

## Effect Of Air Pollution On Oral Cavity: A Review

Dr. Renuka Nagarale<sup>1</sup>, Dr. Neetu Kadu<sup>2</sup>, Aafiya Khan<sup>3</sup>, Aliya Saman Khan<sup>3</sup>,  
Zainab Barejiya<sup>3</sup>

<sup>1</sup>(Hod Of Public Health Dentistry, Mardc, India)

<sup>2</sup>(Assistant Professor Of Public Health Dentistry, Mardc, India)

<sup>3</sup>(Under Graduate Interns Of Public Health Dentistry, Mardc, India)

---

### Abstract:

**Background:** Air pollution is a global environmental issue with significant impacts on human health. While extensive research has been conducted on the systemic effects of air pollution, limited attention has been given to its influence on oral health. This literature review aims to bridge this gap by exploring the relationship between air pollution and various aspects of oral cavity, including dental caries, periodontitis, dental erosion, genotoxic effects, oral cancer, and oral clefts.

**Materials and Methods:** A literature search was conducted using different platforms to explore the relationship between air pollution and oral health. The search included randomized controlled trials, cohort studies, cross-sectional studies, and reviews and only 50 met the eligibility criteria and were included in this review article.

**Conclusion:** This comprehensive literature review highlights the versatile impact of air pollution on oral health, emphasizing the need for further research to understand the direct relationships and potential interference. Collaborative efforts are needed for improved clinical practices and public health outcomes in addressing the critical intersection of environmental and oral health concerns.

**Key Word:** air pollution, oral, pollution effects.

---

Date of Submission: 21-12-2023

Date of Acceptance: 31-12-2023

---

### I. Introduction

Air pollution is the introduction of chemical, physical, or biological agents into the indoor or outdoor environment, causing alterations to the inherent characteristics of the atmosphere. Common sources contributing to this phenomenon encompass household combustion devices, motor vehicles, industrial facilities, and other sources [1]. Typically, urban areas are more polluted than rural areas. [2] According to the World Health Organization (WHO), air pollution is the largest environmental risk factor for human diseases, contributing to around one in eight deaths worldwide.[1]

Pollutants of notable public health concern include particulate matter (PM), carbon monoxide, ozone, nitrogen dioxide, and sulfur dioxide generated due to chemical or photochemical reactions in the atmosphere. These constitute the major components of the Traffic-Related Air Pollution (TRAP) mixture. [3] Outdoor and indoor air pollution affect the respiratory system and other diseases impacting morbidity and mortality rates. From smog hanging over cities to smoke inside the home, air pollution is a major threat to health.[4]

Both short- and long-term exposure to air pollution can lead to various diseases, including stroke, chronic obstructive pulmonary disease, trachea, bronchus and lung cancers, asthma, and lower respiratory infections.[5] There is evidence of links between exposure to air pollution and type 2 diabetes, obesity, systemic inflammation, Alzheimer's disease, and dementia.[6],[7] The International Agency for Research on Cancer has classified air pollution, particularly PM2.5, as a leading cause of cancer.[8]

The oral cavity represents one of the main passages for the introduction of air pollutants in the human body before they arrive in the lungs and the GI tract, leading to the commencement of their systemic effects; hence, the direct contact of air pollutants with the periodontium takes place during their passage through the mouth.[9] Other areas where air pollutants affect the oral cavity are teeth (leading to dental caries)[10], oral mucosa (genotoxic effects & oral cancer)[11], and prenatal exposure of the mother leading to oral clefts in newborn.[12]

This topic is of major concern because many researchers have discussed the impact of air pollution on systemic health, but very little research has been done on its effect on oral health. Understanding its impact on oral health can contribute to the broader understanding of the health concerns associated with environmental pollution. Moreover, while researching the topic a certain gap in the scientific literature regarding this matter was observed, exploring this relationship can contribute to filling this knowledge gap. Research in this area also

combines various fields i.e., environmental science, public health, general health, and dentistry, making it an interdisciplinary field that can potentially draw attention and interest from a wide range of researchers and professionals. We also aim to prompt changes in clinical practices, oral hygiene recommendations, and perhaps even alteration of the approach of dental professionals in affected areas.

