AMERICAN JOURNAL OF APPLIED MEDICAL SCIENCE
ISSN: 2996-5101 (online) | ResearchBib (IF) = 10.81 IMPACT FACTOR
Volume-4| Issue-3| 2026 Published: |30-03-2026

# IMPACT OF ATMOSPHERIC POLLUTION ON THE DENTOFACIAL SYSTEM IN TASHKENT RESIDENTS: FOCUS ON ORTHODONTIC IMPLICATIONS

https://doi.org/10.5371/zenodo.18890124

**Bakhtiyorova Nigorakhon Kahramon kizi**
*Master's Student, Tashkent State Medical University, Tashkent, Uzbekistan*
**Abdullakhonova Ominakhon Muhammadayubkhon kizi**
*Master's student, Tashkent State Medical University, Tashkent, Uzbekistan*
**Shaamuhamedova Feruza Abdulhakovna**
*Associate Professor, Department of Orthodontics and Prosthodontics, Tashkent State Medical University, Tashkent, Uzbekistan*

**Abstract**

**Background.** Tashkent, the capital of Uzbekistan, ranks among the world's most air-polluted megacities, with a 2024 annual average PM2.5 concentration of 31.5 μg/m³ — 6.3 times the WHO guideline of 5 μg/m³. The combined effect of windblown desert dust, coal combustion, and vehicle emissions generates a complex pollutant mixture that may substantially affect craniofacial and dental development in residents, particularly in children.

**Objective.** To systematically review current evidence on the mechanisms linking atmospheric pollution — with emphasis on PM2.5, PM10, $NO_2$, and heavy metals (Pb, Cd) — to disorders of the dentofacial complex, with particular focus on orthodontic implications for Tashkent's population.

**Methods.** A narrative review was conducted using PubMed, Scopus, Web of Science, and Google Scholar databases (2005–2025). Search terms included: "air pollution", "particulate matter", "dental caries", "periodontal disease", "malocclusion", "orthodontics", "craniofacial development", "heavy metals", "lead", "cadmium", "bone mineral density". Environmental data for Tashkent were retrieved from open sources: IQAir, World Bank (2024), and Uzhydromet.

**Results.** Chronic exposure to PM2.5 and PM10 promotes oxidative stress–mediated NF-κB activation, elevating IL-1β and TNF-α and stimulating osteoclastogenesis. Lead and cadmium adsorbed on PM particles impair enamel mineralisation and alveolar bone density. $NO_2$ inhibits osteoblastic activity and reduces bone mineral content, particularly during childhood growth windows

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(ages 1–4 years, per Generation R cohort data). Chronic nasal obstruction secondary to pollution-driven rhinitis shifts children to oral breathing, driving well-documented orthodontic sequelae: narrowed maxillary arch, retrognathia, increased vertical facial growth, and Class II/III malocclusion. Tashkent's seasonal PM peaks (November: up to 75.5 μg/m³ PM2.5) and dominant pollutant sources (36% windblown dust, 28% heating combustion) create a year-round exposure pattern that may amplify orthodontic treatment complexity and relapse risk.

**Conclusions.** Tashkent's air pollution profile constitutes a clinically significant environmental risk factor for orthodontic pathology. Integration of ecological indicators into orthodontic treatment planning and long-term retention protocols, as well as targeted epidemiological studies, are urgently needed.

**Keywords**

air pollution; PM2.5; particulate matter; malocclusion; orthodontics; craniofacial development; heavy metals; bone mineral density; Tashkent; Uzbekistan.

## Introduction

Atmospheric air pollution is one of the leading modifiable risk factors for the global burden of disease. According to 2024 estimates, more than 4.1 million people die annually from the consequences of fine particulate matter (PM2.5) exposure, and associated disability-adjusted life years (DALYs) exceed 118 million [1]. Central Asia, and Uzbekistan in particular, ranks among the regions with the highest exposure levels: according to IQAir data, Tashkent placed third among the world's most polluted major cities in 2024, with an annual mean PM2.5 concentration of 31.5 μg/m³ — 6.3 times the guideline of the World Health Organization (WHO) [2, 3].

