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Air pollution as a silent driver of osteoporosis: A narrative review of vitamin D impairment and skeletal vulnerability in female populations

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Abstract

The intensification of urban air pollution and the rising concentration of particulate matter have emerged as significant environmental barriers to vitamin D synthesis, posing increasing risks to women's skeletal health. Consequently, reliance on sunlight in metropolises is no longer sufficient to meet physiological requirements. This narrative review investigates the synergy between exposure to atmospheric pollutants (such as PM2.5 and NOx) and the concurrent prevalence of vitamin D deficiency and early-onset osteoporosis in women. A narrative search was conducted across international (PubMed, Scopus, Web of Science, Google Scholar) and national (SID, Magiran) databases for studies published until 2025. From an initial pool of 412 records, 27 high-quality studies were selected for final inclusion following a rigorous three-stage screening process based on title, abstract, and full-text evaluation. Literature analysis revealed that air pollutants, by absorbing UVB radiation, reduce serum 25(OH)D levels in pregnant and menopausal women by up to 18.8% and increase the risk of vitamin D deficiency by up to 3.5-fold. Beyond reducing vitamin D, particulate matter directly shifts the bone remodeling balance toward resorption by inducing oxidative stress and systemic inflammation, leading to a significant decrease in bone mineral density (BMD) in the spine and hip. The results indicate that air pollution is an independent risk factor for women's health. Health policy-making must focus on reducing traffic-related pollutants and revising vitamin D supplementation protocols in polluted regions to prevent debilitating fractures in the future.

Keywords: Air Pollution, Vitamin D deficiency, Osteoporosis, Women, Bone health.

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Introduction

Air pollution, as a major environmental challenge, has garnered significant attention in both modern and developing societies (1-3). With rapid urbanization and industrial expansion, atmospheric pollutants have extensively affected human quality of life, leaving populations in constant exposure to particulate matter (PM) and toxic gases (4,5). Among the numerous consequences of this issue, vitamin D deficiency is recognized as a global pandemic affecting more than one billion people worldwide (6). Although vitamin D is naturally produced through cutaneous synthesis triggered by ultraviolet B (UVB) radiation, several environmental factors can disrupt this vital process (7). Air pollutants, particularly fine particulate matter (PM2.5), PM10, and nitrogen oxides (NOx), significantly impede the synthesis of this vitamin by absorbing and scattering UVB radiation before it reaches the earth's surface (8,9).

A primary concern regarding the concurrent impact of air pollution and vitamin D deficiency is the specific vulnerability of women across various physiological stages (10,11). This heightened vulnerability is largely attributed to distinct hormonal fluctuations (such as declines in estrogen during menopause) and unique metabolic demands (e.g., increased calcium requirements during pregnancy), which make women more susceptible to the downstream effects of low vitamin D levels. Numerous studies have demonstrated that women during pregnancy and menopause are at a higher risk of complications arising from this deficiency due to these specific metabolic demands and hormonal fluctuations (12-14). For instance, exposure to pollutants from biomass burning and urban particulate matter has been significantly associated with reduced serum levels of 25-hydroxyvitamin D (25(OH)D) in menopausal and pregnant women (15,9). Epidemiological evidence suggests that the prevalence of vitamin D deficiency among women residing in polluted urban areas, such as Tehran and Beirut, remains alarmingly high despite these regions being sunny; living in highly polluted areas substantially increases the odds of developing this deficiency (16,14).

One of the most critical clinical consequences of this condition in women is the acceleration of bone resorption and the early onset of osteoporosis (17). Osteoporosis, characterized by decreased bone mineral density (BMD) and the microarchitectural deterioration of bone tissue, imposes a heavy burden on healthcare systems (18,19). Recent studies have confirmed that air pollution exacerbates the pathogenesis of osteoporosis not only by reducing vitamin D levels but also through direct mechanisms such as inducing oxidative stress and systemic inflammation (20,21). Pollutants like nitrogen dioxide (NO2) and particulate matter can shift the balance of bone remodeling toward resorption by stimulating inflammatory cytokines, leading to decreased bone density, particularly in sensitive areas such as the lumbar spine and hip (22,20,23). This process doubles the risk of bone fractures in postmenopausal women, who naturally face declining estrogen levels (24-26).

Given the potential risks of air pollution to women's skeletal and metabolic health, a precise understanding of the interaction between environmental pollutants and vitamin D status has become an essential priority. While extensive research has focused on the respiratory effects of air pollution, a knowledge gap remains in comprehensively examining the synergistic mechanisms of air pollution in reducing vitamin D and its direct impact on women's bone health. This narrative review aims to evaluate the existing evidence regarding the role of urban air pollution in exacerbating vitamin D deficiency and to analyze the potential of this environmental factor in accelerating osteoporosis in women, ultimately providing strategies to reduce the disease burden in high-risk populations.