## **II. Material And Methods**

A comprehensive literature search was conducted from October to December 2023 to investigate the relationship between air pollution and the oral cavity. A literature search was performed on electronic databases, including PubMed, Schematic Scholar, EBSCO, and Google Scholar, using keywords such as "air pollution", "pollution effect", "oral cavity," and related terms. Only online articles in English were included. Out of 1500 articles found, randomized controlled trials, cohort studies, cross-sectional studies, and review articles were considered, investigating air pollution's impact on the oral cavity and case studies were excluded. Initial screening based on titles and abstracts was conducted to exclude irrelevant articles. Only 54 research papers were included in this review. Primary data sources included peer-reviewed journals, scientific databases, and references from relevant review articles, newspaper articles, and press releases. The collected data were then assembled to write this review article.

### **Dental caries and air pollution**

Dental caries represents a complex and multifactorial biofilm-mediated disease, involving the indigenous oral bacteria and the process of demineralization within the affected tooth. This process is characterized by an imbalance that leads to increased demineralization of the tooth structure (Weiner RC 2013). [13] Kabulbekov (1991) and Aa (1991) both identified a link between caries morbidity and environmental factors such as carbon oxide. They also noted the macro- and microelement composition of saliva and teeth tissue reduction in certain dental elements in children living in polluted areas. [10]

Dautov (2003) further supported this, finding a lower prevalence and severity of caries in children living in less polluted regions. The findings indicated that in ecologically tense and relatively pure regions, a nursery situated in a control region exhibited the lowest prevalence and severity of caries. [14] In a research study conducted by Abdazimov AD in 1992, white rats were exposed to industrial pollutants like aerosols and gases resulting in an increased incidence of caries by 30-40%, exacerbating the severity of the carious process. These effects coincide with disturbances in metabolic processes and disruptions to blood and salivary enzymatic systems. [15]

Jakhete (2012) expanded on these findings, highlighting the role of environmental factors such as passive smoke exposure in the development of dental caries in low-income children.[16] Since smoke containing nicotine can harm teeth and gingiva, making oral hygiene worse, researchers are now looking into how children exposed to secondhand smoke might be more likely to develop dental caries. (Munira 2021, Aligne 2003, Weiner 2013) [17],[18],[13]

Weiner RC 2013, [13] has explained that various theoretical frameworks substantiate the proposition that passive smoke may serve as a contributory factor in the etiology of caries, one of them is that the children exposed to secondhand smoke exhibited diminished levels of Vitamin C, [19] and lower Vitamin C levels are linked to weakened immune function and the proliferation of *Streptococcus Mutans*. Passive smoke exposure is correlated with decreased saliva production and diminished buffering capacity in affected children, potentially leading to reduced remineralization, compromised bacterial clearance, and heightened bacterial colonization [19]. Furthermore, heavy metals like cadmium present in secondhand smoke may constitute independent risk factors for dental caries. [19] A study published in 2003 found serum cotinine levels (by-product of nicotine) in children is associated with dental caries in deciduous teeth. [18] The same study concluded that a quarter of children would have never developed caries in their deciduous teeth had environmental tobacco smoke exposure been eliminated. [16] Researchers also observed an increased occurrence of dental caries among children residing in households with frequent exposure to passive smoke. (Shenkin JD, Broffitt B 2004) [20]

Exposure to passive smoke during pregnancy can impact fetal development, increasing the risk of low birth weight and prematurity, which are linked to enamel hypoplasia. Enamel hypoplasia involves underdeveloped tooth enamel, making the tooth surface more susceptible to cavity formation and early infection by *Streptococcus mutans*, a major contributor to dental caries. The thin enamel layer is more vulnerable to the acidic effects, and nicotine exposure may disrupt normal tooth development, increasing the risk of dental caries.[16] Studies, such as Tanaka et al., have demonstrated a correlation between maternal smoking, cumulative postnatal environmental tobacco smoke exposure, and an elevated prevalence of dental caries in children.[21]

