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SOLAR RADIATION + AIR POLLUTION
How do they impact skin aging?

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EDITORIAL
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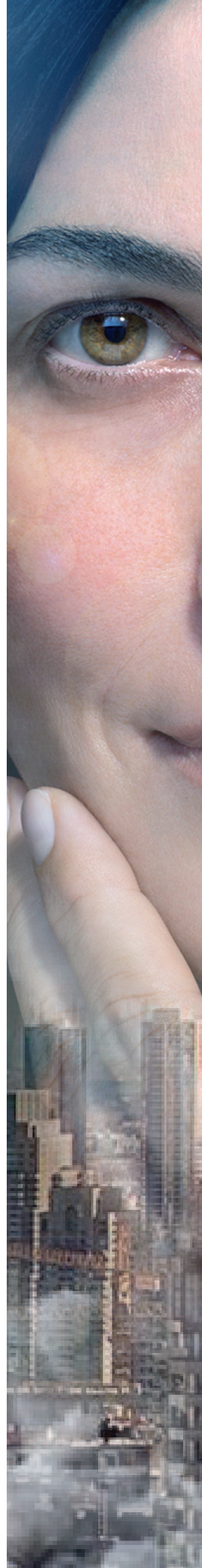
The relationship between the **skin and its environment** has raised major concerns during the last decades. The link between chronic **solar exposure** and photoaging is well established and there is now evidence of the contribution of **air pollution** to premature skin aging.

Solar induced-skin aging, also coined **photoaging**, is characterized by the formation of coarse **wrinkles**, irregular **pigmentation** and **elastosis**. Schroeder *et al.* have outlined the role of infrared A (IRA) radiation, within solar radiation, in photoaging. By penetrating deeply into the skin, **IRA alters skin homeostasis** *via* the production of **oxidative stress** (see Protection against Infrared A Radiation, p.4). Solar exposure is not the only foe of skin health. There is an increasing body of evidence showing that ambient air pollution, especially **airborne particle** and **gases** such as nitrogen dioxide and ozone, has adverse effects on the skin and might influence skin aging as well. Vierkötter *et al.* have reported that exposure to **traffic-related particle matter is significantly associated with an increased risk of developing pigment spots and wrinkles on the face** (see Airborne Particles and Skin Aging, p.5).

Moreover, urban pollution has detrimental effect on the skin beyond photoaging. Lefebvre *et al.* have reported that **air pollution impacts the quality of skin**: hydration, sebum excretion rate and greasy skin (see Impact of urban pollution on the quality of skin, p.6). Solar exposure and air pollution seem to have similar activating mechanisms. Skin pigmentation, i.e. lentigines, appears to result from the activation of aryl hydrocarbon receptor by both polycyclic aromatic hydrocarbons-loaded particles and UV radiation. Nakamura *et al.* have summarized the existing evidences that support the **role of environmental toxicants in the pathogenesis of lentigines** (see Environment-induced lentigines, p.7).

Nonetheless, skin care strategies are available to **prevent or minimize hazards of the environment**. They generally include the application of **broad-spectrum sunscreens** to protect from both UVB and UVA radiation and the topical application of **antioxidants** to quash the harmful effects of IRA radiation.



PROTECTION AGAINST INFRARED A RADIATION

From: Photoprotection beyond Ultraviolet Radiation Effective Sun Protection Has to Include Protection against Infrared A Radiation-Induced Skin Damage.
Skin Pharmacology and Physiology 2010;23:15–17

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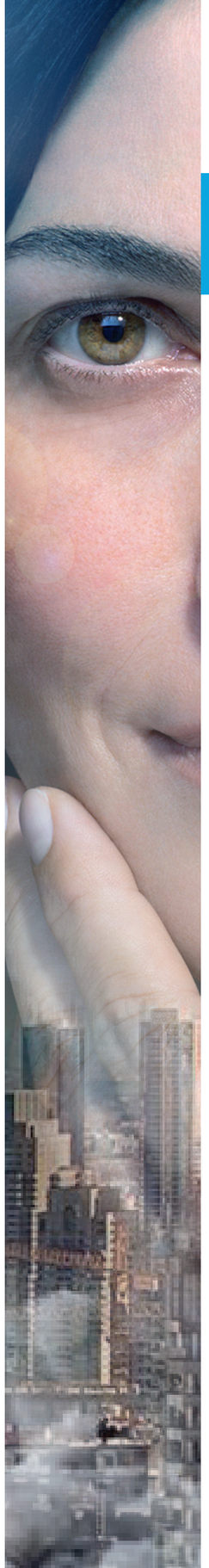
Aging results from the combined action of **intrinsic and extrinsic factors**. Clinical symptoms of extrinsic skin aging include coarse **wrinkles**, irregular **pigmented spots** and **elastosis**. Exposure to solar **radiation**, as well as other environmental factors, **contributes significantly to the extrinsic skin aging**. The process by which solar radiation gradually induces clinical and histological changes in skin is termed "**photoaging**". Photoaging is characterized by the formation of coarse **wrinkles, uneven skin pigmentation, loss of elasticity** and disturbance of skin barrier function. Ultraviolet (UV) radiation has long been associated with photoaging. However, several independent studies carried out during the last decade indicate that **infrared** (IR) radiation **contributes to photoaging** and IR has been shown to induce wrinkles formation.