Method

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This narrative review was conducted to aggregate authoritative scientific literature and evaluate existing evidence. International databases, including PubMed, Scopus, Web of Science, ScienceDirect, and Google Scholar, as well as national databases (such as SID and Magiran), were searched without any time restrictions.

Search strategy

The search was performed using specialized English keywords and their Persian equivalents, combined through the logical application of Boolean operators (AND, OR) as follows:

- Environmental/Pollution terms: "Air Pollution", "Urban Air Pollution", "Ambient Air Pollution", and "Smog".
- Vitamin D terms: "Vitamin D Deficiency", "Low Vitamin D", and "25-hydroxyvitamin D".
- Skeletal Health terms: "Osteoporosis", "Bone Mineral Density" (BMD), and "Bone Health".
- Population terms: "Women" and "Female".

Study selection and screening process

The initial electronic search yielded 412 records Published until 2025. After removing 145 duplicates, 267 unique citations were screened. In the first phase, 184 articles were excluded based on title and abstract relevance. The remaining 83 full-text articles were rigorously assessed, leading to the exclusion of 56 studies due to lack of gender-disaggregated data, focus on animal models, or insufficient control for seasonal variables. Ultimately, 27 high-quality studies were selected for final inclusion and data extraction. The detailed process of study identification, screening, and inclusion is illustrated in the PRISMA flow diagram (Figure 1).

Inclusion and exclusion criteria

Inclusion criteria

Original research articles (cross-sectional, cohort studies), systematic reviews, and meta-analyses that specifically investigated the direct association between exposure to major atmospheric pollutants (particulate matter [PM10, PM2.5], nitrogen dioxide [NO_2], and sulfur dioxide [SO_2]) and serum 25-hydroxyvitamin D levels or bone mineral density (BMD) in female populations. Special emphasis was placed on studies evaluating vulnerable groups, including pregnant, premenopausal, and postmenopausal women residing in highly polluted geographical regions (e.g., Iran, China, and Lebanon).

Exclusion criteria

1. Studies focusing solely on animal models without relevance to human physiology.
2. Research involving only male populations or lacking gender-disaggregated data.
3. Case reports, editorials, or articles for which the full text was unavailable.
4. Studies that did not account for environmental confounders, particularly seasonal variations and solar radiation levels, in their primary analyses. “Insufficient control for seasonal variations” was defined as the failure to include season of blood sampling or solar irradiance as a covariate in multivariate models when assessing the relationship between air pollution and vitamin D status.

Quality assessment of included studies

The methodological quality of the 27 included studies was independently evaluated by two reviewers (HFK and MB) using established tools. For cross-sectional and cohort studies, the Newcastle-Ottawa Scale (NOS) was applied, with scores ranging from 0 to 9. Studies scoring ≥7 were considered high quality, scores of 5–6 moderate quality, and scores <5 low quality. For systematic reviews and meta-analyses, the AMSTAR-2 checklist was used. Any disagreements between reviewers were resolved through consensus discussion. Only studies with moderate to high quality (NOS ≥5 or AMSTAR-2 moderate confidence) were retained for data extraction.

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Data extraction and synthesis

Key data were extracted from the 27 eligible sources using a standardized data extraction form developed by the authors. The extracted information included:

- Bibliographic details: first author, year of publication, country
- Study design: cross-sectional, cohort, systematic review, or meta-analysis
- Population characteristics: sample size, life stage of women (pregnancy, perimenopause, postmenopause), geographic region (e.g., Tehran, Beirut, China)
- Exposure measurement: type of pollutant (PM2.5, PM10, NO_2, SO_2, AQI), exposure duration (short-term vs. long-term), and source of air quality data (e.g., ground monitors, satellite)
- Outcome measures: serum 25(OH)D level (ng/mL or nmol/L), prevalence of vitamin D deficiency, bone mineral density (g/cm²), osteoporosis diagnosis
- Statistical associations: odds ratios (OR), regression coefficients, percentage changes, and 95% confidence intervals
- Confounders controlled: particularly seasonal variations, solar radiation, body mass index, age, and socioeconomic status

