Review Article

Effects of air pollution on the skin: A review

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Abstract

The increase in air pollution over the years has had major effects on the human skin. Various air pollutants such as ultraviolet radiation, polycyclic aromatic hydrocarbons, volatile organic compounds, oxides, particulate matter, ozone and cigarette smoke affect the skin as it is the outermost barrier. Air pollutants damage the skin by inducing oxidative stress. Although human skin acts as a biological shield against pro-oxidative chemicals and physical air pollutants, prolonged or repetitive exposure to high levels of these pollutants may have profound negative effects on the skin. Exposure to ultraviolet radiation has been associated with extrinsic skin aging and skin cancers. Cigarette smoke contributes to premature aging and an increase in the incidence of psoriasis, acne and skin cancers. It is also implicated in allergic skin conditions such as atopic dermatitis and eczema. Polyaromatic hydrocarbons are associated with extrinsic skin aging, pigmentation, cancers and acneiform eruptions. Volatile organic compounds have been associated with atopic dermatitis. Given the increasing levels of air pollution and its detrimental effects on the skin, it is advisable to use strategies to decrease air pollution.

Key words: Ozone, particulate matter, pollution, polycyclic aromatic hydrocarbons, skin, ultraviolet radiation, volatile organic compounds

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Introduction

Pollution is defined as contamination of the earth's environment with materials which interfere with human health, quality of life, or the natural functioning of the ecosystem. The major types of pollution are water pollution, air pollution, noise pollution and soil pollution. The World Health Organization defines air pollution as contamination of the indoor or outdoor environment by any chemical, physical, or biological agent that modifies the natural characteristics of the atmosphere.[1] The sources of air pollution could be natural sources such as volcanic eruptions, forest fires, biological decay, pollen grains, marshes and radioactive materials or human-made sources such as thermal power plants, industries, vehicular emissions, household combustion devices, fossil fuel burning and agricultural activities. Pollutants of the major public health concern include particulate matter, carbon monoxide, ozone, nitrogen dioxide and sulfur dioxide.[1] Air pollution is responsible for a large proportion of health-related problems.[2]

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Mechanism of Skin Damage by Air Pollutants

Living organisms are exposed to air pollutants which have major effects on the human skin. Air pollutants can exist as solids, liquids, gases and particulate matter. These are absorbed directly through the skin into the subcutaneous tissue or via hair follicles and sweat/sebaceous glands. Rapid urbanization and increased energy consumption worldwide have exposed the human body to increased quantities of ambient air pollution. The skin, being the largest and outermost body organ, acts as a physical, chemical and an immunological barrier against the environmental factors. Human skin is exposed not only to natural environmental factors but also to pollutants of anthropic origin.[3] Whenever a prolonged and repetitive exposure to environmental stressors exceeds the skin's normal defensive potential, there is a disturbance in the skin barrier function leading to the development of various skin diseases.[3] Major air pollutants which affect the skin are solar ultraviolet radiation, polycyclic aromatic hydrocarbons, volatile organic compounds,

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nitrogen oxides, particulate matter, cigarette smoke, heavy metals and arsenic.

Air pollutants exert a harmful effect on the skin by increasing oxidative stress which counters the skin's antioxidant defenses. There is a depletion of enzymatic (glutathione peroxidase, glutathione reductase, superoxide dismutase and catalase) and non-enzymatic (Vitamin E, Vitamin C and glutathione) antioxidant capacity. Free radicals and reactive oxygen species are generated that interact with the lipid-rich plasma membrane to initiate the lipid peroxidation reaction cascade. Reactive oxygen species also stimulate the release of pro-inflammatory mediators which results in the accumulation of neutrophils and other phagocytic cells that further generate free radicals, thereby resulting in a vicious cycle. Oxidative stress initiates complex biological processes resulting in genetic damage, activation of transcription factors such as activator protein 1 and nuclear factor kappa B, and signalling pathways such as extracellular signal-regulated kinases, c-Jun N-terminal kinases and p38 mitogen-activated protein kinases, involved in cell growth and differentiation and in the degradation of the connective tissue of the dermis. Air pollutants induce severe alterations of the normal functions of lipids, deoxyribonucleic acid and/or proteins in the human skin via oxidative damage, leading to extrinsic skin aging, inflammatory or allergic conditions such as contact dermatitis, atopic dermatitis, psoriasis, acne and skin cancer.[4,6-8]

Air Quality Guidelines

The World Health Organization air quality guidelines are based on four major air pollutants, namely particulate matter, ground-level ozone, nitrogen dioxide and sulfur dioxide.[1] The World Health Organization guidelines for the various air pollutants are shown in Table 1. The Revised National Ambient Air Quality Standards, 2009, are also shown in Table 1.[9] Another measure for air pollution is air quality index based on five major air pollutants regulated by the Clean Air Act:- ground-level ozone, particulate matter, carbon monoxide, sulfur dioxide and nitrogen dioxide. Its value lies between 1 and 500, with higher values indicating more air pollution. Air quality index value of 50 represents a good air quality with some potential to affect public health, while an air quality index value over 300 represents hazardous air quality. Real-time records of air quality index in New Delhi show that it is between 150 and 170, which is categorized as unhealthy.[10] This article focuses on the detrimental effects of air pollutants on various skin disorders.