Jakhete, 2012 also explains that exposure to environmental tobacco smoke elevates the possible risk of asthma, respiratory ailments, and middle ear infections. These conditions and their treatments may contribute to an increased risk of developing dental caries. [16] The author also quotes a study (Mehta A, 2009), conducted

with the objective of occurrence of dental caries in asthmatic patients, the results showed a significantly higher prevalence of caries in the study population. [22] In cases of rhinitis, children may breathe through an open mouth due to nasal congestion. This results in a dry mouth, increasing the risk of caries as saliva, which possesses antibacterial properties, is reduced. Moreover, saliva serves as a buffering agent against acid production, physically clears tooth surfaces of debris, and carries immunological and bacteriostatic properties. Due to a decreased salivary flow until their glands fully mature, children are more vulnerable to caries. [16]

### **Periodontitis and Air pollution**

Numerous studies have substantiated a positive correlation between air pollution and periodontitis- In a cross-sectional study conducted in South Korea (Marruganti 2023), periodontitis exhibited an independent association with slight elevations in outdoor levels of PM10, ozone, and SO<sub>2</sub>. Additionally, an inverse correlation was observed between the occurrence of periodontitis and levels of NO<sub>2</sub> and humidity. When specifically examining severe periodontitis, consistent findings were noted for PM10 and ozone levels, while the previously observed relationships with SO<sub>2</sub>, NO<sub>2</sub>, and humidity dissipated.[9]

The intricate interplay between air pollution and periodontitis unfolds through both direct and indirect mechanisms, shaping a complex nexus between environmental factors and oral health. Directly, air pollutants establish contact with the periodontium via the intake of contaminated food and water [9], instigating local effects characterized by heightened inflammation and oxidative stress, similar to pulmonary responses observed in the lungs. It is hypothesized that this "direct" local impact, marked by increased cytokines expression and elevated reactive oxygen species (ROS) concentration within the periodontium, could induce periodontal inflammation and contribute to the development of periodontitis [23]., yet a deeper understanding of the precise molecular pathways involved remains a critical avenue for future research

Simultaneously, indirect effects manifest at the systemic level, where chronic exposure to air pollutants induces low-grade inflammation, recognized as a central feature in various chronic diseases. This systemic inflammation, in turn, has been proposed as a unifying mechanism linking conditions such as obesity and diabetes with periodontitis.[24]The investigation into the influence of inflammatory markers on periodontitis remains unexplored.

In another retrospective cohort study (HJ Lin 2021) conducted in Taiwan establishes a compelling link between prolonged exposure to higher air pollutant levels and an elevated risk of periodontitis [25].This evidence underscores the need for further exploration into whether reduced exposure to air pollutants could mitigate the risk of periodontitis and the underlying mechanisms involved.

Furthermore, investigations made by JD Sutton 2017 into non-smokers reveal that exposure to environmental tobacco smoke (ETS) or secondhand smoke constitutes a significant risk factor for periodontitis over a lifetime [26]. Alveolar bone loss is a remarkable event in periodontitis, and ETS causes an increase in alveolar bone loss in the periodontal tissues of rat models (Li 2021) [27].

Beyond the local implications, the association between periodontitis and overall health unfolds in multidimensional ways. Respiratory diseases, particularly chronic obstructive pulmonary disease (COPD), exhibit epidemiological ties to poor oral hygiene and periodontal bone loss. The role of oral bacteria, such as *Porphyromonas gingivalis*, in respiratory infections is elucidated through mechanisms involving the aspiration of oral pathogens into the lungs and the modification of mucosal surfaces to facilitate the adhesion and colonization of respiratory pathogens. Additionally, periodontal disease-associated enzymes in saliva may play a crucial role in altering mucosal surfaces and impacting the clearance of pathogenic bacteria[28]. The overreaction of the inflammatory process, a commonality between periodontal disease and emphysema, sheds light on the association between periodontitis and COPD, emphasizing the need for improved oral hygiene in at-risk populations [29]. Contrasting this, an intriguing inverse association emerges between periodontitis and respiratory allergies like hay fever and HDM (house dust mite), aligning with the hygiene hypothesis. (Friedrich 2006)[30]

To summarize, the complicated links between air pollution and periodontitis necessitate a thorough understanding and strategic actions.

