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Impact of air pollution on otolaryngological disease

Wpływ zanieczyszczenia powietrza na choroby z zakresu otolaryngologii

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Abstract

Introduction and Objective. Air pollution is a major environmental health problem associated with increased respiratory morbidity and mortality. Pollutants such as particulate matter (PM2,5, PM10), nitrogen dioxide, sulpher dioxide, ozone, and carbon monoxide may damage the respiratory tract through oxidative stress, epithelial dysfunction, and immune dysregulation. Because the upper airways are the first site of contact with inhaled pollutants, otolaryngological diseases are particularly susceptible to environmental exposure. The aim of this review is to summarize current evidence regarding the impact of air pollution on ENT diseases and their underlying mechanisms.

Brief description of the state of knowledge. Current studies demonstrate significant associations between air pollution and disorders such as rhinitis, chronic rhinosinusitis, otitis media, and laryngeal diseases. Both short- and long-term exposure to pollutants increase disease incidence, symptom severity, and healthcare utilisation. Oxidative stress, chronic inflammation, epithelial barrier dysfunction, and impaired mucociliary clearance appear to play central roles in disease pathogenesis. Children and the elderly are especially vulnerable to pollution-related upper airway diseases.

Summary. Air pollution is an important environmental determinant of otolaryngological diseases. Its effects involve inflammatory, oxidative, and structural mechanisms contributing to both acute and chronic upper airway pathology. Recognition of environmental exposure, implementation of preventive strategies, and further research on disease mechanisms may improve prevention and clinical management of ENT disorders.

Key words

air pollution, otolaryngological diseases, oxidative stress

Streszczenie

Wprowadzenie i cel pracy. Zanieczyszczenie powietrza stanowi istotny problem zdrowotny prowadzący do zwiększonej zachorowalności i śmiertelności z powodu chorób układu oddechowego. Zanieczyszczenia, takie jak pyły zawieszone (PM2,5, PM10), dwutlenek azotu, dwutlenek siarki, ozon oraz tlenek węgla, mogą uszkadzać drogi oddechowe poprzez stres oksydacyjny, dysfunkcję bariery nabłonkowej oraz zaburzenia odpowiedzi immunologicznej. Górne drogi oddechowe jako pierwsze mają kontakt z wdychanymi zanieczyszczeniami i są szczególnie narażone na ich szkodliwe działanie. Celem pracy było podsumowanie aktualnej wiedzy dotyczącej wpływu zanieczyszczenia powietrza na choroby laryngologiczne oraz omówienie leżących u ich podstaw mechanizmów.

Opis stanu wiedzy. Aktualne badania wykazują istotny związek pomiędzy ekspozycją na zanieczyszczenia powietrza a występowaniem chorób takich jak nieżyt nosa, przewlekłe zapalenie zatok przynosowych, zapalenie ucha środkowego oraz choroby krtani. Zarówno krótko-, jak i długoterminowa ekspozycja na zanieczyszczenia zwiększa częstość występowania chorób, nasilenie objawów oraz liczbę kontaktów z ochroną zdrowia. Kluczową rolę w patogenezie odgrywają stres oksydacyjny, przewlekły stan zapalny, uszkodzenie bariery nabłonkowej oraz zaburzenia klirensu śluzowo-rzęskowego. Szczególnie narażone na negatywny wpływ zanieczyszczeń są dzieci oraz osoby starsze.

Podsumowanie. Zanieczyszczenie powietrza stanowi istotny środowiskowy czynnik ryzyka chorób laryngologicznych. Jego działanie obejmuje różne mechanizmy wspólnie przyczyniające się do rozwoju zarówno ostrych, jak i przewlekłych chorób górnych dróg oddechowych. Uwzględnianie ekspozycji środowiskowej, wdrażanie działań profilaktycznych oraz dalsze badania nad mechanizmami chorób mogą przyczynić się do poprawy profilaktyki i leczenia schorzeń laryngologicznych.