Air Pollutants and their Role in Skin Diseases

Ultraviolet radiation

Ultraviolet radiation is a physical pollutant. The solar spectrum consists of ultraviolet A (320–400 nm), ultraviolet B (290–320 nm) and ultraviolet C (200–290 nm). More than 95% of ultraviolet A and 1–5% of ultraviolet B reach the Earth's surface, whereas most ultraviolet C is absorbed by the ozone layer and oxygen in the atmosphere.[11] The depletion of stratospheric ozone by environmental pollutants such as photochemical smog, supersonic aircraft flights and refrigerant gases increases the penetration of the shorter ultraviolet wavelengths to the ground level. The effects of ultraviolet radiation on human skin differ depending on the wavelength. Ultraviolet A exposure results in extrinsic skin aging (photoaging) characterized by coarse wrinkles, solar elastosis and pigment irregularities. Aging results from the combined action of intrinsic and extrinsic factors. The general aging process which is genetically determined and occurs in all skin by passing time is called intrinsic aging [Figures 1 and 2] whereas skin aging induced by environmental factors is termed as extrinsic aging. Clinical signs of extrinsic aging include solar elastosis [Figure 3], pigment spots [Figure 4], coarse wrinkles and telangiectasias. The differences between intrinsic and extrinsic aging are tabulated in Table 2.[12-15]

Photoaging is mainly caused by ultraviolet A. In an *in vitro* study, it was seen that ultraviolet B may also contribute to photoaging by increasing stratifin. Stratifin is a member of the 14-3-3 protein family, secreted by keratinocytes and its expression was more in skin exposed to ultraviolet B than in skin protected by ultraviolet B.[16] The mechanism of premature/extrinsic skin aging induced by ultraviolet rays is a complex process, which is triggered by various pathways, namely activation of receptors, mitochondrial damage, protein oxidation, alteration of Ca^{2+} levels, telomere damage and arylhydrocarbon receptor activation. The stimulation of receptor pathway is induced by the generation of reactive oxygen species on exposure to ultraviolet radiation, smoking and air pollutants. As a result, the cell surface receptors of cytokines and growth factors in keratinocytes as well as fibroblasts are activated which leads to the intracellular stimulation of intracellular kinases, inducing the transcription of nuclear transcription factor activator protein 1 and nuclear factor kappa B. Increased activator protein 1 transcription decreases the gene expression of the major dermal collagens I and III in fibroblasts leading to a reduction in collagen synthesis. Activator protein 1 also increases the synthesis of matrix metalloproteinases in keratinocytes and fibroblasts resulting in the increased degradation of mature dermal collagen. On the other hand, nuclear factor kappa B stimulates the transcription of inflammatory cytokines resulting in the accumulation of neutrophils. The collagenases in neutrophils help in collagen degradation.

Ultraviolet B alone is responsible for sunburn. Ultraviolet A and B both have been implicated in cutaneous immunosuppression and skin cancers (photocarcinogenesis) such as malignant melanoma, basal cell carcinoma and squamous cell carcinoma.[3,7,17] There is a growing concern worldwide regarding the increased incidence of skin cancer. Excessive exposure to sunlight and tanning

Table 1: Guidelines for limit of air pollutants[1]

Air pollutant	Maximum limit (WHO,2006)	Revised National Ambient air quality standards (2009)
Particulate matter having size between 2.5 and 10 μm	Mean value of 20 μg/m^3 over a year or 50 μg/m^3 over 24 h	Mean value of 60 μg/m^3 over a year or 100 μg/m^3 over 24 h
Ozone at ground level	Mean value of 100 μg/m^3 over 8 h	Mean value of 100 μg/m^3 over 8 h
Nitrogen dioxide	Mean value of 0 μg/m^3 over a year or 200 μg/m^3 over 1 h	Mean value of 40 μg/m^3 over a year or 80 μg/m^3 over 24 h
Sulfur dioxide	Mean value of 20 μg/m^3 over 24 h or 500 μg/m^3 over 10 min	Mean value of 50 μg/m^3 over a year or 80 μg/m^3 over 24 h

Table 2: Differences between intrinsic and extrinsic (photoaging)[12-15]