Traditionally, environmental medicine research has focused on the respiratory, cardiovascular, and neurological effects of pollutant exposure. The dental — and in particular orthodontic — consequences of prolonged air pollution remain substantially less studied, despite the physiological plausibility of biological mechanisms linking atmospheric pollutants to the condition of the dentofacial system [4, 5]. The dentofacial complex is positioned at the intersection of the respiratory and digestive tracts, placing it in direct contact with inhaled aerosol particles, heavy metals, and toxic gases [6].

Three categories of consequences are of particular concern in the context of orthodontics: (1) direct impairment of dental hard tissue mineralisation and jaw bone growth by PM-associated heavy metals; (2) suppression of osteoblastic activity by $NO_2$ and PM2.5, affecting facial skeletal growth rates and the stability of

orthodontic treatment outcomes; and (3) chronic nasal obstruction secondary to pollutant-driven allergic sensitisation, which acts as a trigger for mouth breathing and the subsequent development of dentofacial anomalies.

Tashkent represents a unique ecological model: a high level of urbanisation, a specific pollution source structure dominated by windblown desert dust (including salt aerosols from the desiccated Aral Sea), seasonal peaks from coal heating, and a large ageing vehicle fleet together create year-round exposure to a diverse mixture of pollutants whose effects on the dentofacial system have not yet been systematically investigated.

**The aim of the present work** is to synthesise current evidence on the mechanisms by which atmospheric pollutants affect the dentofacial system, with emphasis on orthodontic implications, and to apply these findings to the ecological profile of Tashkent.

**Review Methodology**

This work was conducted as a narrative literature review. Searches were performed in PubMed/MEDLINE, Scopus, Web of Science, and Google Scholar covering publications from 2005 to 2025. The following search terms and their combinations were applied (in English and Russian): "air pollution", "particulate matter", "PM2.5", "PM10", "$NO_2$", "heavy metals", "lead", "cadmium", "malocclusion", "orthodontics", "craniofacial development", "bone mineral density", "dental caries", "periodontal disease", "oral breathing", "mouth breathing", "nasal obstruction", "enamel hypoplasia".

Inclusion criteria: original studies (randomised controlled trials, cohort, cross-sectional, and experimental studies), systematic reviews, and meta-analyses examining direct or indirect effects of atmospheric pollutants on the dentofacial system, facial skeletal bones, or orthodontic outcomes. Exclusion criteria: studies restricted to occupational or industrial exposure without reference to ambient air; animal data without human extrapolation; works with unverifiable methodology.

Environmental data for Tashkent were obtained from open sources: the IQAir air quality monitoring platform (historical data 2019–2025), the World Bank (Report on Air Quality Management Improvement in Uzbekistan, 2024), the State Committee of the Republic of Uzbekistan for Ecology and Environmental Protection (Uzhydromet), and several analytical publications based on Uzhydromet data. The final reference list comprises 25 sources cited directly in the text.

**Results**

***Ecological Profile of Tashkent: Air Pollution Data***

Tashkent is the largest city in Central Asia, with a population exceeding 2.7 million. Located in a semi-arid zone, the city's pollutant source structure differs

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Volume-4| Issue-3| 2026 Published: |30-03-2026

fundamentally from that of Western European or East Asian megacities [7]. According to the World Bank (2024), the main anthropogenic and natural sources of PM2.5 in Tashkent are: windblown dust (≈36%), including transboundary transport of salt aerosols from the desiccated bed of the Aral Sea; coal- and heavy fuel oil-fired heating systems (≈28%); road transport (≈16%); and industrial enterprises (≈13%) [8].