Słowa kluczowe

zanieczyszczenie powietrza, choroby laryngologiczne, stres oksydacyjny

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INTRODUCTION

Air pollution has emerged as one of the most significant environmental threats to human health worldwide. It is responsible for approximately 6.7 millions of deaths annually and represents a major contributor to global morbidity [1]. Outdoor as well as indoor air pollution are important sources of exposure. Nowadays, a substantial proportion of time is spend in enclosed environments making indoor exposure highly relevant as the pollutant concentrations may be equally or even more harmful than those outdoors [1]. Air pollutants such as particulate matter (PM2.5 and PM10), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO) create a complex mixture of chemical and biological components that can adversely affect the respiratory system [2]. Due to their small size, fine particles are capable of penetrating deep into the airways and even entering the systemic circulation, where they contribute to both local and systemic inflammatory responses [3]. Oxidative stress, epithelial damage, and immune dysregulation are the main pathways responsible for collectively impair the natural defence mechanisms of the respiratory tract [2]. The upper respiratory tract, including the nasal cavity, paranasal sinuses, pharynx, and larynx, represents the first line of contact with inhaled environmental pollutants. Consequently, it is particularly subjected to their damaging effects.

Exposure to air pollution is suggested to contribute to a wide range of otolaryngological conditions, including allergic and non-allergic rhinitis, chronic rhinosinusitis, otitis media, and upper respiratory tract infections [4]. Epidemiological studies have demonstrated that both short-term and long-term exposure to air pollutants increase the incidence, severity, and recurrence of these conditions, contributing significantly to healthcare burden and reduced quality of life [5]. Moreover, recent studies have also shown relationship between specific pollutants and upper airway diseases. For example, particulate matter and nitrogen dioxide have been linked to an increased risk of pharyngitis and chronic rhinitis, highlighting the direct impact of environmental exposure on disease development [6]. Furthermore, children, as a particularly vulnerable population, appear to be overly affected due to their developing respiratory systems and higher relative exposure [2].

Despite the research, the relationship between air pollution and otolaryngological diseases still remains only partially understood. Therefore, a comprehensive review and further studies are crucial to better understand the underlying mechanisms and clinical implications. **The aim of this review is to summarize the current knowledge on the impact of air pollution on otolaryngological diseases.**

MATERIALS AND METHOD

A literature review was conducted in accordance with selected PRISMA principles. Primarily the PubMed database was searched for studies published especially within the last 10 years using key words, including 'air pollution', 'PM2.5', 'PM10', 'rhinitis', 'chronic rhinosinusitis', 'otitis media', 'laryngeal disorders', and 'oxidative stress'.

Original studies, systematic reviews, meta-analyses, and clinically relevant experimental studies evaluating the relationship between air pollution and otolaryngological diseases were included. Preference was given to recent studies with high methodological quality and direct relevance to ENT pathology. Duplicate publications, conference abstracts without full-text availability, and studies unrelated to upper airway diseases were excluded.

Air pollution. Air pollution is a complex mixture originating from various sources, including natural sources, consisting of both solid and gaseous components. The most relevant pollutants include particulate matter (PM2.5 and PM10), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), carbon monoxide (CO), and ozone (O_3), which are primarily generated by combustion of fossil fuels, industrial activity, traffic emissions, and domestic heating [7, 8]. Due to their small size, small molecules can deposit within the upper airways and penetrate deeper into the respiratory tract, while gaseous pollutants directly interact with the airway epithelium [9]. These exposures affects the upper respiratory tract by increasing the number of incidences of infections and inflammatory conditions. Even a short-term and medium-term exposure to air pollutants is linked to a higher frequency of symptoms such as rhinitis, cough, and pharyngitis in both children and adults [10, 8]. Pollutants induce oxidative stress, epithelial damage, and immune dysregulation, which impair mucociliary clearance and increase susceptibility to pathogens [9]. In addiction, air pollution may modify the respiratory microbiome, contributing to chronic inflammation and disease progression. Overall, these findings indicate that environmental exposure to air pollutants plays a significant role in the development and aggravation of upper airway diseases.

Role of oxidative stress in different laryngological diseases. Oxidative stress is one of the main mechanism in the pathogenesis of a wide range of otorhinolaryngological diseases, linking environmental exposure, inflammation, and tissue damage. It is characterised by an imbalance between excessive production of reactive oxygen species (ROS) and insufficient antioxidant defence, leading to cellular injury affecting DNA, as well as proteins, and lipids [11]. In the upper airways, oxidative stress disrupts epithelial barrier integrity, impairs mucociliary clearance, and modifies natural immune responses, thus increasing susceptibility to infections and chronic inflammatory conditions such as chronic rhinosinusitis and nasal polyposis [12, 13].