### **Dental erosion due to Air Pollution**

Research has shown that exposure to certain environmental factors, in the battery industry, can lead to erosion of teeth (Elsbury, 1951; Malcolm, 1961). [31][32] Dental erosion was seen in the oral cavity of girls working in an atmosphere containing dust composed of a mixture of tartaric acid, sucrose, magnesium sulphate, and sodium bicarbonate. The authors further describe that it may be because these chemicals settle on the teeth normally exposed by the lips forming a highly concentrated solution in the saliva locally and converting the insoluble calcium salts of the enamel and dentine into soluble calcium tartrate. [31] In another study (Malcolm D, 1961), the impact of sulphuric acid on the teeth of individuals working in the storage battery industry was explored. [32] This study revealed that only male workers exposed to acid mist exhibited erosion of the incisor

teeth however erosion did not increase further in extent after reaching the level of lip. The author also puts forth various factors influencing erosion which include exposure duration, lip level, and acid concentration in the air. [32] Similar observation was seen by other researchers in other studies. (Ten, H. J. and Bruggen Cate, 1968) [33]

Workers in industries other than battery or galvanizing may also face elevated risks of dental erosion. However, insufficient prevalence data from chemical and pharmaceutical workers, tin and munition manufacturers, and cleaners do not permit to draw conclusions. Additionally, the influence of potential confounders like upper respiratory tract medical issues must be considered.[34] To gain a more comprehensive understanding of the risk of occupational dental erosion, it is imperative to collect additional prevalence data from larger study populations.

### **Genotoxic Effects of Air Pollution on Oral Cavity**

An interesting concept that came forth while researching this topic was the relation of genotoxic effects on oral cavity due to air pollution. Cavalcante 2017 conducted a research in school children with an objective to determine meta-nuclear (MN) alterations in cells from the buccal (oral) mucosa of children associated with the school environment, gender, exposure to cigarette smoke and vehicular traffic. He found that children exposed to passive cigarette smoke exhibited higher levels of nuclear abnormalities in buccal cells, indicating DNA damage. Moreover, he also found that female children had higher amounts of nuclear abnormalities when compared to male children. [11] This may be attributed to alterations or spontaneous errors in DNA repair mechanisms, or it may be due to the influence of puberty, characterized by hormonal changes. Another possible reason could be related to the existence of two copies of the X chromosome in women, indicating an increased probability of loss of this chromosome as a MN, when compared to the remaining chromosomes. (Fenech and Bonassi, 2011) [35]

Tests that determine the meta-nuclear (MN) frequency and alterations in cells of buccal mucosa are important predictors of genetic damage caused by mutagenic and genotoxic agents. However, there are various other factors that cause increase of these frequencies such as lifestyle, environmental and occupational exposures, diet, medical procedures, diseases, gender, age, tobacco, and alcohol consumption as well and inherited genetic defects in DNA repair mechanisms (Suk et al., 2013, Nefic et al., 2013). [36,37] Nevertheless, other studies also reached on certain conclusions that they found no significant effects of any of such factors suggesting that that air pollution stands out as the major source of genotoxicity (Domingues 2018)[38]. It was also found that the frequency of MN in oral epithelial cells increases with age (Bonassi et al., 2011) [39]. Furthermore, genotoxic effects were also found in people with occupational exposure to wood dust (Celik et al., 2006) [40].

No evidence of lesions were seen in the oral cavity, while researching further into the topic due the increase in MN frequencies, however the effects of genotoxicity are observed on the overall health. The need to highlight this effect in this topic is important because the oral cavity serves as a site of such pathological findings that could potentially lead to long term systemic illness that can also pass on into generations. This also supports the argument of the statement mentioned earlier in this article that the oral cavity is the main passage for the introduction of air pollutants in the body. [9]

### **Oral cancer and air pollution**

Oral cancer poses a serious and escalating concern in various parts of the world. In 2012, the global incidence and deaths attributed to oral cancer were estimated at 300,000 and 145,000, respectively[41]. The risk of oral cancer due to exposure to air pollution is documented in a study, revealing that increased concentrations of fine particulate matter (PM<sub>2.5</sub>) in air pollution are associated with a 43% higher chance of developing oral cancer[42]. Another study focusing on Taiwanese men indicates that higher concentrations of PM<sub>2.5</sub> may be linked to an increased risk of oral cancer (YH Chu 2019)[43].