The seasonal pollution pattern is characterised by two pronounced peaks. The summer–autumn period (June–November) is associated with maximum dusty-particle concentrations: IQAir data show that monthly mean PM2.5 reached 75.5 μg/m³ in November 2019 [9]. The winter period (December–February) is dominated by combustion products, with elevated $SO_2$, CO, and polycyclic aromatic hydrocarbons (PAHs), which are of particular concern for residential neighbourhoods not connected to district heating systems [8].

Annual AQI data for 2020–2025 show a gradual improvement: AQI declined from 99 (2020) to 77 (2025), though it remains in the "Moderate" category according to the US EPA scale, and the annual mean PM2.5 (31.5 μg/m³ in 2024) still far exceeds the WHO standard [2, 10]. In December 2024, the Ministry of Health of Uzbekistan adopted a new PM2.5 air quality standard aligned with WHO recommendations, marking an important first step toward systematic air quality management policy in the country [8].

***Table 1. Major atmospheric pollutants in Tashkent and their dental and orthodontic significance***

| Pollutant | Annual mean concentration (2024) | WHO guideline | Exceedance | Dental / orthodontic significance |
|---|---|---|---|---|
| PM2.5 | 31.5 μg/m³ | 5 μg/m³/yr | 6.3× | Adsorption of Pb, Cd → dental caries, alveolar bone resorption |
| PM10 | ≥40 μg/m³ | 15 μg/m³/yr | >2.5× | Chronic periodontal inflammation, impaired osteogenesis |
| $NO_2$ | ~42 μg/m³* | 10 μg/m³/yr | >4× | Reduced BMD, inhibition of jaw growth |
| $SO_2$ | 4–7 ppb* | 40 μg/m³/day | Within limits | Enamel demineralisation under chronic exposure |

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ISSN: 2996-5101 (online) | ResearchBib (IF) = 10.81 IMPACT FACTOR
Volume-4| Issue-3| 2026 Published: |30-03-2026

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|---|---|---|---|---|
| CO | 299 ppb* | 4 mg/m³/day | Moderate | Tissue hypoxia → impaired periodontal trophism |
| Pb, Cd (PM-bound) | Data limited | Pb: 0.5 µg/m³ | Monitoring needed | Mineralisation disorders of dental hard tissues, malocclusion |

*Note. * — data based on current monitoring station measurements (IQAir, AQI.IN, 2024–2025); BMD — bone mineral density; PAHs — polycyclic aromatic hydrocarbons. WHO guidelines: PM2.5 — 5 µg/m³ (annual), 15 µg/m³ (24-hour); PM10 — 15 µg/m³ (annual).*

***Table 2. Source apportionment of PM2.5 pollution in Tashkent (World Bank, 2024)***

| Source | Share of PM2.5 (%) | Specific pollutants |
|---|---|---|
| Wind-blown dust (Aral Sea basin deserts) | ~36% | Silica dust, heavy metals, salts |
| Heating (coal, heavy fuel oil, biomass) | ~28% | $SO_2$, CO, PAHs, Pb, Cd |
| Road transport | ~16% | $NO_2$, CO, PM2.5, Pb (ageing vehicle fleet) |
| Industry | ~13% | $SO_2$, NOx, heavy metals |
| Other sources | ~7% | Construction dust, agricultural burning |

*Source: World Bank. Air Quality Assessment for Tashkent and the Roadmap for Air Quality Management Improvement in Uzbekistan. 2024.*

***Effects of Fine Particulate Matter (PM2.5/PM10) on the Dentofacial System***

Fine particulate matter PM2.5 (diameter ≤2.5 µm) and PM10 (diameter ≤10 µm) are the most thoroughly studied atmospheric pollutants in the context of general health. Upon inhalation, they penetrate deep into the airways, while ultrafine fractions (PM0.1) can translocate across the alveolar barrier into the bloodstream, reaching distant organs and tissues, including the periodontal tissues and the alveolar bone [11].

