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C. Diehl
Universitá Degli Studi Guglielmo Marconi, Rome, Italy

Air pollution and the skin

It is well known that air pollution is for a long time a major concern for public health. Being designated by WHO as the «world's largest single environmental health risk», its consequences in term of mortality and morbidities are serious and well-documented. On the contrary, the effects of air pollution on the human skin have not been deeply studied, and day after day we discover that it can affect the health and aesthetic appearance of the skin more than previously reported. This review is summarizing the various air pollutants and reporting their influence at cell level and their incidence on the occurrence and worsening of different dermatological diseases.

Key words

Skin, air pollution, pollutants, particulate matter, ozone, polycyclic aromatic hydrocarbons, nitrogen oxides, cigarette smoking, skin cancer, atopic dermatitis, skin aging.

The human skin, in particular the upper layer of the epidermis (stratum corneum SC), plays the role of a barrier: the barrier function of the skin is «La raison d'être» of the epidermis [1]. This barrier function is provided by the corneocytes themselves, tightly opposed to each other, and by the patterned lipid lamellae located in the extracellular spaces between the corneocytes. This barrier function prevents the intrusion within the organism of exogenous particles or substances present in the environment, but also regulates the skin hydration, by modulating the transepidermal water loss (TEWL).

Obviously, the skin acts as a shield against exogenous factors, and is the first line of defence of the human organism in front of environmental stressors. Unsurprisingly, the skin will be the first target of air pollutants and will reflect their deleterious effects.

Depending on the nature of these pollutants and the integrity of the skin, the mode of penetration of pollutants differ [2]. The protective ability of the skin is not unlimited, and problems arise when an abnormal exposure to environmental stressors exceeds the skin's normal defensive power [3].

Air pollutants commonly found in the atmosphere

Air pollution is the introduction of particulates, biological molecules, or other harmful materials into the atmosphere. Some of them are of environmental origin, whilst others are of anthropic origin, i. e. provoked by humans (for instance cigarette smoking). The US Environmental Protection Agency (EPA) [4] has listed seven criteria pollutants as shown in Table.

Gaseous air pollutants include NO, NO_2, CO, SO_2, ozone and volatile organic compounds (VOC) and are mainly originated from the combustion of fossil fuels (coal, petrol and natural gas) by automobiles, aircrafts, boilers, power generators... CO is also produced by tobacco smoke.

Persistent organic pollutants (POPs) are compounds resistant to environmental degradation and capable of long-range transport and bioaccumulation in humans and animals [5]. They include herbicides, pesticides, dioxin and its derivatives. Pesticides and herbicides are obviously used in agriculture, and dioxin is produced in the course of industrial processes such as smelting, chlorine bleaching and manufacturing of pesticides and herbicides [5]. They are also by-products of volcanic eruptions. They can accumulate in edible plants and cattle, and are susceptible of being part of human diet.

Particulate matter (PM) is a complex blend of liquid and/or solid droplets suspended in gas, whose average size is between 2.5 and 10 μm in diameter. They are produced by automobile exhausts, power plants and various industries. Interestingly, modern diesel engines produce ultrafine particles, whose diameter is < 0.1 μm [5]. These ultrafine particles may gain systemic access.

Ozone (O_3) is normally found in low concentrations at ground level, originating from the stratospheric O_3 and hydrocarbons released by plants and soil [2]. However, it can also be formed as a by-

Table. **Criteria pollutants monitored in the USA by the National Ambient Air Quality Standard**

Ambient air pollutant	Averaging time	Standard level	Source type	Common sources
Carbon monoxide	8 h	10 mg/m^3 (9 ppm)	Anthropogenic	Vehicle emission Fuel combustion
Lead	Quarterly	0.15 μg/m^3	Anthropogenic	Combustion of leaded fuel Metal refineries Waste incinerators Battery factories Battery waste
Nitrogen dioxide	1 h	188 μg/m^3 (100 ppm)	Anthropogenic	Vehicle emission Fuel combustion Wood burning
Ozone	8 h	147 μg/m^3 (0.075 ppm)	Natural	Formed by chemical reaction of VOC and NO_2 produced by plants and trees
			Anthropogenic	Formed by chemical reaction of VOC and NO_2 produced by fuel combustion
				Smog
Particulate matter PM 2.5	Quarterly	12.5 μg/m^3	Anthropogenic	Vehicle emission Combustion of fuel Industrial activity Agricultural burnings Garbage incineration
Particulate matter PM 10	24 h	150 μg/m^3	Natural	Road dust Forest fires
			Anthropogenic	Unpaved roads
Sulphur dioxide	1 h	196 μg/m^3 (75 ppb)	Natural	Volcanoes
			Anthropogenic	Fuel combustion Electric utilities Industrial processes

product of certain human activities, with the interaction of sunlight (UVR), hydrocarbons, VOCs and NOx, representing a major active component of the pro-oxidant smog [6].