Living in areas exposed to air emissions from petroleum and chemicals is correlated with a higher likelihood of developing cancer in the buccal cavity and pharynx for both men and women (J Kaldor 1984)[44]. A hospital-based case-control study in Fujian, China, demonstrates that patients with a family history of cancer on the environmental exposure index are associated with an increased risk of oral cancer[45]. A quantitative review summarizing epidemiological findings notes that exposure to asbestos and polycyclic aromatic hydrocarbons is associated with oral and pharyngeal cancer risk. (F P Bailly) [46].

However, a review conducted by Raaschou-Nielsen in 2006 concluded that there is no increased risk of childhood cancer, including oral cancer, associated with exposure to traffic-related residential air pollution [47]. On contrary, in a meta-analysis by S. Josyula in 2015, Household air pollution (HAP) is associated with an increased risk for oral, nasopharyngeal, pharyngeal, and laryngeal cancers [48].

Despite these findings, researchers have also acknowledged that the connection between air pollutants and their potential contribution to oral cancer remains unclear. Additional research is needed to comprehensively understand the potential impact of air pollution on the risk of developing oral cancer.

### **Oral clefts and air pollution**

Maternal exposure to air pollution is associated with the development of congenital anomalies, such as cleft lip and cleft palate (CL/CP). Various studies have been conducted to explore this association - The Taiwan study indicated that exposure to outdoor air O<sub>3</sub> (ozone) during the first and second month of pregnancy may increase the risk of CL/P (Hwang and Jaakkola, 2008) [49]. Additionally, a meta-analysis reported that ozone exposure consistently showed the strongest association with an increased risk of orofacial cleft anomalies compared to other air pollutants (Ajit Rao et al., 2016). It is a concern that large numbers of pregnant women, globally encounter comparable levels of O<sub>3</sub> [50].

Ambient exposure to PM<sub>2.5</sub>, PM<sub>10</sub>, O<sub>3</sub>, and CO during the first trimester of pregnancy was associated with an increased risk of CLP/CPO, as found in a 2018 study conducted in Wuhan, China [51]. A systematic review and meta-analysis by Zhi Meng in 2023 revealed a significant statistical correlation between exposure to PM<sub>10</sub>, PM<sub>2.5</sub>, O<sub>3</sub>, and the risk of OFCs in the second month of pregnancy[52].

There are mixed findings regarding the relationship between air pollution and CL/CP. Studies from New Jersey found limited evidence of associations between CPO and O<sub>3</sub> exposure, as well as between CLP and SO<sub>2</sub> exposure (Marshall et al., 2010)[53].

Not only pollutants but also maternal exposure to dust and pesticides increases the risk of cleft lip and cleft palate (Spinder 2017) [54]. Further epidemiological investigations are required to validate these connections and to understand the reason of exposure to particular pollutants during the most vulnerable periods of pregnancy.

### **III. Conclusion:**

The review identified a gap in scientific literature regarding the direct relationship between air pollution and the oral cavity. A common notion that was observed that most of the authors, could not point out the direct link between both the variables as most of the effects are usually multifactorial in nature. Therefore more experimental studies with ample sample size is required for strong evidences. The oral cavity, as the main passage for the introduction of air pollutants, plays a crucial role in systemic health. Understanding these connections could contribute to changes in clinical practices, oral hygiene recommendations, and the approach of dental professionals, ultimately improving public health outcomes. The interdisciplinary nature of this research area invites collaboration across environmental science, public health, general health, and dentistry.