	Intrinsic aging	Extrinsic (photoaging)
Texture of skin	Dry, pale with certain degree of laxity	Very dry, atrophic, sallow, very lax
Sagging of skin	More prominent	Less prominent[14]
Wrinkles	Fine wrinkles	In addition, coarse wrinkles forming deep furrows (pathognomic)
Hyperpigmentation	Some freckles	Lot of irregularly pigmented freckles, heterogeneity of pigmentation (specific for photoaging)
Other features	Benign neoplasms	Premalignant lesions, actinic keratoses, telangiectasias
Histology (epidermis)	Flattening of the dermal-epidermal junction, decrease in melanocyte and Langerhans cell density	Variable epidermal thickness, some cytologic atypia, uneven distribution of melanocytes, significant decrease in Langerhans cells
Histology (dermis)	Loss of extracellular matrix, increased levels of collagen-degrading metalloproteinases, loss of fibroblasts and vascular network, and, in particular, loss of the capillary loops that occupy the dermal papillae	Dermal elastosis (deposition of abnormal amorphous elastic material in the papillary dermis), abundant inflammatory cells in the dermis, and degenerative changes in collagen and elastic fibers

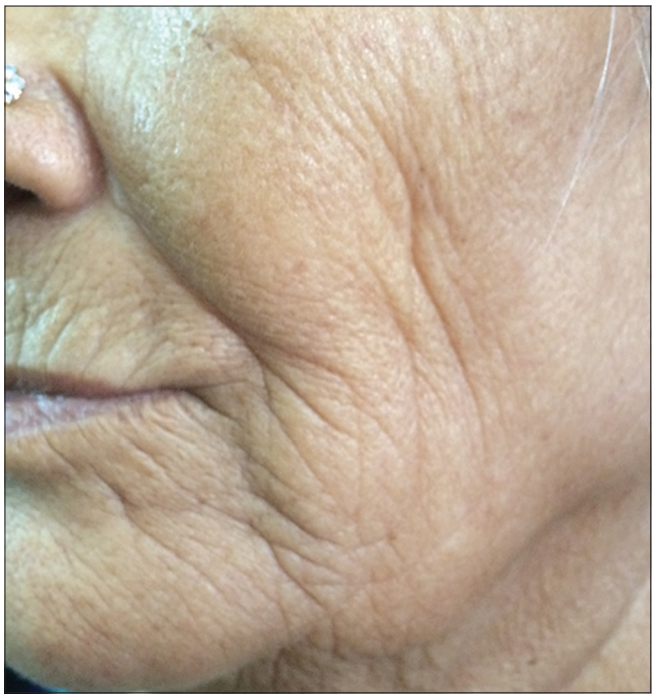

Figure 1: Intrinsic aging. Sagging and lax skin with deep wrinkles in a 63-year-old woman

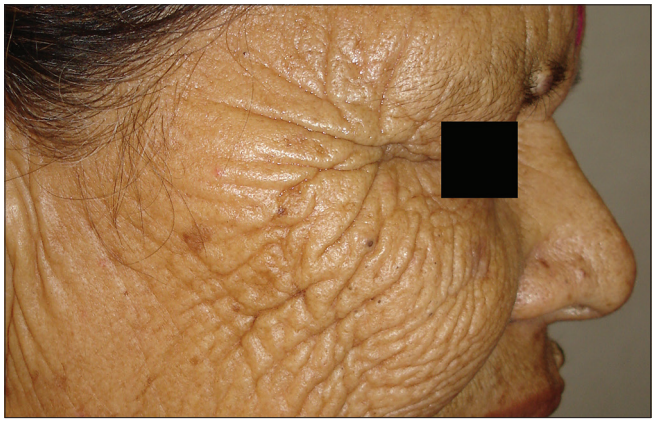

Figure 3: Extrinsic aging. Solar elastosis in a 40-year-old woman living in hilly region