Heavy metals such as cadmium, lead or mercury are commonly emitted by natural sources such as volcanoes, and by industrial ones like waste incineration, and industrial processes. Lead is mainly produced by the combustion of leaded gasoline, still in force in many countries worldwide.

Traffic-related pollutants (TRP) consist of the emission resulting from the combustion of gasoline and diesel. They contain mainly CO_2, CO, NO_2, VOCs, PM and lead, but also polycyclic aromatic hydrocarbons (PAHs) and benzene.

How do air pollutants impact the human skin?

Solar ultraviolet radiation (UVR)

Is not related to pollution and may occur at any case. However, even small changes in stratospheric O_3 (ozone) increase the penetration of UVA and UVB at ground level and our exposure to the same. The depletion of stratospheric O_3 by chlorofluocarbons (CFCs) and other industrially produced O_3 destructive compounds presents a major problem in the environment and for human health [6]. Despite frequent warnings, the stratospheric O_3 layer is still thinner than one century ago, while there is an O_3 hole in the Antartica and other openings over northern USA [2]. Depending on their wavelength, the penetration of UVR into the skin is different. UVB is largely absorbed by the epidermis, limiting its damages to the superficial skin layer, while UVA penetrates deeply into the dermis. Due to its action on the fibroblasts, UVA is a major contributor to skin aging. UVA in combination with environmental pollutants significantly increases visible photodamage to the skin [7]. Both UVA and UVB are linked to the development and progression of skin cancers such as malignant melanoma, basal cell carcinoma (BCC), and squamous cell carcinoma (SCC) through different mechanisms. The effects of O_3 depletion induced an increase in skin cancers in

countries related with the Antarctic ozone hole, like Chile [8]. It is predicted that for every 1 % decrease in ozone there is a 2 % increase in UVB irradiance, and therefore a 2 % increase in skin cancer [9].

Polycyclic Aromatic Hydrocarbons (PAHs)

PAHs can activate xenobiotic metabolism, which converts PAHs to quinones. Quinones are redox-cycling chemicals, which produce reactive oxygen species, and are therefore thought to be key compounds in PM toxicity [10]. Aryl hydrocarbon receptor ligands, such as dioxin or PAHs, have been shown to induce melanocyte proliferation and thereby skin tanning in mice [11]. Long term-exposed skin to PM-bound PAHs either through hair follicle or transepidermal absorption may lead to oxidative stress and skin aging [12]. PAHs have been implicated in the development of skin cancer. Activated PAHs produce epoxides and diols, respectively, which bind to DNA and initiate cutaneous carcinogenesis [13]. The carcinogenic action of benzo[a]pyrene was shown to be enhanced with the interaction of UVA [14]. PAHs can lead to acneiform eruptions [13] such as chloracne, a systemic toxic disease caused by the exposure to chloracnegens (halogenated aromatic hydrocarbons) and characterized by acneiform skin lesions such as comedones and cysts mainly on the face (outer sides of the eye and behind the ears) and neck [15].

Volatile Organic compounds (VOCs)

VOCs, with the presence of sunlight and NOx, cause the formation of photochemical oxidant products-mainly O_3- at ground level, also called summer photochemical smog [6]. Oral exposure of rats to Hexachlorobenzene (HCB) induces precancerous skin lesions in the rat and correlation of skin lesions with immune parameters suggest a specific involvement of the immune system in HCB-induced skin lesions [16]. Normal human keratinocytes exposed to VOCs increased the expression of pro-inflammatory cytokines such as IL1β, IL-8 and TNF-α, suggesting that these environmental pollutants may act as modulating factors of cutaneous inflammation by affecting the ability of keratinocytes to release pro-inflammatory cytokines and may favour the development of inflammatory and/or allergic reaction such as atopic dermatitis (AD) or eczema [17]. It was shown that exposure to VOCs — at concentrations commonly found in indoor environments — can damage the epidermal barrier and enhance the symptoms on sensitized subjects with AD [18].