Near IR, **IRA** (760–1440 nm) **penetrates deeply into the dermis**. Even at low doses, IRA radiation can trigger, in fibroblasts, gene-regulatory effects and the production of cytosolic and **mitochondrial reactive oxygen species**. Consequently, **IRA alters the equilibrium of the extracellular matrix and disturbs dermal homeostasis**. Indeed, IRA exposure leads to a **decrease of collagen** *de novo* synthesis and to an **increase of matrix metalloproteinase 1** (MMP-1, one of the main collagen-degrading enzyme) production and activity.

Unlike UV radiation, there are currently no filters available against IRA. An alternative approach for protection against IRA is **the use of antioxidants**. The use of mitochondria-targeting antioxidants has been shown to provide effective *in vitro* protection against IRA-induced production of MMP-1. *In vivo*, **the topical application of such antioxidants** prior to IRA exposure abrogates the IRA-induced detrimental shift in dermal gene expression.
An **efficient protection** against solar radiation should not exclusively focus on UV radiation but also **include protection against IRA** radiation.



AIRBORNE PARTICLES AND SKIN AGING

From: Airborne Particle Exposure and Extrinsic Skin Aging.
Journal of Investigative Dermatology 2010 Dec;130(12):2719-26

A. Vierkötter, T. Schikowski, U. Ranft, D. Sugiri, M. Matsui, U. Krämer
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The skin is **chronically exposed to the environment**, which strongly affects skin physiology, leading to extrinsic or premature aging. Among other environmental factors, **airborne particulate matter** (PM) represents a dire environmental threat for human skin. Micro-particles, with polycyclic aromatic hydrocarbons (PAH) adsorbed on their surface, can **penetrate skin** either through hair follicles or through the epidermis. Micro-particles exert their detrimental skin effects through the **generation of oxidative stress**.

Investigations lead during **two years** on **400 Caucasian women** aged 70–80 years living either in a **polluted urban area** (Ruhr, Germany, mean level of traffic-related particle emission of 899.9 kg/year/km^2) or in a **less polluted rural area** (Borken, Germany, mean level of traffic-related particle emission of 225.7 kg/year/km^2) revealed **that long-term exposure to air pollution is strongly associated to extrinsic skin aging**. More precisely, an increase in **soot**, an increase in **particles from traffic** and an increase of PM_{10} (PM with an aerodynamic diameter of 10 µm) background concentrations were associated with **20% more pigment spots on the face** (forehead and cheeks) and more **pronounced nasolabial folds**, as assessed by means of the **SCINEXA** (score of intrinsic and extrinsic skin aging). **Sunburns in childhood** and sunbed use were associated with **more spots on forehead**. Exposure to **tobacco smoke** (current or prior) was associated with more wrinkles on upper lips, increased elastosis, more pronounced telangiectasia and laxity of the face. Interestingly, **light skin type** was associated with **less pigmented spots** on the face, **less coarse wrinkles** under the eyes and on upper lips, **less elastosis**, but more **pronounced telangiectasia**, in comparison to darker skin type.

Thus, among several environmental factors, **airborne particle pollution might influence skin aging**.

IMPACT OF URBAN POLLUTION ON THE QUALITY OF SKIN

From: Evaluation of the impact of urban pollution on the quality of skin: a multicentre study in Mexico.
International Journal of Cosmetic Science, 2015, 37, 329–338

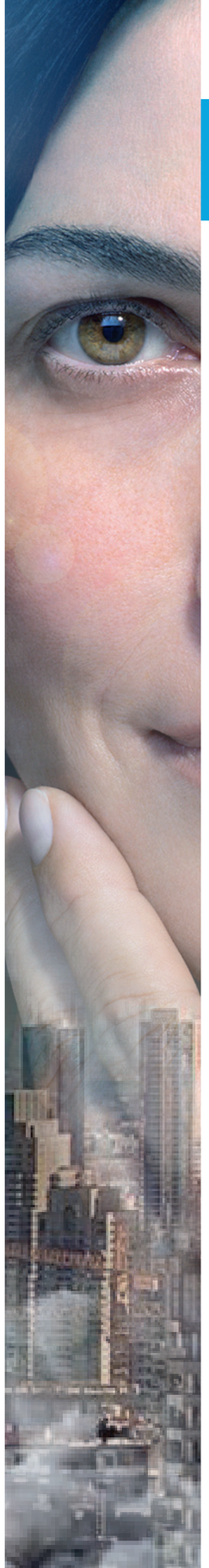
M.-A. Lefebvre, D.-M. Pham, B. Boussouira, D. Bernard, C. Camus and Q.-L. Nguyen from L'Oreal Research & Innovation, France

The environment can affect the skin **more than just increasing extrinsic aging**. Indeed, **air pollution also alters the quality of the skin**. For example, ozone, considered as one of the main pollutant, produces **oxidative stress** in the stratum corneum, interferes with **wound healing** processes and can increase the occurrence of skin conditions such as urticaria, eczema, contact dermatitis, rash and other non-specific eruption.