### **References**

- [1]. World Health Organization. Air Quality Guidelines: Global Update 2005: Particulate Matter, Ozone, Nitrogen Dioxide, And Sulfur Dioxide. World Health Organization; 2006.
- [2]. Boudreau D, Mcdaniel M, Sprout E, Turgeon A. Pollution. Education. National Geographic Society. 19 October 2023. Retrieved From: <https://Education.Nationalgeographic.Org/Resource/Pollution/>
- [3]. Guarnieri M, Balmes Jr. Outdoor Air Pollution And Asthma. *The Lancet*. 2014;383(9928):1581-1592.
- [4]. World Health Organization; Air Pollution; Health Topics; Retrieved From: [https://www.who.int/health-topics/air-pollution#tab=tab\\_3](https://www.who.int/health-topics/air-pollution#tab=tab_3)
- [5]. European Environment Agency. How Air Pollution Affects Our Health; Topics; 18 October 2023. Retrieved From: <https://www.eea.europa.eu/en/topics/in-depth/air-pollution/eow-it-affects-our-health#:~:text=Both%20short%2d%20and%20long%2dterm,Asthma%20and%20lower%20respiratory%20infections>
- [6]. Santos Nv, Yariwake Vy, Marques Kd, Veras Mm, Fajersztajn L. Air Pollution: A Neglected Risk Factor For Dementia In Latin America And The Caribbean. *Front. Neurol.* 2021;12:684524.
- [7]. Paul Kc, Jerrett M, Ritz B. Type 2 Diabetes Mellitus And Alzheimer's Disease: Overlapping Biologic Mechanisms And Environmental Risk Factors. *Current Environmental Health Reports*. 2018 Mar;5:44-58.
- [8]. International Agency For Research On Cancer (Iarc), Who; Outdoor Air Pollution A Leading Environmental Cause Of Cancer Deaths; Lyon/Geneva, 17 October 2013; Retrieved From: <https://www.iarc.who.int/news-events/iarc-outdoor-air-pollution-a-leading-environmental-cause-of-cancer-deaths/>
- [9]. Marruganti C, Shin Hs, Sim Sj, Grandini S, Laforí A, Romandini M. Air Pollution As A Risk Indicator For Periodontitis. *Biomedicine*. 2023;11(2):443.
- [10]. Kabulbekov Aa, Amrin Kr. Effects Of Air Pollution On The Etiology Of Dental Caries. *Gigiena I Sanitariia*. 1991 (4):6-8.
- [11]. Cavalcante Dn, Sposito Jc, Crispim Bd, Nascimento Av, Grisolia Ab. Genotoxic And Mutagenic Effects Of Passive Smoking And Urban Air Pollutants In Buccal Mucosa Cells Of Children Enrolled In Public School. *Toxicol. Mech. Methods*. 2017;27(5):346-51.
- [12]. Zhu Y, Zhang C, Liu D, Grantz KI, Wallace M, Mendola P. Maternal Ambient Air Pollution Exposure Preconception And During Early Gestation And Offspring Congenital Orofacial Defects. *Environ. Res*. 2015;140:714-20.
- [13]. Wiener Rc. Children With Special Health Care Need's Association Of Passive Tobacco Smoke Exposure And Dental Caries: 2007 National Survey Of Children's Health. *J. Abnorm. Child Psychol*. 2013;1.
- [14]. Dautov Ff, Lysenko Gn, Lysenko Ai. Impact Of Environmental Air Pollution On Dental Morbidity In Children. *Gigiena I Sanitariia*. 2003 (4):42-3.
- [15]. Abdazimov Ad. An Experimental Study Of The Action Of Industrial Aerosols And Toxic Gases On Dental Status. *Stomatologija*. 1992 (2):8-10.