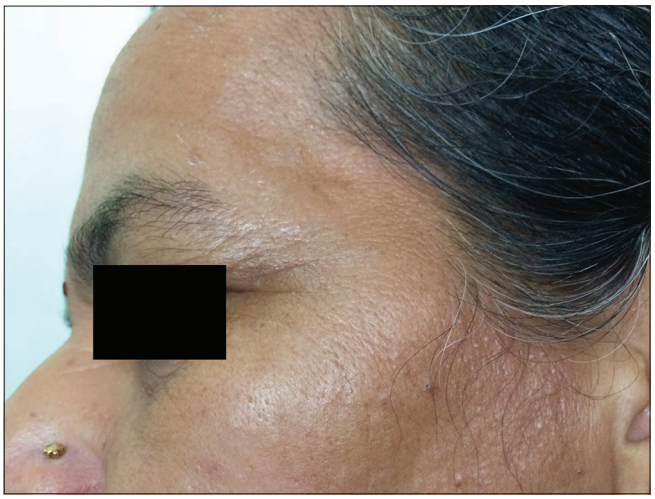

Figure 2: Intrinsic aging. Fine wrinkles in a 45-year-old woman

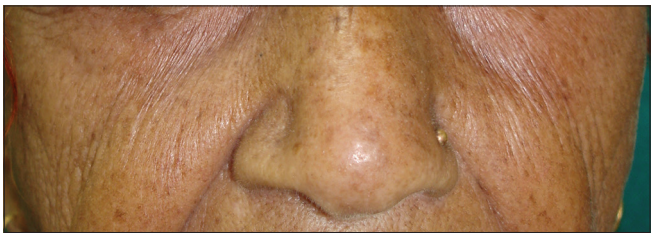

Figure 4: Extrinsic aging. Freckles in a 55-year-old woman

lamps is responsible for cumulative damage, which induces immunosuppression responsible for skin cancer.[18] Fair-skinned people are more susceptible than dark-skinned individuals. Australia has the world's highest rate of skin cancer which is due to high sunshine levels. Depletion of ozone increases the penetration of ultraviolet rays to the earth's surface, leading to an increase in incidence of skin cancers, and a decrease in their age of onset.[19,20] Ultraviolet A and B damage deoxyribonucleic acid through different mechanisms. Melanocytes in the basal layer of epidermis produce the pigment melanin, which protects the neighbouring keratinocytes from ultraviolet radiation. However, prolonged exposure to sunlight can induce carcinogenesis. Ultraviolet A and B are absorbed by proteins, lipids, nuclear and mitochondrial deoxyribonucleic acid, causing a cascade of oxidative events

which results in the deterioration of structure and function of cells. Ultraviolet A causes mutation of tumor suppressor p53 gene in the basal layer which has stem cells, and thus leads to carcinogenesis, which is due to deoxyribonucleic acid damage, gene mutation and immune suppression.

Ultraviolet A in combination with common environmental pollutants, such as polycyclic aromatic hydrocarbons, significantly increases visible photodamage in the skin.[21] Ultraviolet A in combination with ozone causes synergistic oxidative stress in human skin.[22] Some air pollutants (ozone, nitrogen dioxide and sulfur dioxide) and scattering particulates (clouds and soot) in the troposphere reduce the effects of shorter wavelength ultraviolet radiation, mainly ultraviolet B, and cause significant reduction in ultraviolet B irradiance in polluted urban areas. There is a reduction of more than 50% in ultraviolet radiation (mainly ultraviolet B) on days with high levels of air pollution. An inverse relationship exists between the total ozone content and ground levels of ultraviolet B radiation. Ultraviolet A, however, is not affected much in the presence of air pollutants and ozone. Therefore, the ratio of ultraviolet B/ultraviolet A is highly dependent on factors such as thickness of ozone layer and air pollution.[23]

Cigarette smoke

Cigarette smoke is a highly complex aerosol composed of thousands of chemical substances, including reactive oxygen species, carbon monoxide, reactive nitrogen species and electrophilic aldehydes.[3] Reactive oxidants and free radicals from cigarette smoke cause oxidative stress or secondary oxidative events and inhibition of antioxidant mechanisms.[24-26] Chemical substances from cigarette smoke increase transepidermal water loss, degeneration of connective tissue in the skin and upregulation of matrix metalloproteinases-1 and 3 which degrade collagen and elastic fibers.[3,27,28]

Smoking causes premature aging which clinically manifests as deeper periorbital wrinkling.[29-31] Premature facial skin aging in smokers, with a characteristic pattern of wrinkling and orange-purple skin discoloration, was defined as smoker's face.[32] Smoker's face typically has lines or wrinkles radiating at the right angles from the upper and lower lips or corners of the eyes, deep lines on the cheeks, or numerous shallow lines on the cheeks and lower jaw. The bony contours become prominent, and the skin is slightly pigmented gray with orange, purple and red complexion. Heavy cigarette smokers were 4.7 times more likely to have facial wrinkles than nonsmokers, independent of sun exposure, although the combination of smoking and sun exposure may have a synergistic effect.[31-33] It has been observed that wrinkling in a 40-year-old smoker resembles that of a 70-year-old nonsmoker.[34] Sometimes, large open and closed comedones with furrows (smoker's comedones) [Figure 5] are seen in the periorbital area, similar to those seen in Favre–Racouchot syndrome. There is yellow discoloration of nails, and in persons who have stopped smoking, a sharp demarcation line may be seen between the yellow nail plate and the newly formed pink nail plate (known as Harlequin nail or quitter's nail). Other changes noticed in smokers include yellowish discoloration of the hair and beard (e.g. smoker's moustache), premature graying and loss of hair, gingival pigmentation (smoker's melanosis), leukoplakia of the tongue (smoker's tongue), oral leukoplakia [Figure 6] and a gray-white keratinized palate with multiple red umbilicated papules that represent inflamed salivary glands (smoker's palate/nicotine stomatitis).[35] Various mechanisms have been postulated for

