



# Exploring the role of anthropogenic air pollutants in the pathogenesis of acne

Badanie roli antropogenicznych zanieczyszczeń powietrza w patogenezie trądziku

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## Abstract

**Introduction and Objective.** Increasing global urbanization has led to significant atmospheric air pollution. Airborne pollutants, including PM, O<sub>3</sub>, CO, NO<sub>x</sub>, and SO<sub>2</sub>, exert detrimental effects on the entire body, contributing to various skin diseases. The aim of this study was to review the literature on the effects of selected air pollutants on the development or exacerbation of acne vulgaris, and to elucidate the mechanisms responsible.

**Brief description of the state of knowledge.** Existing studies offer insights into the substantial impact of anthropogenic air pollution on the onset and aggravation of acne. Pollution particles affect the skin on various levels, compromising the protective sebum layer, triggering oxidative stress and inflammatory responses, and instigating noteworthy alterations in skin cell structures.

**Summary.** Reduction in the prevailing levels of anthropogenic air pollutants is crucial for curbing the incidence of acne and enhancing public health. This skin condition detrimentally impacts the quality of life and the mental well-being of those affected, particularly in an era that champions an idealized appearance. The incorporation of substances with antioxidant and anti-inflammatory properties, fortifying the skin barrier, could also prove beneficial. Further research is imperative to deepen our understanding of the relationship between air pollution and acne, as well as to explore effective strategies for shielding the skin from pollution.

## Key words

air pollutions, acne, skin

## Streszczenie

**Wprowadzenie i cel pracy.** Postępująca globalna urbanizacja doprowadziła do znacznego zanieczyszczenia powietrza atmosferycznego. Występujące w powietrzu zanieczyszczenia, takie jak PM, O<sub>3</sub>, CO, NO<sub>x</sub>, SO<sub>2</sub>, oddziałują negatywnie na cały organizm, wywołują też różne choroby skóry. My skupimy się na wpływie zanieczyszczeń powietrza na rozwój oraz zaostrzenie trądziku. Celem pracy był przegląd literatury na temat wpływu wybranych zanieczyszczeń powietrza na rozwój lub zaostrzenie trądziku, a także wyjaśnienie mechanizmów, które za to odpowiadają.

**Opis stanu wiedzy.** Dostępne badania dostarczyły informacji na temat istotnego wpływu antropogenicznych zanieczyszczeń powietrza na rozwój i zaostrzenie trądziku. Cząstki zanieczyszczeń oddziałują na skórę na wielu poziomach, niszcząc pokrywającą ją ochronną warstwę sebum, wywołując stres oksydacyjny i stany zapalne oraz powodując znaczące zmiany w strukturze komórek skóry.

**Podsumowanie.** Redukcja aktualnego natężenia antropogenicznych zanieczyszczeń powietrza ma kluczowe znaczenie dla poprawy ogólnego stanu zdrowia publicznego i zmniejszenia częstości występowania trądziku, który wpływa niekorzystnie na jakość życia i psychikę osób nim dotkniętych, co stanowi problem zwłaszcza w dzisiejszych czasach, promujących idealny wygląd. Korzystne może okazać się również stosowanie substancji o właściwościach przeciwutleniających i przeciwzapalnych, wzmacniających barierę skórą. Niezbędne są dalsze badania, aby pogłębić nasze zrozumienie związku między zanieczyszczeniem powietrza a trądzikiem, a także zbadać skuteczne strategie ochrony skóry przed zanieczyszczeniami.

## Słowa kluczowe

zanieczyszczenia powietrza, trądzik, skóra

## INTRODUCTION

Acne vulgaris, an inflammatory skin condition of multifaceted origin, is marked by heightened sebum secretion, an inflammatory response to Cutibacterium

Acnes, and excessive keratinization of hair follicles [1, 2]. The pathogenesis of acne encompasses genetic, hormonal, and environmental factors. Among the environmental factors, attention is directed towards exploring the impact of anthropogenic air pollution on the initiation and aggravation of acne.