The key molecular mechanism underlying the pathological effect of PM on the oral cavity is the generation of reactive oxygen species (ROS), which trigger the NF-κB-dependent pro-inflammatory cascade, elevating levels of IL-1β, IL-6, and TNF-α [4, 12]. Chronic elevation of these cytokines in periodontal tissues promotes osteoclast differentiation, progressive alveolar bone resorption, and chronification

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ISSN: 2996-5101 (online) | ResearchBib (IF) = 10.81 IMPACT FACTOR
Volume-4| Issue-3| 2026 Published: |30-03-2026

of periodontitis — a condition that substantially complicates orthodontic treatment and increases the risk of relapse after its completion.

A further critical property of PM2.5 and PM10 is their capacity to adsorb and concentrate heavy metals — principally lead (Pb) and cadmium (Cd) [4, 13]. For Tashkent residents, this is particularly relevant: an ageing vehicle fleet that historically consumed leaded fuel (used in part until the 2010s) and coal-based heating are consistent sources of these metals. Inhalation of PM particles carrying lead and cadmium ions introduces them into the body bypassing the gastrointestinal barrier, resulting in higher bioavailability than the oral route of exposure.

From a paediatric dentistry and orthodontic perspective, the effect of PM on developing dental enamel and dentine is most significant. Primary teeth accumulate heavy metals throughout the entire mineralisation period (in utero and during the first 3 years of life), making them valuable biomarkers of chronic environmental exposure [14]. Studies of children from industrially polluted regions consistently report higher concentrations of Pb and Cd in primary tooth dentine, as well as elevated prevalence of dental caries and enamel hypoplasia compared with children from less polluted areas [14, 15].

***Role of Nitrogen Dioxide ($NO_2$) in Disrupting Facial Skeletal Bone Homeostasis***

Nitrogen dioxide is one of the hallmark pollutants of vehicular traffic load. According to Tashkent monitoring data, $NO_2$ concentrations reach approximately 42 μg/m³ — four times the WHO annual guideline of 10 μg/m³ [10]. Its orthodontic relevance derives from its capacity to disrupt bone homeostasis during the period of active facial skeletal growth.

The large Generation R cohort study (Netherlands, n = 5,966 children) identified a statistically significant inverse association between $NO_2$ and PM2.5 exposure at ages 1–4 years and bone mineral density (BMD) and bone mineral content (BMC) measured at age 6 years by dual-energy X-ray absorptiometry (DXA). Each 5 μg/m³ increment in PM2.5 concentration was associated with a reduction in BMD of 10.3 (95% CI: −15.8; −4.7) and in BMC of 14.6 (95% CI: −20.7; −8.4) [16]. The authors identified the age window of 1–4 years as a critical period of susceptibility for the effect of $NO_2$ on bone tissue.

In the context of the dentofacial system, slowed appositional growth of the basal bone of the maxilla and mandible under chronic $NO_2$ exposure may manifest as shortened jaw bodies, a tendency toward skeletal Class III (through relative maxillary retrognathia), or a vertical facial growth pattern. It should be emphasised

that this hypothesis, as applied to Tashkent, requires testing in a purpose-designed epidemiological study.

At the molecular level, $NO_2$ induces Th1/Th2 immune imbalance, activates the JAK-STAT signalling pathway, and suppresses osteoblastogenesis through oxidative damage to mitochondria of osteogenic precursor cells [17]. Concurrently, chronic $NO_2$ exposure reduces vitamin D levels by impairing cutaneous synthesis of its precursor (7-dehydrocholesterol) via increased tropospheric ozone, leading to secondary deterioration of calcium–phosphate metabolism and, consequently, of mineralisation of developing teeth and bones [18].