At the molecular level, ROS activate key inflammatory signalling pathways, including NF-κB, and induce endoplasmic reticulum stress responses, further amplifying cytokine production and sustaining chronic inflammation [13]. Moreover, oxidative stress contributes to structural and functional damage in the cochlea and vocal folds, where it promotes cellular degeneration, fibrosis, and impaired tissue regeneration [14, 15]. Simultaneous impact of oxidative stress, hypoxia, and inflammation creates a cycle that aggravates disease progression and tissue remodelling [16]. All things considered, these findings highlight oxidative stress as a key unifying mechanism across otorhinolaryngological disorders.

Focusing particularly on otolaryngological disorders, it is observed that air pollution plays a significant role in their development and intensity. To begin with, air pollution is increasingly recognized as a significant environmental risk

factor of rhinitis. Various studies demonstrate a consistent association between exposure to both particulate matter ($PM_{2.5}$, PM_{10}) and gaseous pollutants, such as nitrogen dioxide (NO_2), sulpher dioxide (SO_2), and carbon monoxide (CO), and the prevalence and severity of rhinitis symptoms [4, 17]. Epidemiological evidence indicates that higher concentrations of these pollutants correlate with increased outpatient visits and symptoms severity, suggesting a dose–response relationship [18].

Air pollutants act as irritants and immunological adjuvants, enhancing epithelial permeability, promoting allergen penetration, and local inflammatory responses within the nasal mucosa [19]. As previously mentioned, this process is largely mediated by oxidative stress and the overproduction of reactive oxygen species [2]. Vulnerable populations, particularly children and individuals exposed during early life, appear to be at higher risk due to developing respiratory and immune systems [20]. Overall, current evidence strongly supports a multifactorial role of air pollution in rhinitis pathogenesis, involving inflammatory, oxidative, and immune-mediated pathways.

Studies demonstrate that exposure to pollutants, particularly particulate matter ($PM_{2.5}$, PM_{10}) and ozone, is associated with increased prevalence and symptom severity in patients suffering from chronic rhinosinusitis (CRS) and related upper airway diseases [21, 22]. Both epidemiological and clinical data suggest that even short exposures exacerbate sinonasal symptoms, depending on individual factors such as gender, comorbid asthma, and seasonal variation [22]. Once again, oxidative stress, epithelial barrier dysfunction, and impairment of mucociliary clearance play curial role in persistent mucosal inflammation [23]. These processes are further mediated by activation of immune pathways and increased production of pro-inflammatory cytokines [24]. Studies also indicate that particulate matter can directly damage sinonasal epithelial cells and alter tissue structure, supporting its role in disease progression [21]. Additionally, long-term exposure to fine particulate matter has been implicated in the development of CRS, supporting the thesis of a unified airway response to environmental irritants [25]. Despite growing evidence, differences in exposure assessment methods limits comparability across studies, insisting on standardised research approaches for better future analysis [23].

Moving on to otitis media, air pollution has been consistently identified as an important risk factor, particularly in paediatric populations. Large population-based studies demonstrate that exposure to pollutants such as particulate matter (PM2.5, PM10) and oxidant gases is associated with increased incidence of acute otitis media (AOM), with effects further amplified by concurrent viral infections, such as influenza [26]. Epidemiological analyses show that short-term increases in pollutants like CO and NO_2 is linked to higher rates of emergency department visits within days of exposure [27]. Similarly, nationwide cohort data indicate that particulate matter has a particularly strong and immediate impact on AOM incidence, especially in children under two years of age [28]. Long-term exposure to traffic-related pollutants, including NO_2, PM2.5, and elemental carbon, has also been shown to increase the probability of developing otitis media in early childhood [29]. Additionally, vulnerable groups such as infants – particularly those born preterm – show increased susceptibility to pollution-related respiratory conditions, including otitis media [30]. These findings indicate that air pollution plays a multifactorial role in otitis media pathogenesis, acting through both acute and chronic mechanisms.

Air pollutants also contribute to both malignant and non-malignant laryngeal disorders. Chronic exposure to particularly particulate matter and toxic compounds produced during combustion of solid fuels, has been associated with an increased risk of respiratory tract cancers [31, 32]. While tobacco and alcohol remain the primary risk factors, environmental exposures such as air pollution, asbestos, and chemical irritants further contribute to carcinogenesis, especially in populations with prolonged indoor exposure [33]. Further data indicate that in low- and middle-income regions where exposure to household air pollution is highest are extremely influenced [34]. In addition to its role in cancer development, air pollution also affects laryngeal function through direct irritation and damage to the vocal fold epithelium. The vocal fold epithelial barrier serves as the first line of defense against environmental insults, and its disruption by pollutants leads to increased permeability, inflammation, and susceptibility to injury [35]. These processes may result in voice disorders such as dysphonia, as well as chronic laryngeal inflammation. To sum up, air pollution contributes to laryngeal pathology through both long-term carcinogenic mechanisms and short-term epithelial injury.