The surge in global urbanization has given rise to considerable atmospheric air pollution. The five main air pollutants listed by the WHO as harmful to public health are:

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particulate matter (mainly PM<sub>10</sub> and PM<sub>2.5</sub>), nitrogen oxides (NO<sub>x</sub>), carbon monoxide (CO), sulphur dioxide (SO<sub>2</sub>) and ozone (O<sub>3</sub>). They are mostly formed as a result of combustion processes (PM, NO<sub>x</sub>, CO, SO<sub>2</sub>) or chemical reactions between gases (O<sub>3</sub> and PM). Sources of these pollutants are primarily broadly human activities, such as heating, transportation, power generation and industry [3]. Air pollutants negatively affect the entire body, and cause various skin diseases, including acne.

## OBJECTIVE

The aim of this study was to conduct a literature review focusing on the impact of specific air pollutants on the initiation or worsening of acne, in order to elucidate the underlying mechanisms.

## MATERIALS AND METHOD

A literature search was conducted using the medical databases PubMed and Google Scholar. Articles were retrieved in English, employing the key words: 'air pollutions', 'acne', 'skin' in appropriate configurations. The analysis encompassed data from 38 scientific references published between 2008 and 2023.

## DESCRIPTION OF THE STATE OF KNOWLEDGE

Ambient air pollution is undoubtedly associated with the occurrence or exacerbation of acne in exposed individuals, as evidenced by the studies cited below.

A study using data from 2010–2013 from the city of Xi'an, China, on the frequency of outpatient visits for post-adolescent acne and corresponding air pollution data, showed that a 10 µg/m<sup>3</sup> increase in PM<sub>10</sub>, SO<sub>2</sub> and NO<sub>2</sub> concentrations at a lag of 0–7 days was associated with a higher frequency of outpatient visits for post-adolescent acne. At the same time, women were more sensitive to the effects of PM<sub>10</sub> than men. However, there was no difference in the effects of SO<sub>2</sub> and NO<sub>2</sub> in either gender [4].

In 2020, a cross-sectional study of 372 adults aged 18–55 in Lebanon using an online survey, found a significant association between the occurrence of inflammatory acne and exposure to high concentrations of NO<sub>2</sub> in ambient air and employment near a power plant emitting PM, NO<sub>2</sub>, CO<sub>2</sub>, CO and SO<sub>2</sub> [5]. In Beijing, China, during a 2-year period a study conducted in collaboration with La Roche-Posay Cosmetic Laboratories explored the association between concentrations of prevalent air pollutants (PM<sub>10</sub>, PM<sub>2.5</sub>, NO<sub>2</sub>, SO<sub>2</sub>) and the frequency of outpatient visits for acne vulgaris. Elevated levels of PM<sub>10</sub>, PM<sub>2.5</sub>, and NO<sub>2</sub> were linked to increased sebum secretion and a higher incidence of acne lesions. Conversely, for SO<sub>2</sub>, a markedly lower occurrence of outpatient visits for acne vulgaris was noted at higher concentrations, suggesting a potential protective role of SO<sub>2</sub> in the pathogenesis of this condition [6].

**How air pollution affects the skin.** The skin, the body's largest organ, plays a crucial role in mediating contact between the internal and external environments, serving

as a protective barrier against harmful external agents. Consequently, it stands as the primary target for the impacts of air pollution on the body. Prolonged and intense exposure to environmental stressors can disrupt the skin's natural protective function, contributing to the onset of various skin diseases, including acne.