***Lead and Cadmium: Specific Mechanisms of Influence on Orthodontic Parameters***

Among the heavy metals associated with Tashkent's atmospheric pollution, lead (Pb) and cadmium (Cd) carry the greatest orthodontic significance. Both metals exhibit high affinity for calcium-binding proteins and compete with $Ca^{2+}$ ions during incorporation into the hydroxyapatite crystal lattice of enamel and dentine, thereby disrupting normal mineralisation and causing structural insufficiency of dental hard tissues [5, 19].

Lead also exerts a pronounced osteotropic effect: accumulating in bone tissue, it impairs remodelling by increasing the RANKL/OPG ratio and enhancing osteoclastogenesis [18]. In relation to orthodontics, chronic lead exposure may: (1) delay the eruption of permanent teeth — a recognised trigger of secondary dentofacial anomalies; (2) reduce alveolar BMD, negatively affecting the stability of biomechanical responses to orthodontic forces; and (3) increase the risk of relapse following active treatment as a result of compromised retention-phase bone quality [5].

Cadmium exerts nephrotoxic effects, disrupting phosphate and calcium reabsorption in the proximal renal tubules, leading to secondary bone demineralisation (an itai-itai–like phenotype under chronic exposure conditions) [13, 20]. In addition, Cd inhibits renal α1-hydroxylase, reducing vitamin D activation and impairing intestinal calcium absorption. For children residing in ecologically disadvantaged districts of Tashkent, this mechanism may substantially impair mineralisation of developing permanent teeth and the basal bone of the jaws [6, 13].

The literature review by Pietrzak-Fiećko et al. (2010) specifically addresses the risk for patients undergoing orthodontic treatment with fixed appliances under conditions of high Pb and Cd environmental burden [5]. The authors note that additional metal release from bracket alloys (nickel, chromium) — occurring through corrosion in an altered oral environment (pH shift, chronic inflammation)

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ISSN: 2996-5101 (online) | ResearchBib (IF) = 10.81 IMPACT FACTOR
Volume-4| Issue-3| 2026 Published: |30-03-2026

— synergistically amplifies the overall toxic effect on the oral mucosa and bone tissue in patients already subject to high ecological load.

***Pollutant-Associated Mouth Breathing as a Key Orthodontic Risk Factor***

Of particular interest to clinical orthodontics is the indirect mechanism by which atmospheric pollution affects the dentofacial system: the formation of chronic nasal obstruction and the consequent shift to oral breathing. This causal chain is the most clinically significant, as mouth breathing is itself recognised as one of the primary drivers of dentofacial anomalies in children.

Chronic exposure to PM2.5 and PM10 disrupts the barrier function of the upper airway mucosa through several mechanisms: damage to the ciliated epithelium, impairment of mucociliary clearance, induction of chronic inflammation, and allergic sensitisation [21, 22]. The result is a high prevalence of allergic rhinitis, nasal polyposis, and chronic adenoid hypertrophy in the paediatric population of large polluted cities. In Tashkent, pollutant-associated allergisation is further potentiated by high levels of biological allergens (pollen, mould spores) within the PM fraction, characteristic of the arid climate.

The orthodontic consequences of chronic mouth breathing are well documented across the entire spectrum of dentofacial pathology. Positioning of the tongue on the floor of the mouth — rather than against the palate — eliminates the functional stimulus for transverse maxillary growth, resulting in maxillary constriction. The change in mandibular position associated with mouth breathing — inferior displacement and retrusion — drives a vertical facial growth pattern with a tendency toward open bite. Chronic hypotonia of the orbicularis oris muscle and reduced labial pressure in mouth-breathing children create conditions for anterior tooth proclination. This constellation of changes is described in the literature as "adenoid facies" and encompasses: Class II malocclusion (Angle), narrowing of the maxillary arch, elongation of the lower facial third, open bite, a high arched palate, and maxillary or mandibular retrognathia [23, 24].