Clinical implications. Air pollution has significant clinical implications for the diagnosis, management, and prevention of otolaryngological diseases, emphasizing the need to integrate environmental factors into routine clinical practice. Given the recognized associations between exposure to pollutants – particularly particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulpher dioxide (SO_2), and carbon monoxide (CO) – and conditions such as rhinitis, chronic rhinosinusitis, otitis media, and laryngeal disorders, clinicians should systematically assess environmental exposure during patient evaluation [17, 36]. A detailed environmental history, including residential location, occupational exposure, use of solid fuels, and time spent in high-pollution environments, is particularly important, as both short- and long-term exposure have been shown to increase disease incidence, severity, and contact with healthcare [10, 27]. This is especially relevant in vulnerable populations such as children and the elderly, who demonstrate increased susceptibility to pollution-related upper airway diseases [7, 30].

From a preventive perspective, reducing exposure to air pollutants may be a key strategy in reducing disease severity. Evidence indicates that both outdoor and indoor air pollution contribute to otolaryngological pathologies, with household air pollution from solid fuel combustion being a major risk factor in low- and middle-income districts [31]. Therefore patients should be advised on practical exposure-reduction strategies, including the use of air purifiers, avoidance of outdoor activity during periods of high pollution, smoking cessation, and improvement of indoor ventilation. Public health interventions, such as cleaner energy policies and pollution control measures, are also essential, as population-level reductions in pollutant exposure have been associated with decreased incidence of respiratory and otolaryngological diseases [37].

The role of the physician extends beyond individual patient care and should include education, early identification

of at-risk individuals, and support for environmental health. Studies have shown that air pollution induces various mechanisms, as previously mentioned, including oxidative stress, epithelial barrier dysfunction, and immune dysregulation, which contribute to both acute infections and chronic inflammatory conditions of the upper airways [9, 11]. Growing knowledge can lead to more targeted therapeutic approaches, including anti-inflammatory and supportive treatments. Furthermore, exposure may act synergistically with other risk factors, such as infections or smoking, amplifying disease severity and complicating clinical management [26, 38].

In conclusion, incorporating environmental exposure assessment into clinical practice, as well as promoting preventive strategies and engaging in public health initiatives, are essential components of modern otolaryngological care. Recognising air pollution may improves individual patient outcomes as well as contributing to reducing the overall load of diseases at the population level.

Further research. Further research on the impact of air pollution on otolaryngological diseases should focus on addressing current gaps in evidence, particularly regarding determinants, exposure assessment, and long-term clinical outcomes. Although numerous epidemiological studies demonstrate associations between pollutants and various disorders, heterogeneity in study design and variability in exposure measurement limit comparability and interpretation of results [39]. Future studies should therefore focus on standardising the methodologies, and may include the use of personal monitoring devices, geospatial modelling, and integration of environmental data with electronic health records. Additionally, more prospective cohort studies are crucial for a better understanding of correlations and to evaluate the long-term effects of chronic exposure, especially in vulnerable populations, such as children and the elderly, who exhibit increased susceptibility to pollution-related diseases [20].

Another essential path of research should target the underlying biological mechanisms. While current evidence highlights the role of oxidative stress, epithelial barrier dysfunction, and immune dysregulation in disease pathogenesis, more detailed studies are required to identify specific biomarkers and therapeutic targets [11]. In particular, emerging areas such as the interaction between air pollution and the respiratory microbiome warrant deeper exploration, as they may provide new insights into disease development and progression [40].

From a clinical and public health perspective, future expertise should also evaluate the effectiveness of preventive and interventional strategies, including air quality improvement policies, indoor air filtration, and behavioural modifications. Evidence suggests that reducing exposure to environmental pollutants can decrease the severity of respiratory diseases, but more high-quality studies are needed to quantify these benefits specifically for otolaryngological conditions [37].

Finally, interdisciplinary collaboration is critical for developing comprehensive strategies aimed at reducing exposure and improving patient outcomes. Overall, advancing research in this field will not only enhance understanding of the disease mechanisms, but also support the development of targeted interventions and evidence-based guidelines for clinical practice.