- [16]. Jakhete N, Gitterman Ba. Environmental Smoke Exposure Associated With Increased Prevalence Of Dental Caries In Low-Income Children. *Int. J. Disabil. Hum. Dev.* 2012 ;11(4):315-20.
- [17]. Munira S, Islam Md, Sekander Md, Alam B. Passive Smoking And Pediatric Dental Caries. *Journal Of Preventive And Social Medicine.* 2021;39. 1-13.
- [18]. Aligne Ca, Moss Me, Auinger P, Weitzman M. Association Of Pediatric Dental Caries With Passive Smoking. *Jama.* 2003;289(10):1258-64.
- [19]. Hanioka T, Ojima M, Tanaka K, Yamamoto M. Does Secondhand Smoke Affect The Development Of Dental Caries In Children? A Systematic Review. *Int J Environ Res Public Health.* 2011 ;8(5):1503-19.
- [20]. Shenkin Jd, Broffitt B, Levy Sm, Warren Jj. The Association Between Environmental Tobacco Smoke And Primary Tooth Caries. *J. Public Health Dent.* 2004;64(3):184-6.
- [21]. Tanaka K, Miyake Y, Sasaki S. The Effect Of Maternal Smoking During Pregnancy And Postnatal Household Smoking On Dental Caries In Young Children. *J. Pediatr.* 2009;155(3):410-5.
- [22]. Mehta A, Sequeira Ps, Sahoo Rc. Bronchial Asthma And Dental Caries Risk: Results From A Case Control Study. *J Contemp Dent Pract.* 2009 ;10(4):59-66.
- [23]. Deo Vm, Bhongade Ml. Pathogenesis Of Periodontitis: Role Of Cytokines In Host Response. *Dentistry Today.* 2010 ;29(9):60-2.
- [24]. Pink C, Kocher T, Meisel P, Dörr M, Markus Mr, Jablonowski L, Grotevendt A, Nauck M, Holtfreter B. Longitudinal Effects Of Systemic Inflammation Markers On Periodontitis. *J Clin Periodontol .* 2015 ;42(11):988-97.
- [25]. Lin Hj, Tsai Sc, Lin Fc, Hsu Yc, Chen Sw, Chou Rh, Lin Cl, Hsu Cy, Chang Kh. Prolonged Exposure To Air Pollution Increases Periodontal Disease Risk: A Nationwide, Population-Based, Cohort Study. *Atmosphere.* 2021 ;12(12):1668.
- [26]. Sutton Jd, Salas Martinez Ml, Gerkovich Mm. Environmental Tobacco Smoke And Periodontitis In United States Non-Smokers, 2009 To 2012. *J. Periodontol.* 2017;88(6):565-74.
- [27]. Li X, Liang X, Li S, Qi X, Du N, Yang D. Effect Of Environmental Tobacco Smoke On Cox-2 And Shp-2 Expression In A Periodontitis Rat Model. *Oral Diseases.* 2021;(2):338-47.
- [28]. Scannapieco Fa. Role Of Oral Bacteria In Respiratory Infection. *J. Periodontol..* 1999 ;70(7):793-802.
- [29]. Mojon, Philippe. Oral Health And Respiratory Infection. *J Can Dent Assoc.* 2002;68(6):340-345.
- [30]. Friedrich N, Völzke H, Schwahn C, Kramer A, Jünger M, Schäfer T, John U, Kocher T. Inverse Association Between Periodontitis And Respiratory Allergies. *Clin Exp Allergy.* 2006;36(4):495-502.
- [31]. Elsbury Wb, Browne Rc, Boyes J. Erosion Of Teeth Due To Tartaric Acid Dust. *Br. J. Ind. Med.* 1951 ;8(3):179.
- [32]. Malcolm D, Paul E. Erosion Of The Teeth Due To Sulphuric Acid In The Battery Industry. *Occup. Environ. Med.* 1961;18(1):63-9.
- [33]. Ten Bruggen Cate Hj. Dental Erosion In Industry. *Br. J. Ind. Med.* 1968;25(4):249-66.
- [34]. Wiegand A, Attin T. Occupational Dental Erosion From Exposure To Acids—A Review. *Occupational Medicine.* 2007 May 1;57(3):169-76.
- [35]. Fenech M, Bonassi S. The Effect Of Age, Gender, Diet And Lifestyle On Dna Damage Measured Using Micronucleus Frequency In Human Peripheral Blood Lymphocytes. *Mutagenesis.* 2011 Jan 1;26(1):43-9.