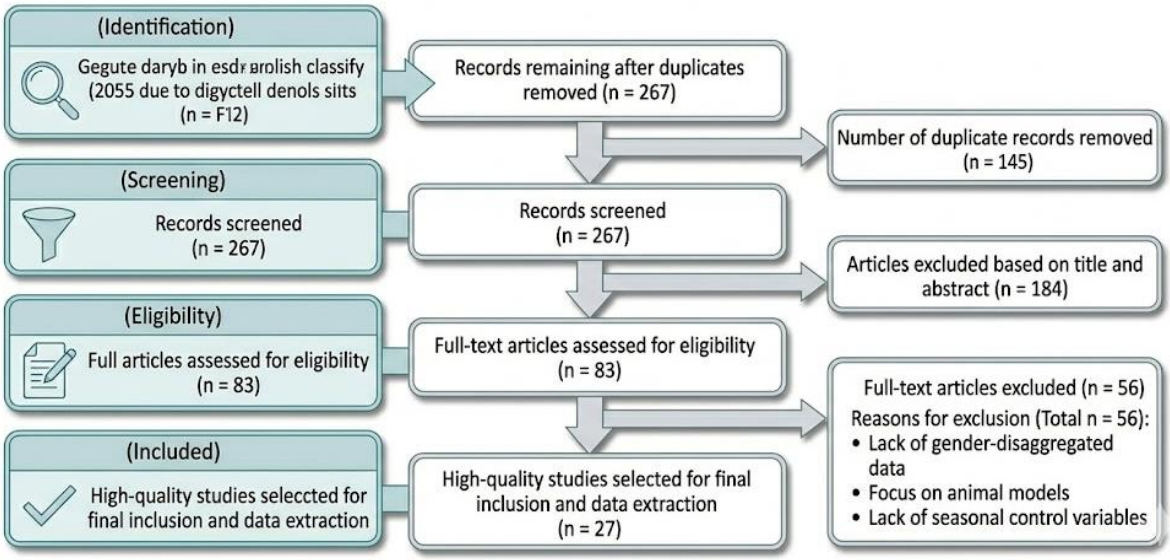

Figure 1. PRISMA diagram for searching resources

Results

The present study demonstrates a significant association between urban air pollution, vitamin D status, and bone health in women. The findings are categorized into three primary axes: (1) Impact of Air Pollution on Serum Vitamin D Levels; (2) Association Between Air Pollution and Risk of Vitamin D Deficiency; and (3) Skeletal Consequences: Reduced Bone Density and Osteoporosis.

Impact of air pollution on serum vitamin D levels

Quantitative assessments indicate that exposure to atmospheric pollutants has a significant inverse effect on serum 25-hydroxyvitamin D (25(OH)D) levels. As shown

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in Figure 2, a prospective cohort study on postmenopausal women revealed that mean serum vitamin D levels during peak pollution periods (driven by biomass burning and PM2.5) were significantly lower than during low-pollution periods (21.5 vs. 24.6 ng/mL). This 18.8% reduction in vitamin D levels highlights the critical importance of seasonal fluctuations in air quality (15). Furthermore, a longitudinal study by Zhao et al. (9) involving 3,285 pregnant women demonstrated that for every 10 µg/m³ increase in PM2.5 and PM10 concentrations during pregnancy, serum vitamin D levels decreased by 4.62% and 5.06%, respectively (9). The primary mechanism for this phenomenon is the absorption of ultraviolet B (UVB) photons by pollutants, which prevents sufficient energy from reaching the skin for cutaneous vitamin D synthesis. He et al. (8), examining over 22,000 individuals in China, reported a strong negative correlation between the Air Quality Index (AQI) and vitamin D levels, with the lowest levels recorded during winter when pollutant concentrations reach their maximum (8).

Association between air pollution and risk of vitamin D deficiency

Statistical analyses show that residing in polluted urban environments significantly increases the Odds Ratio (OR) of clinical vitamin D deficiency (27). As illustrated in , various studies have reported a 3Figure substantial increase in risk. Chedid et al. (16) found in a Lebanese adult population that living in high-pollution areas (such as metropolitan Beirut) compared to clean areas increases the risk of vitamin D deficiency by 45% (OR=1.45) (16). Similarly, Phimphilai et al. (15) reported that seasonal exposure to severe air pollution (haze periods) increases the risk of vitamin D deficiency by 3.5 times (OR=3.50), an