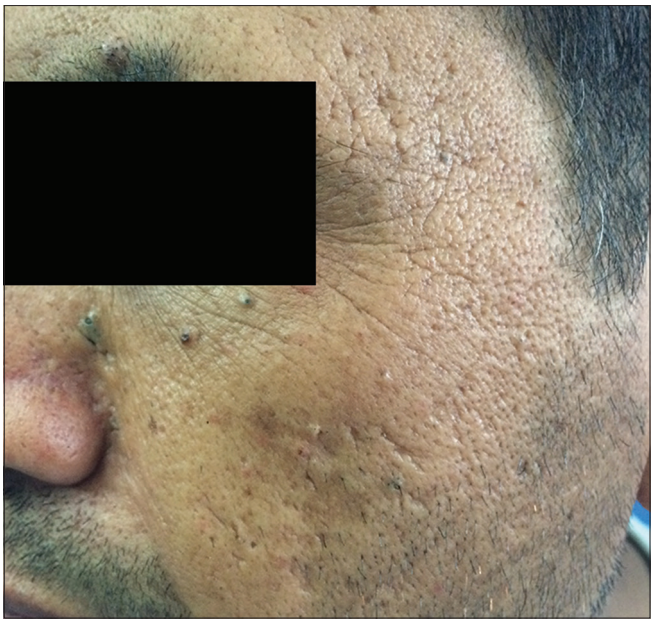

Figure 5: Smokers comedones. Comedones in periorbital area and fine wrinkles in a 35-year-old man

premature aging caused by cigarette smoke. In mice models, second-hand smoke, also known as environmental tobacco smoke, involuntary smoke and passive smoke caused premature aging by increased cytoplasmic translocation of high-mobility group box 1 protein, and hence, the loss of collagen.[36] Transcription of p16INK4a has been associated with aging, and p16INK4a is a known gerontogen. In murine models, cigarette smoke and ultraviolet light have augmented the transcription of p16INK4a.[37] Cigarette smoke extract caused senescence of fibroblasts, possibly by oxidative stress injury and inhibition of antioxidant defense activity in *in vitro* studies.[38] Cigarette smoke-induced early growth response-1 induces the expression of cysteine-rich 61 in human skin dermal fibroblasts which may be the cause of premature aging.[39]

An association between cigarette smoke and psoriasis has been reported in several epidemiologic studies. In a Norwegian cross-sectional study, male smokers had a significantly increased risk of developing psoriasis.[40] A meta-analysis suggested that there is a significant association between smoking and psoriasis with a relative risk of 1.88 for smoking in patients with psoriasis versus patients without psoriasis.[41] In addition, there is a dose-dependent relationship between the development of psoriasis and the number of cigarettes smoked. A population-based twin study showed that childhood exposure to environmental tobacco smoke was significantly associated with psoriasis in the whole population, with an odds ratio of 1.28. Smokers with a history of >5 pack-years of cigarette had an increased risk of psoriasis with an odds ratio of 2.18. The same study showed that genetic factors could explain only 20% of the correlation between psoriasis and smoking, whereas non-shared environmental factors explained even less at 8 percent.[42] Several single nucleotide polymorphisms located in the CHRNA5/A3/B4 gene cluster have been linked to smoking behaviour and nicotine dependence, but these known single nucleotide polymorphisms were not found to be linked with psoriasis incidence or severity in the Chinese population.[43] The effect of smoking could be mediated by the reactive oxygen species and by the imbalance between oxidants and antioxidants indicated by low levels of