DISCUSSION

The present review highlights air pollution as a significant environmental factor contributing to the pathogenesis and progression of multiple otolaryngological diseases. Current evidence consistently demonstrates associations between exposure to particulate matter ($PM_{2.5}$, PM_{10}), nitrogen dioxide (NO_2), sulphur dioxide (SO_2), ozone (O_3), and carbon monoxide (CO) and increased incidence, severity, and recurrence of upper airway disorders, such as rhinitis, chronic rhinosinusitis (CRS), otitis media, and laryngeal diseases [17, 21, 41]. Although many studies are observational, the reproducibility of findings across different populations and study designs strongly supports the clinical relevance of environmental pollution in ENT pathology.

Among upper airway disorders, allergic rhinitis and CRS appear to be the conditions most consistently associated with chronic air pollution exposure. Epidemiological studies demonstrate that higher levels of particulate matter and traffic-related pollutants correlate with increased prevalence of rhinitis symptoms, greater disease severity, and more frequent visits to healthcare facilities [17, 18]. Similarly, patients with CRS exposed to elevated concentrations of pollutants show more severe symptoms and poorer disease control [22]. Importantly, several studies identified dose–response relationships, suggesting that increasing pollutant exposure directly contributes to worsening upper airway inflammation, rather than acting solely as a trigger in predisposed individuals [17].

The underlying mechanisms appear to be multifactorial, with oxidative stress playing a key role. Exposure to pollutants promotes excessive production of reactive oxygen species (ROS), resulting in epithelial injury, impaired mucociliary clearance, and activation of pro-inflammatory pathways [11, 13]. Chronic oxidative stress further disrupts epithelial barrier integrity, facilitating allergen penetration and increasing susceptibility to infections and chronic inflammation [9]. Several studies also demonstrated altered expression of inflammatory and immune-related genes in CRS and nasal polyposis, supporting the concept that pollution-induced epithelial dysfunction contributes directly to disease pathogenesis [12]. Emerging evidence additionally suggests that air pollution may alter the respiratory microbiome, creating dysbiosis that further amplifies inflammatory responses and disease progression [40].

Air pollution also plays an important role in paediatric ENT diseases, particularly otitis media. Both short-term and long-term exposure to particulate matter and traffic-related pollutants were associated with increased incidence of acute otitis media and higher rates of emergency department visits among children [27, 28]. Infants and young children appear especially vulnerable due to immature immune and respiratory defence mechanisms [30]. These findings are clinically important given the high prevalence of otitis media and its impact on hearing, speech development, and healthcare burden.

In addition to inflammatory diseases, evidence suggests that air pollution contributes to laryngeal pathology through both chronic irritation and carcinogenic mechanisms. Pollutants can disrupt the vocal fold epithelial barrier, increasing susceptibility to inflammation, vocal dysfunction, and tissue injury [35]. Long-term exposure to household air pollution and combustion-derived toxic compounds has

also been associated with increased risk of respiratory tract cancers, including laryngeal cancer [31, 38]. While tobacco and alcohol remain the dominant risk factors, environmental pollution may act synergistically with these exposures and contribute to cumulative carcinogenic effects.

The clinical implications of these findings are substantial. Environmental exposure should be considered during routine otolaryngological assessment, particularly in patients with chronic or recurrent upper airway symptoms. Detailed environmental history taking, including residential conditions, occupational exposure, smoking status, and indoor air quality, may improve identification of modifiable risk factors [37]. Preventive strategies, such as reducing exposure to traffic-related pollution, improving ventilation, smoking cessation, and use of cleaner household fuels, may contribute to lowering the disease burden at both the individual and population levels.

Despite growing evidence, several limitations remain. Due to the large number of available publications, this review primarily focused on studies with high methodological quality and direct clinical relevance to otolaryngological diseases. However, many studies rely on ecological or retrospective designs and differ considerably in exposure assessment methods, making direct comparison difficult [39]. Furthermore, confounding factors, such as smoking, socio-economic status, climate, and occupational exposure, are not always fully controlled. Additional prospective studies using standardized exposure measurements and molecular biomarkers are needed to better define causal relationships and identify patients at greatest risk.

Overall, current literature strongly supports the role of air pollution as a major environmental determinant of otolaryngological diseases. Its effects involve inflammatory, oxidative, immunological, and structural mechanisms that contribute to both acute and chronic upper airway pathology. Improved understanding of these processes may support the development of more effective preventive strategies and targeted therapeutic approaches in ENT practice.