Airborne contaminants can penetrate the skin either directly or indirectly. Substances such as particulate matter (PM), polycyclic aromatic hydrocarbons (PAHs) and ground-level ozone (O<sub>3</sub>) can be absorbed directly by the skin through trans-epidermal absorption or via hair follicles and sweat ducts. These pollutants subsequently accumulate in the stratum corneum and penetrate into the deeper layers of the skin. Indirect distribution to the dermis and subcutaneous tissue of air pollutants entering the body by inhalation and ingestion (among others PM) occurs via systemic circulation [7]. Air pollutants are involved in the pathogenesis of acne vulgaris in several ways: through changes in sebum composition, effects on the skin microflora, production of reactive oxygen species (ROS), induction of the inflammatory response, and activation of the aryl hydrocarbon receptor (AhR). Air pollutants also increase sebum secretion which can aggravate acne [6]. They also negatively affect sebum quality.

A study comparing the facial skin parameters of people from Mexico City – where high levels of air pollution have been demonstrated – with those from Cuernavaca, with (less air pollution, showed that the skin of those living in the city with high air pollution was characterized by increased and faster sebum secretion, compared to the skin of those less exposed to pollution. The residents of Mexico City, compared to those in Cuernavaca, also showed significant changes in sebum composition, such as reduced amounts of squalene and vitamin E, as well as higher levels of lactic acid, while the stratum corneum showed the presence of numerous carbonylated proteins, high levels of ATP and lower levels of IL-1α [8].

In 2008, a similar study conducted in Shanghai, China, investigated the impact of urban pollution on the skin, compared residents from the central, more polluted area with those from a rural district with lower air pollution levels. The results revealed that individuals in less air-polluted areas exhibited biochemical parameters indicative of skin health, including an elevated squalene-to-lipid ratio, lower lactic acid content, and improved stratum corneum integrity, in contrast to those living in the city centre. However, no significant difference was observed in the level and rate of sebum secretion between the two groups [9].

The varying findings on the rate of sebum secretion among individuals exposed to air pollution in the mentioned studies may be attributed to differences in skin parameters, with participants being of Caucasian descent (Mexico) and Asian descent (China), as well as disparities in the air pollution composition in these regions. Nonetheless, the studies affirm that air pollution leads to a reduction in natural antioxidant substances present in sebum, such as vitamin E and squalene. Elevated levels of ATP and oxidized proteins in the stratum corneum among individuals exposed to high levels of outdoor pollution suggest damage to this skin layer. Collectively, these factors compromise the skin's normal protective functions and may influence the onset or exacerbation of acne.

O<sub>3</sub> and PAHs are recognized culprits in squalene oxidation, a phenomenon notably heightened in the presence of UV

radiation. Squalene, among the compounds governing the skin's oxygen balance, undergoes peroxidation, fostering the creation of an anaerobic environment [10]. Furthermore, the direct blockage of skin pores by air pollution particles also promotes the development of an anaerobic environment, leading to excessive proliferation of anaerobic bacteria like *Cutibacterium acnes* (formerly *Propionibacterium acnes*). In response to the heightened growth of *Cutibacterium acnes*, hair follicle keratinocytes release inflammatory cytokines, thus encouraging the formation of pustules [11]. It is also known that ozone, through its bactericidal effects, can cause the loss of up to half of the normal skin microbiome [12]. Additionally, polycyclic aromatic hydrocarbons (PAHs) have the potential to bring about detrimental alterations in the composition and functionality of the skin microbiota [13, 14]. This could be associated with acne, as demonstrated by a study on the skin microbiota in individuals with acne, which revealed significant changes in the composition of the skin microbial community throughout the course of the disease [15].

Most air pollutants, including O<sub>3</sub> and NO<sub>x</sub>, exert pro-oxidative effects on the skin. The resulting free radicals and reactive oxygen species cause lipid peroxidation in skin cells, which induces the release of inflammatory factors. These engage phagocytic cells of the immune system, which further exacerbate oxidative stress by producing free radicals [16]. Reactive oxygen species (ROS) and lipid peroxidation products formed in the skin after exposure to air pollution can activate inflammasomes, involved in the inflammatory response of the skin [17]. Inflammasomes are protein complexes present in the cytosol of cells that are responsible for the innate immune response by releasing inflammatory cytokines: IL-1 B, IL-18. The afore-mentioned interleukins protect the body from harmful agents, such as air pollutants [18]. On the other hand, it has been proven that excessive release of IL-1 B is crucial in the pathogenesis of acne, and significantly affects its exacerbation [19].