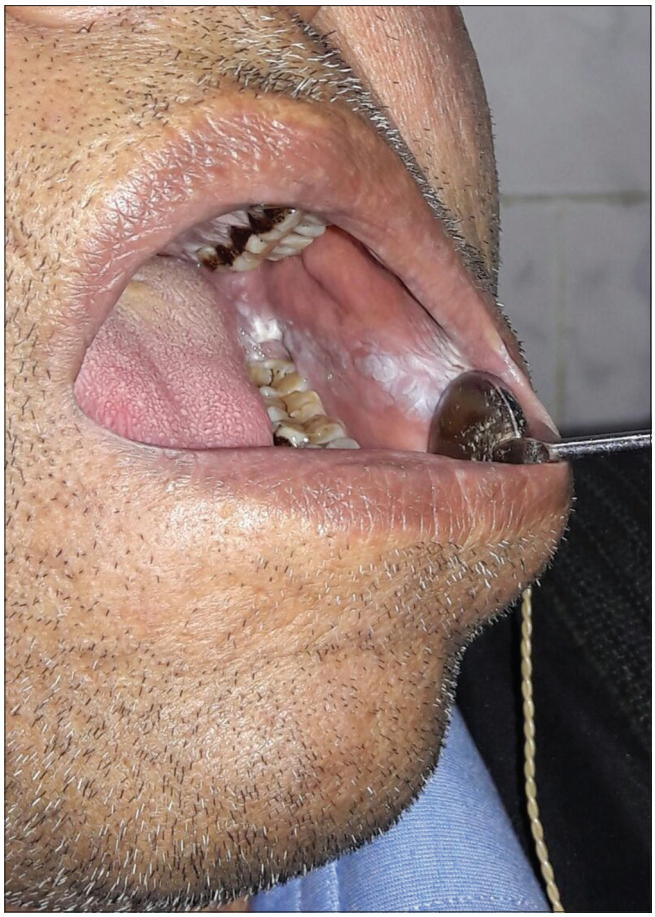

Figure 6: Oral leukokeratosis in a smoker

vitamin C and glutathione, and high levels of superoxide dismutase and malonaldehyde.[3,44]

Schäfer *et al*. reported a high prevalence of acne among smokers and described a correlation between the acne severity and the number of smoked cigarettes in post adolescent women, where predominantly comedonal acne was seen compared to the papulopustular form.[45] The authors reported that although the correlation between acne and smoking is still controversial, there is a hyperkeratinizing effect of cigarette smoke compounds, and in particular, of nicotine. Nicotine, an agonist of acetylcholine present in the cigarette, induces comedogenesis via the stimulation of acetylcholine-nicotinic receptor on epidermal keratinocytes.[46]

A meta-analysis done in 2010 shows that tobacco smoking is associated with cutaneous squamous cell carcinoma with an odds ratio of 1.52 while the association between smoking and basal cell carcinoma and other nonmelanoma skin cancers is controversial.[3,25,47-50] In mice models, 4-(methylnitrosoamino)-1-(3-pyridyl)-1-butanone acted as tumor initiator causing skin cancer and lung adenomas.[19]

Interestingly, many case series and case-control studies report that smoking is associated with a lower prevalence of aphthous ulcers, Behcet's disease, herpes labialis, Kaposi's sarcoma (in acquired immune deficiency syndrome) and pemphigus vulgaris.[49] The various skin diseases aggravated by smoking are tabulated in Table 3.

Table 3: Skin diseases aggravated by cigarette smoke

Commonly associated skin changes
Premature skin aging
Hyperpigmentation of oral mucosa
Submucous fibrosis
Yellowish discoloration of nails
Smokers comedones
Skin diseases with definite evidence of aggravation with smoking
Cutaneous squamous cell carcinoma
Hidradenitis suppurativa
Systemic lupus erythematosus
Oral cancers
Palmoplantar pustulosis
Psoriasis
Skin diseases with conflicting evidence of aggravation with smoking
Basal cell carcinoma
Discoid lupus erythematosus
Acne

Polycyclic aromatic hydrocarbons

Polycyclic aromatic hydrocarbons are among the most widespread and dangerous organic pollutants.[20] Polycyclic aromatic hydrocarbons are absorbed on the surface of suspended particulate matter in the air in urban areas.[51] They are converted into quinines, redox-cycling chemicals that produce reactive oxygen species.[52] Irrespective of the route of entry in the human body, they are found in almost all the internal organs, especially in the lungs and digestive tract. The main source of atmospheric polycyclic aromatic hydrocarbon benzo(a) pyrene is residual wood burning, the other sources being automobile exhaust, diesel fumes, metallurgical industry, production of plastics, pesticides, dyes, cigarette smoke and smoke resulting from the combustion of organic material.[21]

Polycyclic aromatic hydrocarbons are associated with extrinsic skin aging, pigmentation, cancers and acneiform eruption. Melanocyte proliferation and skin pigmentation have been observed in mice.[53] Scrotal cell carcinoma due to coal soot in British chimney sweeps was reported in 1775, and polycyclic aromatic hydrocarbon was the carcinogen responsible for it. Coal soot is more carcinogenic than wood soot as it contains a higher amount of polycyclic aromatic hydrocarbons. Among polycyclic aromatic hydrocarbons, benzo(a) pyrene has been shown to cause nonmelanoma cancers whereas 7,12-dimethylbenz(a) anthracene is capable of inducing lymphoma in hamsters. Ultraviolet A enhances the carcinogenic action of benzo(a) pyrene.[54] However, Sowada *et al*. have demonstrated that resident skin flora, predominantly micrococci, can degrade benzo(a) pyrene, thus forming an innate mechanism of defense against polycyclic aromatic hydrocarbons.[55,56]