Nitrogen oxides (NOx)

Results of a study [19] indicate that a short period of exposure to low concentrations of NO_2 affects the skin of patients with atopic eczema, as well as normal skin. It is known that NO_2 causes oxidative damage resulting in the generation of free radicals that may oxidize amino acids in tissue proteins. NO_2 also initiates lipid peroxidation of polyunsaturated fatty acids in pulmonary cell membranes. Similar mechanisms might be responsible for the effect of NO_2 on healthy skin, as well as for that on the skin of patients with atopic eczema. These changes evidently influence the epidermal barrier function as measured by TEWL, which increased significantly during NO_2 exposure. An East-West German comparative study examining different types and levels of air pollution, i. e. sulphurous (industrial; East) and oxidising (urban; West), showed that the prevalence of atopic eczema was greatest in East Germany. When various direct and indirect parameters of air pollution exposure were measured, the greatest association with atopic eczema was found with NO_2 exposure (indoor use of gas without a cooker hood), and close proximity to roads with heavy traffic [20].

Particulate matter (PM)

In another study [21], air pollution exposure was significantly correlated to extrinsic skin aging signs, in particular to pigment spots and less pronounced to wrinkles. An increase in dust and particles from traffic (per 475 kg per year and square km) was associated with 20 % more pigment spots on forehead and cheeks. Background particle pollution was measured in low residential areas of cities without busy traffic and therefore is not directly attributable to traffic but rather to other sources of particles, which was also positively correlated to pigment spots on face. These results indicate that particle pollution might influence skin aging as well. The mechanisms of PM-health effects are believed to involve oxidative stress and inflammation. This was recently confirmed [22] by exposing reconstructed human epidermis (RHE) to various concentrations of concentrated ambient particles. A local ROS production increase generated by metals present in the particulate was observed, contributing to lipids oxidation. There was also an increase of NF-κB nucleus translocation as well as cyclooxygenase 2 and cytochrome P450 levels. Use of transition electron microscopy in this experiment permitted to show that PMs were able to penetrate skin tissues. The increased production of ROS such as superoxide and hydroxyl radical by PM exposure increases MMPs including MMP-1, MMP-2, and MMP-9, resulting in the degradation of collagen. These processes lead to increased inflammatory skin diseases and skin aging [23].

Ozone

The actions of O_3 could be amplified in the presence of other air pollutants, where concomitant exposure to UV irradiation and O_3 could reveal synergistic oxidative stress effects in the skin [6]. Experimental evidence shows that O_3 can induce damage in the epidermis of murine skin, reduce the level of antioxidants such as α-topopherol (vitamin E) and ascorbic acid (vitamin C) and increase malondialdehyde (MDA), a lipid peroxidation product. These effects lead to barrier perturbations, the production of lipid ozonation products and inflammation [6]. Exposing normal human epidermal keratinocytes to concentrations of ozone that have been measured in cities, hydrogen peroxide and IL-1α levels both increased while ATP levels decreased and ozone increased DNA damage as evaluated by Comet assay [24].

Xu et al. have confirmed the cutaneous toxic effect of O_3 in humans [25]. Collecting data from patients from urban areas of Shangai that had visited emergency rooms for skin conditions; they monitored levels of several pollutants including O_3. The data, from almost 70.000 patients collected over almost two years, shows a clear exposure—response relationship between increased O3 concentration and skin conditions such as urticaria, eczema, contact dermatitis, rash/other non-specific eruption, and infected skin disease.

Cigarette Smoking (CS)