Studies have shown that O<sub>3</sub> can cause activation of the NLRP1 inflammasome through redox regulation [20]. In addition, *Propionibacterium Acnes* has been proven to activate the NLRP3 inflammasome in acne lesions [21].

The inflammatory response in the skin is also induced by polycyclic aromatic hydrocarbons (PAHs), commonly found in air pollution and carried by particulate matter. Benzo[a]pyrene – a PAH formed by burning wood, organic matter and found in car exhausts, among others – can induce the secretion of IL-8 from keratinocytes [22]. Also, PM and heavy metal particles can increase the expression of interleukin-6 and interleukin-8. These compounds additionally increase the levels of caspase-14 in the epidermis, which is responsible for keratinization of the upper layers of the epidermis and skin hydration [23].

It is worth noting that increased levels of ROS, IL-8 and IL-6 have been observed in the skin of patients with inflammatory acne [24, 25].

Ozone and PAHs activate the aryl hydrocarbon receptor (AhR) [26, 27], which is a transcription factor responsible for the expression of cytochrome P450, CYP1A1 and CYP1A2 genes involved in xenobiotic metabolism [28]. AhR activation in keratinocyte cells leads to increased inflammation, mediated by interleukin-8 [29] and cyclooxygenase 2 [30] in response to reactive oxygen species, which can further aggravate acne.

## SUMMARY

Clinical studies unequivocally confirm a positive correlation between exposure to ambient air pollutants and the occurrence or exacerbation of acne. While individual air pollutants impact the skin in slightly different ways, the majority induce or exacerbate acne by altering the rate and composition of sebum secretion, influencing the skin's microflora, inducing oxidative stress, and triggering inflammation. The sole air pollutant demonstrated to exhibit a protective function against acne is SO<sub>2</sub> [6], a finding consistent with other studies that highlight the efficacy of sulfur in acne treatment [31].

Moreover, the topical application of formulations containing antioxidants, such as vitamin C and vitamin E, which inhibit lipid peroxidation and inflammation, may prove beneficial in preventing the detrimental effects of air pollution on acne development.

Additionally, it is noteworthy that natural substances like curcumin and resveratrol have demonstrated potent inhibition of inflammasome-mediated IL-1B synthesis of NLRP3, both *in vivo* and *in vitro* [32, 33, 34]. Research has also indicated the potential effectiveness of certain antioxidants with inflammasome-targeted effects in treating acne [35, 36, 37]. Promising anti-acne properties have also been demonstrated by emodin, which combines all of these features, leading to a reduction in the secretion of the pro-inflammatory cytokines IL-6, IL-8, IL-1B and IL-18, and inhibiting the expression of the NLRP3 inflammasome in sebocytes exposed to *Cutibacterium acnes* [38].

Further investigation is warranted to explore the correlation between air pollution and acne. Additionally, there is a need for studies aimed at developing effective strategies to protect the skin from the adverse effects of pollution. Undoubtedly, the best way to reduce the incidence of acne vulgaris, reduce the severity of the disease's symptoms and improve the overall health conditions of the population would be to reduce the amount of man-made air pollution through concerted global action.