REFERENCES

1. Vilcins D, Christofferson RC, Yoon JH, et al. Updates in Air Pollution: Current Research and Future Challenges. Ann Glob Health. 2024;90(1):9. Epub 20240201. doi:10.5334/aogh.4363. PubMed PMID: 38312715; PubMed Central PMCID: PMC10836163
2. Dondi A, Carbone C, Manieri E, et al. Outdoor Air Pollution and Childhood Respiratory Disease: The Role of Oxidative Stress. Int J Mol Sci. 2023;24(5). Epub 20230222. doi:10.3390/ijms24054345. PubMed PMID: 36901776; PubMed Central PMCID: PMC10001616
3. Holgate S. Air pollution is a public health emergency. BMJ. 2022;378:o1664. Epub 20220711. doi:10.1136/bmj.o1664. PubMed PMID: 35817464
4. Zhang ZQ, Li JY, Guo Q, et al. Association between air pollution and allergic upper respiratory diseases: a meta-analysis. Eur Respir Rev. 2025;34(176). Epub 20250625. doi:10.1183/16000617.0266-2024. PubMed PMID: 40562438; PubMed Central PMCID: PMC12220745
5. Liu Y, Wang Y, Dong J, et al. Association between air pollution and emergency department visits for upper respiratory tract infection in Lanzhou, China. Environ Sci Pollut Res Int. 2022;29(19):28816–28. Epub 20220106. doi:10.1007/s11356-021-17932-2. PubMed PMID: 34989991
6. Chen F, Zhang J, Xie J, Fu X. Causal relationships between air pollutants and upper respiratory tract infections: A two-sample, Mendelian randomization study. J Chin Med Assoc. 2025;88(6):481–5. Epub 20250430. doi:10.1097/JCMA.0000000000001242. PubMed PMID: 40307969; PubMed Central PMCID: PMC12718758
7. Wawryk-Gawda E, Miga-Orczykowska N, Matusiak J, et al. Air pollution exposure and the burden of pediatric upper respiratory tract infections in emergency departments: a retrospective time-series study. Eur J Pediatr. 2025;184(12):753. Epub 20251113. doi:10.1007/s00431-025-06572-0. PubMed PMID: 41225162; PubMed Central PMCID: PMC12612014
8. Ratajczak A, Badyda A, Czechowski PO, et al. Air Pollution Increases the Incidence of Upper Respiratory Tract Symptoms among Polish Children. J Clin Med. 2021;10(10). Epub 20210516. doi:10.3390/jcm10102150. PubMed PMID: 34065636; PubMed Central PMCID: PMC8156299
9. Glencross DA, Ho TR, Camiña N, et al. Air pollution and its effects on the immune system. Free Radic Biol Med. 2020;151:56–68. Epub 20200130. doi:10.1016/j.freeradbiomed.2020.01.179. PubMed PMID: 32007522
10. Huang JY, Feng W, Sang GX, et al. Association between short-term exposure to ambient air pollutants and the risk of hospital visits for acute upper respiratory tract infections among adults: a time-series study in Ningbo, China. BMC Public Health. 2024;24(1):1555. Epub 20240610. doi:10.1186/s12889-024-19030-7. PubMed PMID: 38858655; PubMed Central PMCID: PMC11163729
11. Meng L, Liu S, Luo J, et al. Oxidative stress and reactive oxygen species in otorhinolaryngological diseases: insights from pathophysiology to targeted antioxidant therapies. Redox Rep. 2025;30(1):2458942. Epub 20250202. doi:10.1080/13510002.2025.2458942. PubMed PMID: 39894944; PubMed Central PMCID: PMC11792148.
12. Mihalj H, Butković J, Tokić S, et al. Expression of Oxidative Stress and Inflammation-Related Genes in Nasal Mucosa and Nasal Polyps from Patients with Chronic Rhinosinusitis. Int J Mol Sci. 2022;23(10). Epub 20220515. doi:10.3390/ijms23105521. PubMed PMID: 35628331; PubMed Central PMCID: PMC9145877