Cigarette smoke (CS) is a highly complex aerosol composed of several thousand chemical substances distributed between the gas and the particulate phase [3]. The presence of high levels of pro-oxidants, such as free radicals, in smoke is well documented. A wealth of evidence supports the notion that a major part of the toxicity associated with CS is related to oxidative stress caused by reactive oxidants and radical species in tobacco smoke itself or by secondary oxidative events, such as lipid peroxidation activated by smoke exposure [26]. The effect of cigarette smoking on the skin was reported for a long time and in 1985 facial features induced by CS were defined as smoker's face, which describes the characteristic changes that happen to the faces of smokers, including accelerated aging facial skin with a characteristic pattern of wrinkling and sallow coloration (orange and purple colour) [27]. CS was shown to be associated with an increase in elastosis, which may contribute to the clinical features of «smoker's face» [28]. Upon treatment with tobacco smoke extract, the expression of MMP-1 and MMP-3 mRNA was significantly increased in a dose-dependent manner and type I and type III collagens were decreased as compared to untreated controls [29]. The effect of CS on cutaneous tissue is associated with not only premature skin aging and wrinkling but also with several other pathologies. A recent literature review concludes in favour of a positive association between the prevalence of smoking and psoriasis as well as an association between smoking and the severity of psoriasis [30]. In addition, smoking has been shown to affect the response of the patients to psoriatic treatments, thus worsening the pathology [31]. In a study on melanoma, 2.298 melanoma cases and 6.654 controls were investigated [32] and there was association between the genetic scores based on the number of smoking behaviour-risk alleles and melanoma risk. However, it was previously reported that smoking was inversely related to cutaneous malignant melanoma [33]. A systematic review and meta-analysis on the association of atopic dermatitis with smoking concluded that active and passive exposure to smoke was associated with increased AD prevalence [34]. A meta-analysis of seven case-control studies and two cohort studies found a 1.5 times increased risk of SLE in current smokers, when compared with non-smokers [35]. The association between cutaneous squamous cell carcinoma (SCC) and smoking has been described in case-control and cohort studies. Notably, in the Nurses' Health Study, smokers had a 50 % increased risk of incident SCC compared with non-smokers [36]. Observational studies have found a much higher prevalence of smoking (up to 90 %) among patients with hidradenitis suppurativa, whereas smoking cessation did not appear to alter disease activity [37]. Clinical evidence and experimental data showed a straight correlation between smoking habit and post-pubertal acne in which the clinically non-inflammatory type is the most frequent [38]. In a US study, smoking was also found having a significant association with the presence of active hand eczema (HE) [39]. In a prospective cohort study in Germany, the severity of occupational HE was increased in smokers. Tobacco smoking was associated with a higher number of days of absence from work and with not staying in the workforce owed to occupational HE [40].

Skin diseases related to air pollution

At this stage, we can state that there is no skin disease whose cause is solely air pollution. However, it appears that a number of skin diseases may be worsened by air pollution. As seen previously, air pollutants mainly act by triggering the oxidative stress, which in turn activates the inflammatory cascade. This is the case of UVR, PAHs, VOCs, NOx, PMs and O_3. For this reason, unsurprisingly,

most of inflammatory diseases will be susceptible of being aggravated by air pollution.

AD, probably the most frequent chronic inflammatory skin disease, is in the first line.

Recent evidence suggests that a variety of air pollutants, such as environmental tobacco smoke, volatile organic compounds, formaldehyde, toluene, nitrogen dioxide, and particulate matter, act as risk factors for the development or aggravation of AD. These air pollutants probably induce oxidative stress in the skin, leading to skin barrier dysfunction or immune deregulation [41]. Interestingly, a study conducted recently in Taipei (Taiwan) investigated the association between ambient bioaerosol exposure and allergic skin diseases [42]. The results indicated that during the study period, contact dermatitis and other eczema were even more prevalent than atopic dermatitis in the study area. Most bioaerosols were positively associated with both skin diseases. Environmental pollutants can result in a variant of acne called «chloracne». Chloracne is caused by systemic exposure to certain halogenated aromatic hydrocarbons «chloracnegens», and is considered to be one of the most sensitive indicators of systemic poisoning by these compounds. Dioxin is the most potent environmental chloracnegen. Most cases of chloracne have resulted from occupational and non-occupational exposures. Non-occupational chloracne mainly resulted from contaminated industrial wastes and contaminated food products. Non-inflammatory comedones and straw-colored cysts are the primary clinical manifestation of chloracne. An increase in the number of cysts is a signal of aggravation of chloracne. Generalized lesions can appear on the face, neck, trunk, exterimities, genitalia, axillary and other areas. The course of chloracne is chronic. The severity of chloracne is related to dosage of exposed chloracnegens, chloracnegenic potency and individual susceptibility [43]. The rise in the prevalence of rosacea in the last years must be related to air pollutants, at least partly, although there is currently no consistent study to demonstrate it.

Skin cancers (melanoma, but also BCC and SCC) are of major concern for dermatologists and for public health. It is well-known that UVR are majorly responsible for the development and progression of these pathologies. Given the incidence of air pollution on the thinning of ozone layer and even appearance of the ozone hole in some areas, it may be asserted that air pollution is the cause of an increased number of skin cancer of all types. Further, the carcinogenic action of PAHs was demonstrated. Increased O_3 levels are also responsible for an increased DNA damage at cell level, which can be a precursor of skin cancers.