The **effect of pollution on the quality of human skin** was evaluated in a multicenter study conducted on 96 individuals (mean age 31) living in a **polluted area** (South-West district of Mexico City, Mexico) and 93 individuals (mean age 33) living in a **less polluted area** (North district of Cuernavaca, 50 km South of Mexico City).

As expected, **several biochemical parameters differ** from the two studied populations. **Moisturizing** (hydration of the stratum corneum), chymotrypsine-like activity (involved into the desquamation process of the stratum corneum), amount of adenosine triphosphate and of interleukin-1α were significantly lower in the Mexico City population than in the Cuernavaca population. Concentrations of vitamin E (a powerful endogenous antioxidant) and squalene (specific compound produced by sebaceous glands) were significantly lower in the Mexico City population, indicating that they are mobilized to correct oxidative conditions. However, sebum excretion rate on forehead, **erythema** on cheeks and amount of oxidized proteins and of lactic acid were significantly higher in the Mexico City population. At the clinical level, familial antecedents of **sensitive skin**, antecedents of **urticaria** and problems of **hair scalp**, **greasy skin** in nasal and peri-nasal zones were significantly higher in the Mexico City population with however less evidence of **dandruffs** and of greasy skin on cheeks.

Thus, **pollutants** such as air components (ozone, nitrogen dioxide…) and industrial residues (soot, polycyclic aromatic hydrocarbon, micro-particles…) significantly modifies the skin homeostasis and therefore **affect the quality of the skin**.



ENVIRONMENT-INDUCED LENTIGINES

From: Environment-induced lentigines: formation of solar lentigines beyond ultraviolet radiation.
Experimental Dermatology, 2015, 24, 407–411

M. Nakamura, A. Morita, S. Seité, T. Haarmann-Stemmann, S. Grether-Beck and J.Krutmann from the IUF - Leibniz Research Institute for Environmental Medicine, Düsseldorf, Germany

Lentigines, the acquired dark brown pigmented spots affecting >90% of white people older than 50 years, are considered as a **clinical hallmark of photoaged skin**. Traffic- and industry-related **air pollution** (soot, diesel exhaust particles, particulate matter, nitrogen dioxide and sulfur dioxide) is significantly associated with an **increased risk of developing pigmented spot**. Thus, several environmental factors, not just solar radiation, contribute to the development of these pigmented spots, therefore coined "**environment-induced lentigo**" (EIL).

The **formation of lentigines** results from a **disturbed crosstalk** between keratinocytes and fibroblasts producing soluble factors affecting **melanocyte** differentiation, proliferation and function. Despite the variety of environment noxae, the common denominator for environment-induced skin pigmentation appears to be the **activation of aryl hydrocarbon receptor** (AhR) signaling pathway. AhR is a ligand-activated transcription factor, expressed in keratinocytes, melanocytes and fibroblasts. It acts as a **sensor of environmental changes in order to maintain skin integrity**. Polycyclic aromatic hydrocarbons-loaded particles present in polluted air and **UV radiation** can activate AhR, **increase melanin synthesis** and thereby foster lentigines formation. Pollutant or UVR-induced AhR signaling might also contribute to the persistence of damaged keratinocytes in human skin. Topical application of AhR antagonists facilitates the clearance of damaged cells from skin and/or reduces the production of soluble factors involved in lentigines formation.

The **inhibition of melanin production**, the blocking of melanocyte stimulation or the suppression of melanin delivery are viable **therapeutic options to treat lentigines**. Such prominent examples of efficient topical therapies include **tretinoin** (retinoic acid or vitamin A acid), hydroquinone, mequinol and adapalene.

Hence, the **prevention of lentigines formation** should consider the variety of environmental factors, not just UV exposure. Obviously, the **use of a broad-spectrum photoprotection**, protecting from both UVB and UVA radiation is still highly recommended. However, an effective prevention should also aim at **reducing** or even **preventing the penetration of ambient relevant pollutants into the skin**. This could be achieved by daily use of **skin cleansing products** or texture-innovating cosmetics to **decrease the pollution load** on the skin surface or by use of skin care strategies directed at **improving skin barrier function**.

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