Epoxides and diols produced by activated polycyclic aromatic hydrocarbons bind to deoxyribonucleic acid, leading to carcinogenesis.[7,54] Polycyclic aromatic hydrocarbons can lead to acneiform eruptions due to the presence of 2,3,7,8-tetrachlorodibenzo-p-dioxin which is a polyhalogenated aromatic hydrocarbon.[7] It is formed in any burning, waste incineration, metal production and fossil-fuel and wood combustion. Sorg *et al*. described chloracne in Viktor Yushchenko's poisoning with 2,3,7,8-tetrachlorodibenzo-p-dioxin in 2005.[57] Chloracne is

a systemic toxic disease caused by the exposure to halogenated aromatic hydrocarbons. It is characterized by comedones and cysts mainly on the face (outer sides of the eye and behind the ears) and neck. Other manifestations of chloracne include fatigue, liver dysfunction, neuropathy and arthritis.[58]

Ground-level ozone

Ozone is a ubiquitous pollutant in the urban environment. Its concentrations in urban environment can range from 0.2 to 1.2 ppm.[59] Mexico City has the highest ozone levels in the world. It is a gaseous oxidant that exists in the stratosphere and troposphere.[70,50] Normally, the concentrations of ozone at ground-level are low. Ozone, after interaction with sunlight (ultraviolet radiation), hydrocarbons, volatile organic compounds and nitrogen oxides, forms a major active component of the photochemical smog.[7,50]

The effect of ozone is mediated by its ability to induce oxidative stress. It leads to the formation of peroxides, aldehydes and lipid ozonation products, as a result of unsaturated fatty acid oxidation and damages the barrier function of epidermis. Thiele *et al*. reported that ozone causes a reduction in the level of antioxidants such as tocopherol (vitamin E) and ascorbic acid (vitamin C) and increases malondialdehyde, a lipid peroxidation product in murine skin causing impairment of barrier function and inflammation.[22,60] In human skin, exposure to ozone caused a 70% decrease in vitamin E concentration in stratum corneum, and 50% reduction in skin microflora.[61] Ozone causes disturbed activity of matrix metalloproteinases. Ozone-induced inflammation is mainly mediated through redox-sensitive transcription factor, nuclear factor kappa B. In an *in vitro* study, cells exposed to ozone demonstrated a dose-dependent increase in p65 subunit nuclear expression as a marker of nuclear factor kappa B activation, while pretreatment with vitamin C mixtures which acted as antioxidants abolished nuclear factor kappa B nuclear translocation. In addition, a significant activation of Nrf2 was observed in keratinocytes treated with the mixtures.[62] Nrf2 is a basic leucine zipper protein that regulates the expression of antioxidant proteins that protect against oxidative damage triggered by injury and inflammation.

Ozone along with ultraviolet A rays and cigarette smoke is a powerful oxidizing agent of squalene. Oxidation of squalene produces squalene by-products, mostly peroxidized forms, which lead to comedogenesis, thus aggravating inflammatory acne. Oxidation of epidermal lipids and disturbed activity of matrix metalloproteinases contribute to wrinkling and extrinsic skin aging.[63] Tropospheric ozone exposure has been associated with urticaria, eczema, contact dermatitis, rashes and infected skin disease.[64]

Particulate matter

Particulate matter in the air consists of complex and varying mixtures of different size and composition. Factories, power plants, refuse incinerators, automobile, construction activities, fires and natural windblown dust are some of the main sources of particulate matter.[65,66]

Particulate matter penetrates skin either through hair follicles or transdermally, and exerts its detrimental effects through the generation of oxidative stress, which contributes to extrinsic skin aging, characterized particularly by pigment spots on the face and nasolabial folds, and less so by coarse wrinkles, solar elastosis and telangiectasia.[67-69] The most harmful components of ambient particulate matter are nanosize particles from traffic sources; these particles can serve as carriers for organic chemicals and metals that are capable of localizing in mitochondria and generating reactive oxygen species. It has been noted that an increase in soot (per 0.5×10^{-5}/m) and particles from traffic (per 475 kg per year and square km) was associated with 20% more pigment spots on the forehead and cheeks. Polycyclic aromatic hydrocarbons are adsorbed on the surface of suspended particulate matter in air of urban areas.[68] Polycyclic aromatic hydrocarbons can activate xenobiotic metabolism which converts polycyclic aromatic hydrocarbons to quinones. Quinones are redox-cycling chemicals which produce reactive oxygen species responsible for particulate matter toxicity.[52]

Although many cohort studies report that there is no association between air pollutants and incidence and prevalence of atopic dermatitis, the severity of symptoms of atopic dermatitis may have a direct association with increased particulate matter.[70] Kim *et al*. also report that with indoor air quality improvement program, the level of particulate matter decreased and there was a significant decrease in the prevalence and severity of atopic dermatitis.[71] The exact mechanism is unclear, but it is proposed that particulate matter may induce inflammation in the skin in a similar fashion as that in the respiratory system.[70]