Urban pollution was also shown to impact the quality of the skin. A recent study performed in Mexico was conducted on one side on individuals living in Mexico City, one of the most polluted cities in the world, and on the other side in Cuernavaca, a smaller city with lesser pollution [44]. The study demonstrated significant quantitative and qualitative modifications of parameters related to sebum excretion in Mexico City compared to Cuernavaca: An increased level of sebum excretion rate, a lower level of vitamin E and squalene in sebum, an increase of lactic acid and a higher erythematous index on the face of the subjects. In the stratum corneum, a significant higher level of carbonylated proteins and a lower level of IL 1α were noticed, as well as a decrease of ATP concentration. From a clinical point of view, a higher frequency of atopic and urticarial skins, a higher frequency of red dermographism, an important seborrheic status at the forehead level and a lower level of dandruffs were noted in

Mexico City population. Air pollutants could also play a role in the occurrence of androgenetic alopecia: environmental cigarette smoke, which is well known as an oxidizing agent, was shown to be closely related to androgenetic alopecia (AGA) [23].

Last but not least from our patients' point of view, air pollution appears to strongly influence skin aging. The excellent work from Vierkotter et al, already mentioned [21], provides epidemiological evidence that traffic related PM represents an important environmental factor that contributes to extrinsic skin aging in humans. This conclusion is based on the present observation that not only (i) an increase in dust, but also (ii) an increase in particles from traffic, and (iii) higher PM10 background concentrations were associated with more pigment spots on the face and more pronounced nasolabial folds. The distance of residence to the closest busy road was also associated with more pigment spots. In this study, smoking history was associated with more wrinkles, more elastosis, and more pronounced telangiectasia.

Conclusions

It is well known that air pollution is for a long time a major concern of public health. In 2014, the US Environmental Protection Agency (EPA) reported that over 142 million Americans live in areas where the air quality fails to meet the National Ambient Air Quality Standards [4]. In the same year, the World Health Organization attributed seven million premature deaths to air pollution exposure and designated air pollution as the «world's largest single environmental health risk» [45]. In an intent to evaluate the costs of air pol-

lution in Ukraine in 2006 [46], the methodology developed by US EPA was adjusted for Eastern European transition countries and was applied for health risk assessment. In total, air pollution related mortality represented about 6 percent of total mortality in Ukraine. The relative mortality risk attributed to air pollution calculated on a population of 100 000 was about 55—59 cases. However, our knowledge regarding the effects on skin health remains limited, and we must deepen it and consider seriously this extrinsic factor at the time of establishing our diagnosis.

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К. Діл

Університет Гульєльмо Марконі, Рим, Італія

Забруднення повітря та шкіра

Забруднення повітря протягом тривалого часу є однією з основних проблем у сфері охорони здоров'я. Воно визнане ВООЗ «найбільшим у світі екологічним ризиком для здоров'я». Існує значна кількість переконливих документальних підтверджень смертності й захворюваності, спричинених цим забрудненням. Водночас проблема впливу забрудненого повітря на людську шкіру не була глибоко вивчена, і щодня ми отримуємо все нові докази того, що забруднення повітря негативно позначається на стані здоров'я та естетичному зовнішньому вигляді шкіри. В огляді узагальнено різні речовини, що забруднюють повітря, проаналізовано їхній вплив на клітинному рівні, наведено дані про частоту виникнення та загострення різних дерматологічних захворювань.

Ключові слова: шкіра, забруднення повітря, речовини, що забруднюють повітря, пиловидні речовини, озон, поліциклічні ароматичні вуглеводні, оксиди азоту, куріння, рак шкіри, атопічний дерматит, старіння шкіри.

К. Дил

Университет Гульельмо Маркони, Рим, Италия

Загрязнение воздуха и кожа

Загрязнение воздуха в течение длительного времени является одной из основных проблем в сфере здравоохранения. Оно признано ВОЗ «крупнейшим в мире экологическим риском для здоровья». Существует значительное количество убедительных документальных подтверждений смертности и заболеваемости, вызванных этим загрязнением. В то же время проблема влияния загрязненного воздуха на человеческую кожу не была глубоко изучена, и каждый день мы получаем все новые доказательства того, что загрязнение воздуха негативно сказывается на состоянии здоровья и эстетическом внешнем виде кожи. В обзоре представлена информация о различных веществах, загрязняющие воздух, проанализировано их влияние на клеточном уровне, приведены данные о частоте возникновения и обострения различных дерматологических заболеваний.

Ключевые слова: кожа, загрязнение воздуха, вещества, загрязняющие воздух, пылевидные вещества, озон, полициклические ароматические углеводороды, оксиды азота, курение, рак кожи, атопический дерматит, старение кожи.

Дані про автора:

Dr. Christian Diehl, Department of Dermatology, Universitá Degli Studi Guglielmo Marconi
Via Plinio, 44, 00193, Rome, Italy
E-mail: chdiehl@hotmail.com