For Tashkent residents continuously exposed to elevated PM concentrations and allergenic bioaerosols, this mechanism of dentofacial anomaly formation is likely to be clinically significant. Indirect support for this assumption may be found in the high proportion of patients with complex skeletal anomalies requiring surgical correction observed by orthodontists at the Tashkent State Dental Institute — although systematic epidemiological data on this point are currently lacking.

***Table 3. Key mechanisms of atmospheric pollutant effects on the dentofacial system and their orthodontic consequences***

| Pollutant | Mechanism of action | Orthodontic consequence | Key sources |
|---|---|---|---|

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ISSN: 2996-5101 (online) | ResearchBib (IF) = 10.81 IMPACT FACTOR
Volume-4| Issue-3| 2026 Published: |30-03-2026

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|---|---|---|---|
| PM2.5 / PM10 | Oxidative stress → NF-κB → ↑IL-1β, TNF-α → osteoclast activation | Alveolar bone resorption, instability of treatment outcomes | Transport, dust storms |
| $NO_2$ | ↓BMD → impaired osteoblastogenesis, Th1/Th2 imbalance | Reduced jaw growth rate, mouth breathing → skeletal Class III | Road transport |
| Lead (Pb) | Competitive $Ca^{2+}$ substitution in hydroxyapatite, ↑RANKL → osteoclastogenesis | Enamel defects (hypoplasia), eruption delay, malocclusion | Ageing vehicles, heating |
| Cadmium (Cd) | Inhibition of renal Ca reabsorption, protein misfolding | Reduced mandibular BMD, elevated periodontitis risk under orthodontic load | Industry, coal |
| $SO_2$ / PAHs | Acid-mediated enamel demineralisation, mutagenic effects | Dental caries → tooth loss → secondary malocclusion | Heating, industry |
| Mouth breathing (↑ PM, allergy) | Altered tongue posture, lip hypotonia, adenoid facies | Distal/open bite, maxillary constriction, retrognathia | Upper airway sensitisation |

*Note. BMD — bone mineral density; PAHs — polycyclic aromatic hydrocarbons; RANKL — receptor activator of nuclear factor κB ligand.*

***Orthodontic Aspects: Impact on Treatment Outcomes***

Beyond the formation of dentofacial anomalies, chronic atmospheric pollutant exposure may exert adverse effects directly on the process and outcomes of orthodontic treatment. This dimension is considerably less represented in the literature; however, several well-grounded hypotheses merit attention.

Orthodontic tooth movement relies on controlled asymmetric alveolar bone remodelling: resorption on the pressure side (osteoclasts) and new bone formation on the tension side (osteoblasts). Chronic periodontal inflammation driven by PM-induced cytokines, combined with reduced BMD secondary to $NO_2$, Pb, and Cd exposure, may disrupt this balance. The theoretical consequences include reduced predictability of tooth movement, prolongation of treatment duration, and elevated risk of relapse at the conclusion of the active phase. Relapse risk is particularly pertinent when nasal obstruction persists, since the functional aetiology of the anomaly has not been eliminated [23, 25].

From a clinical standpoint, orthodontists practising in a high air-pollution environment should consider several practical implications: (1) the elevated

likelihood of subclinical periodontal inflammation in patients with prolonged residence in ecologically disadvantaged urban districts warrants professional hygiene and inflammatory parameter assessment before initiating active treatment; (2) in patients with nasal obstruction, consultation with an otolaryngologist and allergist with restoration of nasal breathing is a sine qua non condition for a stable orthodontic result; (3) retention protocols for patients from heavily polluted districts should be planned to be more prolonged and intensive than standard.

**Discussion**

The present review is, to the best of our knowledge, the first to systematise evidence on the possible mechanisms by which atmospheric pollution affects the dentofacial system within the specific ecological context of Tashkent. The analysis demonstrates that the multifactorial pollution profile of Tashkent — combining desert dust, coal combustion products, and transport emissions — creates a complex of biological exposures that concurrently targets all levels of the pathogenetic chain underlying dentofacial anomaly formation.