## REFERENCES

1. Zaenglein AL, Pathy AL, Schlosser BJ, et al. Guidelines of care for the management of acne vulgaris. *J Am Acad Dermatol*. 2016;74(5):945–973. e33.
2. Dréno B, Pécastaings S, Corvec S, et al. *Cutibacterium acnes* (*Propionibacterium acnes*) and acne vulgaris: a brief look at the latest updates. *J Eur Acad Dermatol Venereol*. 2018;32(S2):5–14.
3. World Health Organization. Types of pollutants [internet]. Available from: <https://www.who.int/teams/environment-climate-change-and-health/air-quality-and-health/health-impacts/types-of-pollutants> (access 2023.12.09)
4. Li X, An SJ, Liu XL, et al. The association between short-term air pollution exposure and post-adolescent acne: the evidence from a time series analysis in Xi'an, China 2021. *Clinic Cosmet Investig Dermatol*. 2021;723–731
5. El Haddad C, Gerbaca NE, Hallit S, et al. Association between exposure to ambient air pollution and occurrence of inflammatory acne in the adult population. *BMC Pub Health*. 2021;21(1):1–14.
6. Liu W, Pan X, Vierkotter A, et al. A time-series study of the effect of air pollution on outpatient visits for acne vulgaris in Beijing. *Skin Pharmacol Physiol*. 2018;31(2):107–113.
7. Krutmann J, Liu W, Li L, et al. Pollution and skin: from epidemiological and mechanistic studies to clinical implications. *J Dermatol Sci*. 2014;76:163–168.
8. Lefebvre MA, Pham DM, Boussouira B, et al. Evaluation of the impact of urban pollution on the quality of skin: a multicentre study in Mexico. *Int J Cosmet Sci*. 2015;37(3):329–338.