13. Jeanson L, Kelly M, Coste A, et al. Oxidative stress induces unfolding protein response and inflammation in nasal polyposis. Allergy. 2012;67(3):403–12. Epub 20111222. doi:10.1111/j.1398-9995.2011.02769.x. PubMed PMID: 22188019
14. Paciello F, Pisani A, Rolesi R, et al. Noise-Induced Cochlear Damage Involves PPAR Down-Regulation through the Interplay between Oxidative Stress and Inflammation. Antioxidants (Basel). 2021;10(8). Epub 20210726. doi:10.3390/antiox10081188. PubMed PMID: 34439436; PubMed Central PMCID: PMC8388985
15. Hirano S, Inufusa H, You F. The Effect of Oxidative Stress on the Human Voice. Int J Mol Sci. 2024;25(5). Epub 20240223. doi:10.3390/ijms25052604. PubMed PMID: 38473848; PubMed Central PMCID: PMC10932051
16. McGarry T, Biniecka M, Veale DJ, Fearon U. Hypoxia, oxidative stress and inflammation. Free Radic Biol Med. 2018;125:15–24. Epub 20180327. doi:10.1016/j.freeradbiomed.2018.03.042. PubMed PMID: 29601945
17. Burte E, Leynaert B, Marcon A, et al. Long-term air pollution exposure is associated with increased severity of rhinitis in 2 European cohorts. J Allergy Clin Immunol. 2020;145(3):834–42.e6. Epub 20200123. doi:10.1016/j.jaci.2019.11.040. PubMed PMID: 31983528
18. Salcan S, Salcan I. The correlation between air pollution and the allergic rhinitis incidence: Erzincan model. Eur Rev Med Pharmacol Sci. 2023;27(2 Suppl):51–6. doi:10.26355/eurrev_202303_31702. PubMed PMID: 36971221
19. Chen SW, Tsai SC, Chang KH, et al. Air Pollution-Associated Rhinitis: Exploring the Preventive Role of Nutritional Supplements Against Particulate Matter-Induced Inflammation. Nutrients. 2025;17(5). Epub 20250227. doi: 10.3390/nu17050829. PubMed PMID: 40077699; PubMed Central PMCID: PMC11902121
20. Liu L, Ma J, Peng S, Xie L. Prenatal and early-life exposure to traffic-related air pollution and allergic rhinitis in children: A systematic literature review. PLoS One. 2023;18(4):e0284625. Epub 20230420. doi: 10.1371/journal.pone.0284625. PubMed PMID: 37079576; PubMed Central PMCID: PMC10118164
21. Leland EM, Vohra V, Seal SM, et al. Environmental air pollution and chronic rhinosinusitis: A systematic review. Laryngoscope Investig Otolaryngol. 2022;7(2):349–60. Epub 20220311. doi:10.1002/lio2.774. PubMed PMID: 35434330; PubMed Central PMCID: PMC9008184
22. Peeters S, Wang C, Bijnens EM, et al. Association between outdoor air pollution and chronic rhinosinusitis patient reported outcomes. Environ Health. 2022;21(1):134. Epub 20221221. doi:10.1186/s12940-022-00948-7. PubMed PMID: 36544141; PubMed Central PMCID: PMC9769041
23. Leland EM, Zhang Z, Kelly KM, Ramanathan M. Role of Environmental Air Pollution in Chronic Rhinosinusitis. Curr Allergy Asthma Rep. 2021;21(8):42. Epub 20210909. doi:10.1007/s11882-021-01019-6. PubMed PMID: 34499234; PubMed Central PMCID: PMC8590820
24. Czerwaty K, Piszczatowska K, Brzost J, et al. Immunological Aspects of Chronic Rhinosinusitis. Diagnostics (Basel). 2022;12(10). Epub 20220929. doi:10.3390/diagnostics12102361. PubMed PMID: 36292050; PubMed Central PMCID: PMC9600442