Chronic pollutant-associated inflammation occupies a central position in this chain. PM2.5-induced NF-κB activation in periodontal tissues and the upper airway mucosa simultaneously sustains alveolar bone resorption and promotes nasal mucosal hyperreactivity and adenoid hypertrophy as the substrate for mouth breathing. Pollutant effects are therefore realised through two parallel mechanisms: direct (mineralisation, osteogenesis) and indirect (respiratory function → muscular balance → dentofacial growth).

Extrapolating the Generation R cohort data [16] to Tashkent's pollution levels yields a concerning estimate: the annual mean PM2.5 in Tashkent (31.5 μg/m³ in 2024) is approximately six times higher than the concentrations at which significant BMD reduction was observed in Dutch children aged 1–4 years (≈5 μg/m³). This places Tashkent's paediatric population at potentially high risk for compromised facial skeletal bone quality — a consideration that should be reflected in orthodontic treatment planning approaches. However, direct extrapolation across populations requires caution: racial and constitutional differences in facial skeletal growth patterns, as well as variations in dietary status and socioeconomic conditions, must be taken into account.

It should be noted that the situation in Tashkent shows a positive trend: according to AQI data, the annual mean AQI declined from 99 in 2020 to 77 in 2025 [10]. The adoption of a new WHO-aligned PM2.5 standard by the Ministry of Health of Uzbekistan in December 2024, and the development of the "Roadmap for Air Quality Management in Uzbekistan" by the World Bank, reflect systemic efforts to reduce the ecological burden [8]. Nevertheless, the accumulated biological effects

of chronic pollutant exposure on the already-forming dentofacial systems of children born and growing up in Tashkent throughout the 2010s and early 2020s will continue to manifest as clinical cases for many years to come.

**Limitations.** The present review has several limitations. First, no studies have been specifically designed to investigate the relationship between Tashkent's air pollution and the prevalence of dentofacial anomalies; all conclusions are therefore extrapolative. Second, the narrative format does not allow for quantitative data synthesis. Third, the availability of systematic epidemiological data on dental morbidity in Uzbekistan is limited, precluding direct comparison of existing ecological data with trends in dentofacial anomaly prevalence.

**Conclusion**

Tashkent's atmospheric pollution — characterised by chronic exceedance of WHO PM2.5 guidelines by a factor of 6.3 — constitutes a systemic environmental risk factor capable of adversely affecting the dentofacial system of city residents through a complex of interrelated mechanisms. Based on the analysis of the literature, the following conclusions are drawn:

1. Chronic PM2.5/PM10 exposure initiates NF-κB-mediated pro-inflammatory conditions in periodontal tissues, promotes osteoclastogenesis, and serves as a vehicle for the systemic introduction of heavy metals (Pb, Cd) that impair enamel and dentine mineralisation, tooth eruption timing, and the BMD of alveolar structures.
2. Nitrogen dioxide at concentrations typical of Tashkent (≈42 μg/m³; WHO guideline 10 μg/m³) inhibits osteoblastic activity during critical periods of facial skeletal growth (ages 1–4 years), potentially reducing the rate of appositional jaw bone growth and worsening orthodontic treatment prognosis.
3. Pollutant-associated chronic nasal obstruction is the key indirect mechanism for dentofacial anomaly formation in Tashkent residents: the shift to mouth breathing predictably results in maxillary arch constriction, a vertical facial growth pattern, and the development of distal and open bite malocclusions and retrognathia.
4. Priority directions for future research include: (a) an epidemiological study of dentofacial anomaly prevalence among Tashkent children stratified by ecological district; (b) assessment of facial skeletal BMD in children as a function of residential PM exposure level; and (c) development of clinical guidelines for the management of orthodontic patients under conditions of chronic atmospheric pollutant exposure.

**Declaration of Interests**

The authors declare no conflict of interest. No external funding was received for this study.

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