- [36]. Suk Wa, Murray K, Avakian Md. Environmental Hazards To Children's Health In The Modern World. *Mutation Research/Reviews In Mutation Research.* 2003 Nov 1;544(2-3):235-42.
- [37]. Nefić H, Mušanović J, Kurteshi K, Prutina E, Turcalo E. The Effects Of Sex, Age And Cigarette Smoking On Micronucleus And Degenerative Nuclear Alteration Frequencies In Human Buccal Cells Of Healthy Bosnian Subjects. *J. Health Sci.* 2013 Dec;3(3):196-204.
- [38]. Domingues Ép, Silva Gg, Oliveira Ab, Mota Lm, Santos Vs, De Campos Eo, Pereira Bb. Genotoxic Effects Following Exposure To Air Pollution In Street Vendors From A High-Traffic Urban Area. *Environ. Monit. Assess.* 2018;190:1-6.
- [39]. Bonassi S, El-Zein R, Bolognesi C, Fenech M. Micronuclei Frequency In Peripheral Blood Lymphocytes And Cancer Risk: Evidence From Human Studies. *Mutagenesis.* 2011;26(1):93-100.
- [40]. Çelik A, Kanik A. Genotoxicity Of Occupational Exposure To Wood Dust: Micronucleus Frequency And Nuclear Changes In Exfoliated Buccal Mucosa Cells. *Environ. Mol. Mutagen.* 2006;47(9):693-8.
- [41]. Ferlay J, Soerjomataram I, Dikshit R, Eser S, Mathers C, Rebelo M, Parkin Dm, Forman D, Bray F. Cancer Incidence And Mortality Worldwide: Sources, Methods And Major Patterns In Globocan 2012. *Int. J. Cancer* 2015;136(5):359-86.
- [42]. Air Pollution Exposure May Be Linked To Higher Risk Of Mouth Cancer. *Br Dent J* 225, 800 2018 Nov 9. Accessed From: <https://doi.org/10.1038/Sj.Bdj.2018.994>
- [43]. Chu Yh, Kao Sw, Tantoh Dm, Ko Pc, Lan Sj, Liaw Yp. Association Between Fine Particulate Matter And Oral Cancer Among Taiwanese Men. *J. Investig. Med.* 2019 ;67(1):34-8.
- [44]. Kaldor J, Harris Ja, Glazer E, Glaser S, Neutra R, Mayberry R, Nelson V, Robinson L, Reed D. Statistical Association Between Cancer Incidence And Major-Cause Mortality, And Estimated Residential Exposure To Air Emissions From Petroleum And Chemical Plants. *Environ. Health Perspect.* 1984;54:319-32.
- [45]. Yan L, Chen F, He B, Liu F, Liu F, Huang J, Wu J, Lin L, Qiu Y, Cai L. A Novel Environmental Exposure Index And Its Interaction With Familial Susceptibility On Oral Cancer In Non-Smokers And Non-Drinkers: A Case-Control Study. *Eur. Arch. Oto-Rhino-L Eur Arch Oto-Rhino-L* 2017;274:1945-50.
- [46]. Paget-Bailly S, Cyr D, Luce D. Occupational Exposures To Asbestos, Polycyclic Aromatic Hydrocarbons And Solvents, And Cancers Of The Oral Cavity And Pharynx: A Quantitative Literature Review. *Int Arch Occup Environ Health.* 2012 May;85:341-51.
- [47]. Raaschou-Nielsen O, Reynolds P. Air Pollution And Childhood Cancer: A Review Of The Epidemiological Literature. *Int. J.* 2006 Jun 15;118(12):2920-9.
- [48]. Josyula S, Lin J, Xue X, Rothman N, Lan Q, Rohan Te, Hosgood Hd. Household Air Pollution And Cancers Other Than Lung: A Meta-Analysis. *Environmental Health.* 2015;14(1):1-1.
- [49]. Hwang Bf, Jaakkola Jj. Ozone And Other Air Pollutants And The Risk Of Oral Clefts. *Environ. Health Perspect.* 2008;116(10):1411-5.
- [50]. Rao A, Ahmed Mk, Taub Pj, Mamoun Js. The Correlation Between Maternal Exposure To Air Pollution And The Risk Of Orofacial Clefts In Infants: A Systematic Review And Meta-Analysis. *J. Oral Maxillofac. Surg.* 2016;7(1).