Antioxidant • Anti-inflammatory 	Oral
Green tea polyphenols (GTPP) ⁷⁰	<ul style="list-style-type: none"> • Antioxidant • Enhance immunity • Decrease MMPs 	Oral
Vitamin C ⁵⁰	<ul style="list-style-type: none"> • Antioxidant • Increases collagen synthesis • Reduces MMP expression • Inhibits tyrosinase 	Topical/oral
Vitamin E ⁵⁰	<ul style="list-style-type: none"> • Antioxidant • Decreases MMP-1 transcription levels 	Topical/oral
Niacinamide (vitamin B3) ⁵²	<ul style="list-style-type: none"> • Antioxidant • Prevents photo-immunosuppression and photo-carcinogen • Prevents the loss of dermal collagen 	Topical/oral
<i>Polypodium leucotomos</i> (PL) ^{71,72,75}	<ul style="list-style-type: none"> • Antioxidant • Anti-inflammatory • Prevents melanogenic signaling pathway activation 	Topical/oral

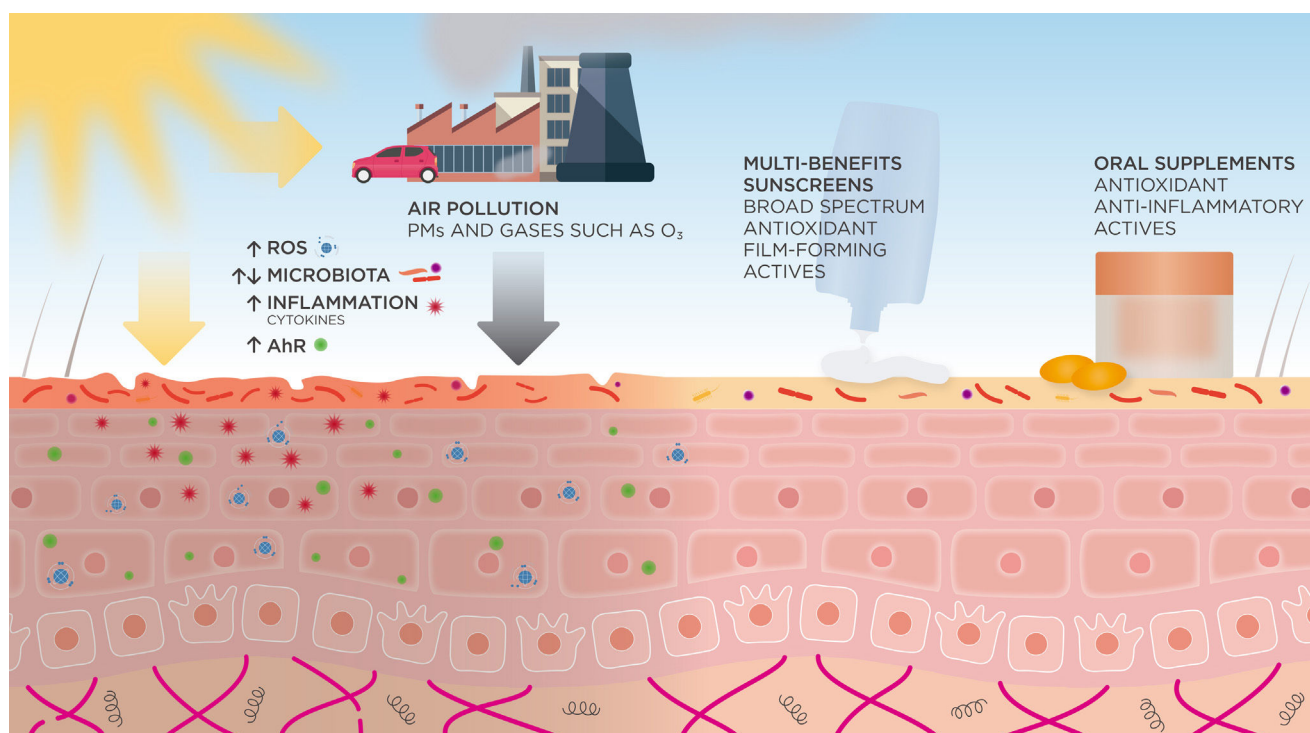


Figure 1 Air pollution and solar radiation effects on skin and non-pharmacologic strategies to counteract them.

Conclusions

The skin easily develops signs of aging, due to accumulating oxidative stress caused by various exposome factors. Among exposomal factors, the maximum damage occurs due to solar radiation and air pollution. We described the effect of air pollution and its synergistic effects with solar radiation on skin aging. It remains to be studied if pollution also interacts with other exposomal factors and whether these potential interactions induce premature aging of the skin. The use of multi-benefit photoprotection (both topical and oral) are promising strategies to counteract the negative effects of exposomal factors. Further investigation is crucial to understand how pollution interacts with other exposomal factors and how to best address this complex interplay for optimal skin health.

CRediT authorship contribution statement

J. Santamaria and L. Prudkin equally contributed to the manuscript drafting and critical revision regarding significant intellectual content. All authors read and approved the manuscript final version.

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Conflicts of interest

J. Santamaria and L. Prudkin are employees at ISDIN. J. Piquero-Casals is a consultant for ISDIN. Y. Gilaberte has received honoraria from ISDIN.

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