25. Zhang Z, Kamil RJ, London NR, et al. Long-Term Exposure to Particulate Matter Air Pollution and Chronic Rhinosinusitis in Nonallergic Patients. Am J Respir Crit Care Med. 2021;204(7):859–62. doi:10.1164/rccm.202102-0368LE. PubMed PMID: 34181862; PubMed Central PMCID: PMC8528530
26. Li C, Jiang X, Wei Y, et al. Air pollutants, seasonal influenza, and acute otitis media in children: a population-based analysis using 22-year hospitalization data. BMC Public Health. 2024;24(1):1581. Epub 20240613. doi:10.1186/s12889-024-18962-4. PubMed PMID: 38867184; PubMed Central PMCID: PMC11170825
27. Zemek R, Szyszkowicz M, Rowe BH. Air pollution and emergency department visits for otitis media: a case-crossover study in Edmonton, Canada. Environ Health Perspect. 2010;118(11):1631–6. doi:10.1289/ehp.0901675. PubMed PMID: 20663739; PubMed Central PMCID: PMC2974705
28. Park M, Han J, Park J, et al. Particular matter influences the incidence of acute otitis media in children. Sci Rep. 2021;11(1):19730. Epub 20211005. doi:10.1038/s41598-021-99247–3. PubMed PMID: 34611241; PubMed Central PMCID: PMC8492675
29. Brauer M, Gehring U, Brunekreef B, et al. Traffic-related air pollution and otitis media. Environ Health Perspect. 2006;114(9):1414–8. doi:10.1289/ehp.9089. PubMed PMID: 16966098; PubMed Central PMCID: PMC1570088
30. Girguis MS, Strickland MJ, Hu X, et al. Exposure to acute air pollution and risk of bronchiolitis and otitis media for preterm and term infants. J Expo Sci Environ Epidemiol. 2018;28(4):348–57. Epub 20171221. doi:10.1038/s41370-017-0006-9. PubMed PMID: 29269754; PubMed Central PMCID: PMC6013343
31. He KJ, Xu Z, Xu J, Gong G. Global and national burden of tracheal, bronchus, and lung cancer attributabled to household air pollution from solid fuels in populations aged 55 and above: an integrated study of frontier and joinpoint regression analysis. BMC Public Health. 2025;25(1):1249. Epub 20250403. doi:10.1186/s12889-025-22466-0. PubMed PMID: 40181324; PubMed Central PMCID: PMC11966841
32. Lao S, Li J, Li W, et al. Global trends in the burden of tracheal, bronchus, and lung cancer attributable to household air pollution. J Thorac Dis. 2025;17(11):9943–57. Epub 20251126. doi:10.21037/jtd-2025-1309. PubMed PMID: 41376946; PubMed Central PMCID: PMC12688501
33. Steuer CE, El-Deiry M, Parks JR, et al. An update on larynx cancer. CA Cancer J Clin. 2017;67(1):31–50. Epub 20161129. doi:10.3322/caac.21386. PubMed PMID: 27898173
34. Liu X, Yang Q, Pan L, et al. Burden of respiratory tract cancers in China and its provinces, 1990–2021: a systematic analysis of the Global Burden of Disease Study 2021. Lancet Reg Health West Pac. 2025;55:101485. Epub 20250130. doi:10.1016/j.lanwpc.2025.101485. PubMed PMID: 39968450; PubMed Central PMCID: PMC11833622
35. Levendoski EE, Leydon C, Thibeault SL. Vocal fold epithelial barrier in health and injury: a research review. J Speech Lang Hear Res. 2014;57(5):1679–91. doi:10.1044/2014_JSLHR-S-13-0283. PubMed PMID: 24686981; PubMed Central PMCID: PMC4557797
36. Li J, Feng C, Ma X, Wu D. Impact of the urban atmospheric environment on otolaryngologic disease outpatient visits in Lanzhou, China. Sci Rep. 2025;15(1):21556. Epub 20250701. doi:10.1038/s41598-025-07352-4. PubMed PMID: 40595082; PubMed Central PMCID: PMC12216470
37. Eguiluz-Gracia I, Mathioudakis AG, Bartel S, et al. The need for clean air: The way air pollution and climate change affect allergic rhinitis and asthma. Allergy. 2020;75(9):2170–84. Epub 20200130. doi:10.1111/all.14177. PubMed PMID: 31916265
38. Ding N, Zhou N, Zhou M, Ren GM. Respiratory cancers and pollution. Eur Rev Med Pharmacol Sci. 2015;19(1):31–7. PubMed PMID: 25635972.
39. Massey CJ, Tullis B, Johnson C, et al. Characterizing air pollution exposure methodologies in rhinology: a scoping review. Int J Environ Health Res. 2025;35(11):3588–603. Epub 20250313. doi:10.1080/09603123.2025.2477585. PubMed PMID: 40079501; PubMed Central PMCID: PMC13019986
40. Xue Y, Chu J, Li Y, Kong X. The influence of air pollution on respiratory microbiome: A link to respiratory disease. Toxicol Lett. 2020;334:14–20. Epub 20200916. doi:10.1016/j.toxlet.2020.09.007. PubMed PMID: 32949622
41. Bowatte G, Tham R, Perret JL, et al. Air Pollution and Otitis Media in Children: A Systematic Review of Literature. Int J Environ Res Public Health. 2018;15(2). Epub 20180203. doi:10.3390/ijerph15020257. PubMed PMID: 29401661; PubMed Central PMCID: PMC5858326