Volatile organic compounds

Emission of volatile organic compounds occurs from the use of organic solvents in paints, varnishes (aliphatic hydrocarbons, ethyl acetate, glycol ethers, methylene chloride and acetone) vehicle refinishing products in repairing car paint, environmental tobacco smoke, stored fuels, exhaust from cars (benzene) and from emissions from industrial facilities (tetrachloroethylene).[6,72,73] It is an important indoor source of air pollutants. A longitudinal study has shown that symptoms of atopic dermatitis increase in children shifted to a new building due to an increase in exposure to volatile organic compounds.[74] Volatile organic compounds along with sunlight and nitrogen oxides form photochemical oxidant products such as ozone at ground level which is the summer photochemical smog. Volatile organic compounds (ingestion of hexachlorobenzene) may induce precancerous skin lesions in rats.[75] Exposure to volatile organic compounds increases cytokines (interleukin-8 and interleukin-1B) in cultured keratinocytes which cause atopic dermatitis or eczema.

Oxides

Nitrogen oxides are emitted mainly from mobile and stationary combustion sources. They react with ozone-forming nitrogen dioxide. Among nitrogen oxides, nitrogen dioxide causes oxidative damage leading to the formation of free radicals that oxidize amino acids in tissue proteins and initiate lipid peroxidation of polyunsaturated fatty acids.[76]

Atmospheric sulfur dioxide is formed from fuel combustion from industrial processes, volcanic activity and forest fires. Carbon monoxide, a product of incomplete combustion from mobile sources, acts on cell metabolism which binds to heme and alters its function.[6] Flexural eczema was associated with traffic-related air pollutants, including nitrogen oxides and carbon monoxide in Taiwan in middle-school children. A study comparing atopic eczema in East and West Germany showed that the prevalence was higher in East Germany (sulfurous type pollution) and also exhibited a stronger association with nitrogen oxides and close proximity to heavy traffic.[77]

Heavy metals

Cadmium, lead and mercury are common air pollutants that pose health hazards. The main sources are volcanoes, waste incineration, cement, iron and steel production and leaded gasoline.[56]

Prevention Strategies

Control of air pollution is necessary to improve the health conditions. There are two steps in the prevention of dermatological diseases due to air pollution: the first is to reduce air pollution and the second is to use strategies to protect oneself from pollutants. In Korea, indoor air quality improvement program was conducted in nine kindergarten classes, following which mean particulate matter 10 levels decreased significantly from 182.7 to 73.4 μg/m^3. Along with that, the prevalence, severity of atopic dermatitis in children and the number of hospital visits per month also decreased significantly, thus showing the benefit of improvement of air quality.[71] The Health Event project in Europe for the improvement of indoor air quality is based on three components, namely optimizing ventilation rates, filtration of outdoor air and indoor source control.[78] This project has shown an improvement in different cardiovascular and respiratory diseases. Such strategies should be studied for dermatological diseases too.

Control of air pollution

The natural sources of pollution are difficult to predict and prevent such as volcano eruptions or forest fires. However, human-made sources can be controlled. Some strategies include less use of personal vehicles, increase in the use of car pools and public modes of transport, supply of low-sulphur petrol, shifting of industries to areas away from the cities, development and usage of industrial machines and methods which are eco-friendly, avoiding burning of garbage in the open, avoidance of smoking and no use of wood and crop residues as fuel for the purpose of household cooking and heating. Various methods have been tried to reduce traffic induced pollution. For example, in New Delhi, biofriendly fuels such as compressed natural gas is used by all public transport vehicles, odd-even formula for private vehicles has been implemented and old diesel vehicles are gradually being phased out.

Personal protection

Strategies for personal protection include physical photoprotection; use of sunscreens; avoidance of areas with public smoking, and around industries; usage of topical antioxidants such as vitamin C and E in formulations along with sunscreen; and of indoor air purifiers and ventilators. People with high occupational risk, such as traffic police and sweepers, should use masks while at work.

Conclusions

Skin is the largest organ of human body, and any factor affecting skin health will impact the body as a whole. Major air pollutants having detrimental effects on the skin include solar ultraviolet radiation, polycyclic aromatic hydrocarbons, volatile organic compounds, nitrogen oxides, particulate matter, ozone and cigarette smoke. Sunlight, cigarette smoke and ambient particulate matter have a role to play in extrinsic skin aging. Smoking has also been associated with skin cancer, psoriasis, acne and skin malignancy. Exposure to ozone has been associated with urticaria, eczema, contact dermatitis and other nonspecific eruptions. Polyaromatic hydrocarbons cause skin cancer, extrinsic skin aging, pigmentation and acneiform eruptions. Oxides have been associated with increased prevalence, as well as exacerbations of atopic dermatitis in children.

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Conflicts of interest

There are no conflicts of interest.

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