9. Lefebvre MA, Pham DM, Boussouira B, et al. Consequences of urban pollution upon skin status. A controlled study in Shanghai area. *Int J Cosmet Sci.* 2016;38:217–223.
10. Butman JL, Thomson RJ, Geiger FM. Unanticipated hydrophobicity increases of squalene and human skin oil films upon ozone exposure. *J Phys Chem B.* 2022;126(45):9417–9423.
11. Danby FW. Ductal hypoxia in acne: is it the missing link between comedogenesis and inflammation? *J Am Acad Dermatol.* 2014;70(5):948–949.
12. Mancebo SE, Wang SQ. Recognizing the impact of ambient air pollution on skin health. *J Eur Acad Dermatol Venerol.* 2015;29: 2326–2332.
13. Alam J, Yadav VK, Yadav KK, et al. Recent advances in methods for the recovery of carbon nanominerals and polyaromatic hydrocarbons from coal fly ash and their emerging applications. *Crystals.* 2021;11(2):88.
14. Leung MHY, Tong X, Bastien P, et al. Changes of the human skin microbiota upon chronic exposure to polycyclic aromatic hydrocarbon pollutants. *Microbiome.* 2020;8(1):1–17.
15. Sun L, Wang H, Huang J, et al. A cross-sectional cohort study on the skin microbiota in patients with different acne duration. *Exp Dermatol.* 2023;32(12):2102–2111.
16. Valacchi G, Sticozzi C, Pecorelli A, et al. Cutaneous responses to environmental stressors. *Ann N Y Acad Sci.* 2012;1271(1):75–81.
17. Ferrara F, Prieux R, Woodby B, et al. Inflammasome Activation in Pollution-Induced Skin Conditions. *Plast Reconstr Surg.* 2021;147(1S–2):15S–24S.
18. Rathinam VAK, Chan FK. Inflammasome, Inflammation, and Tissue Homeostasis. *Trends Mol Med.* 2018;24:304–318.
19. ElAttar Y, Mourad B, Alngomy HA, et al. Study of interleukin-1 beta expression in acne vulgaris and acne scars. *J Cosmet Dermatol.* 2022;21(10):4864–4870.
20. Ferrara F, Pambianchi E, Pecorelli A, et al. Redox regulation of cutaneous inflammasome by ozone exposure. *Free Radic Biol Med.* 2020;152: 561–570.
21. Li ZJ, Choi DK, Sohn KC, et al. Propionibacterium acnes activates the NLRP3 inflammasome in human sebocytes. *J Invest Dermatol.* 2014;134:2747–2756.
22. Zi Y, Jiang B, He C, et al. Lentinan inhibits oxidative stress and inflammatory cytokine production induced by benzo(a)pyrene in human keratinocytes. *J Cosmet Dermatol.* 2020;19(2):502–507.
23. Park SY, Byun EJ, Lee JD, et al. Air Pollution, Autophagy, and Skin Aging: Impact of Particulate Matter (PM10) on Human Dermal Fibroblasts. *Int J Mol Sci.* 2018;19(8):2254.
24. Suvanprakorn P, Tongyen T, Prakhongcheep O, et al. Establishment of an Anti-acne Vulgaris Evaluation Method Based on TLR2 and TLR4-mediated Interleukin-8 Production. *In Vivo.* 2019;33(6):1929–1934.
25. Chen X, Min S, Chen C, et al. Influence of RETN, IL-1, and IL-6 gene polymorphisms on the risk of acne vulgaris in the Chinese population. *J Cosmet Dermat.* 2022;21(10):4965–4973.
26. Pambianchi E, Hagenberg Z, Pecorelli A, et al. Tension as a key factor in skin responses to pollution. *Sci Rep.* 2023;13(1):16013.
27. Ahn EK, Yoon HK, Jee BK, et al. COX-2 expression and inflammatory effects by diesel exhaust particles in vitro and in vivo. *Toxicol Lett.* 2008;176(3):178–187.
28. Vogel CFA, VanWinkle LS, Esser C, et al. The aryl hydrocarbon receptor as a target of environmental stressors – Implications for pollution mediated stress and inflammatory responses. *Redox Biol.* 2020; 34:11530.
29. Tsuji G, Takahara M, Uchi H, et al. An environmental contaminant, benzo(a)pyrene, induces oxidative stress-mediated interleukin-8 production in human keratinocytes via the aryl hydrocarbon receptor signaling pathway. *J Dermatol Sci.* 2011;62:42–49.
30. Lee CW, Lin ZC, Hu SC, et al. Urban particulate matter down-regulates flaggrin via COX2 expression/PGE2 production leading to skin barrier dysfunction. *Sci Rep.* 2016;6(1):27995.
31. Hashem NM, Hosny A, Abdelrahman AA, et al. Antimicrobial activities encountered by sulfur nanoparticles combating Staphylococcal species harboring sccmecA recovered from acne vulgaris *AIMS Microbiol.* 2021;7(4):481
32. Gruber JV, Holtz R. In vitro expression of NLRP inflammasome-induced active Caspase-1 expression in normal human epidermal keratinocytes (NHEK) by various exogenous threats and subsequent inhibition by naturally derived ingredient blends. *J Inflamm Res.* 2019;12:219–230.
33. Chang YP, Ka SM, Hsu WH, et al. Resveratrol inhibits NLRP3 inflammasome activation by preserving mitochondrial integrity and augmenting autophagy. *J Cell Physiol.* 2015;230:1567–1579.
34. Kong F, Ye B, Cao J, et al. Curcumin represses NLRP3 inflammasome activation via TLR4/MyD88/NF-κB and P2X7R signaling in PMA-induced macrophages. *Front Pharmacol.* 2016;7:369.
35. Yang G, Lee HE, Yeon SH, et al. Licochalcone A attenuates acne symptoms mediated by suppression of NLRP3 inflammasome. *Phytother Res.* 2018;32:2551–2559.
36. Guo M, An F, Yu H, et al. Comparative effects of schisandrin A, B, and C on propionibacterium acnes-induced, NLRP3 inflammasome activation-mediated IL-1β secretion and pyroptosis. *Biomed Pharmacother.* 2017;96:129–136.
37. Yang G, Lee SJ, Kang HC, et al. Repurposing auranofin, an antirheumatic gold compound, to treat acne vulgaris by targeting the NLRP3 inflammasome. *Biomol Ther.* 2020;28(5):437–442.
38. Liu S, Luo XH, Liu YF, et al. Emodin exhibits anti-acne potential by inhibiting cell growth, lipogenesis, and inflammation in human SZ95 sebocytes. *Sci Rep.* 2023;13(1):21576.