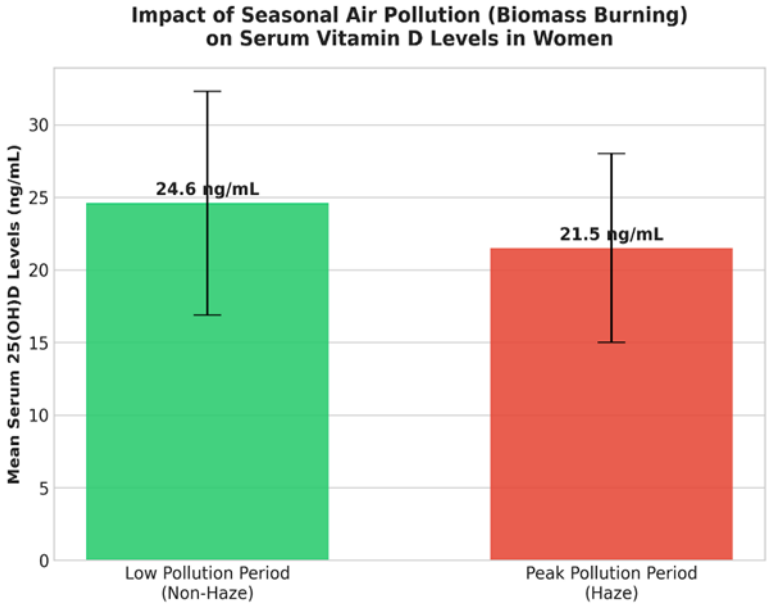

Figure 2. Comparison of mean serum vitamin D levels during low air pollution (Non-Haze) and peak pollution (Haze) periods in postmenopausal women (15).

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alarmingly high figure. In Iran, review studies by Ghazizadeh et al. (6) and Sepandi et al. (14) confirmed that the prevalence of vitamin D deficiency among women in industrial and polluted cities (such as Tehran, Mashhad, and Ahvaz) is significantly higher than in other regions. For instance, the prevalence of vitamin D deficiency in pregnant women in Tehran has been reported as high as 76.8%, directly correlating with the winter season and the air inversion phenomenon (6,14).

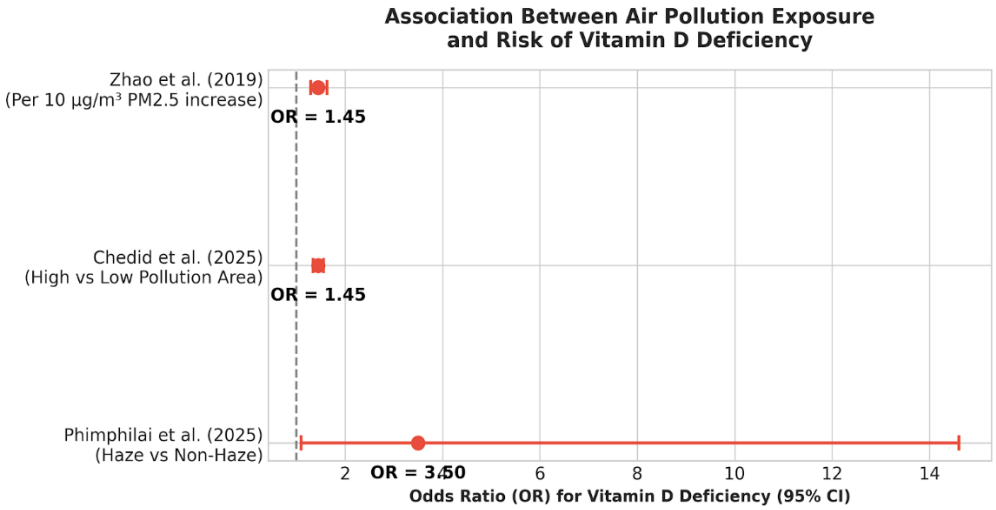

Figure 3. Forest plot representing the increased risk (Odds Ratio - OR) of vitamin D deficiency associated with air pollution exposure in selected studies. As illustrated, all included studies report a significant increase in risk (OR > 1) (16,15).

Skeletal consequences: Reduced bone density and osteoporosis

Beyond indirect effects via vitamin D reduction, air pollutants exert direct toxic effects on bone tissue (22). As shown in Figure 4, long-term exposure to gaseous pollutants and particulate matter is associated with decreased bone mineral density (BMD) in the sensitive lumbar spine region. A cohort study by Prada et al. (23) showed that PM10 (5-year average) had the most significant negative impact on bone density, leading to a reduction of 0.064 g/cm² in lumbar spine BMD. Additionally, nitrogen oxides (NO2 and NO), primarily originating from urban traffic, were significantly associated with decreased bone density (23).

This decline in bone density places postmenopausal women who are already predisposed to osteoporosis due to estrogen depletion at a double risk for bone fractures. Pang et al. (20), in their systematic review, detailed the mechanisms of this damage, showing that pollutants increase the activity of osteoclasts (bone-resorbing cells) by inducing oxidative stress and systemic inflammation (20). As summarized in Table 1, evidence from diverse countries and population groups consistently emphasizes the negative role of air pollution in skeletal health.

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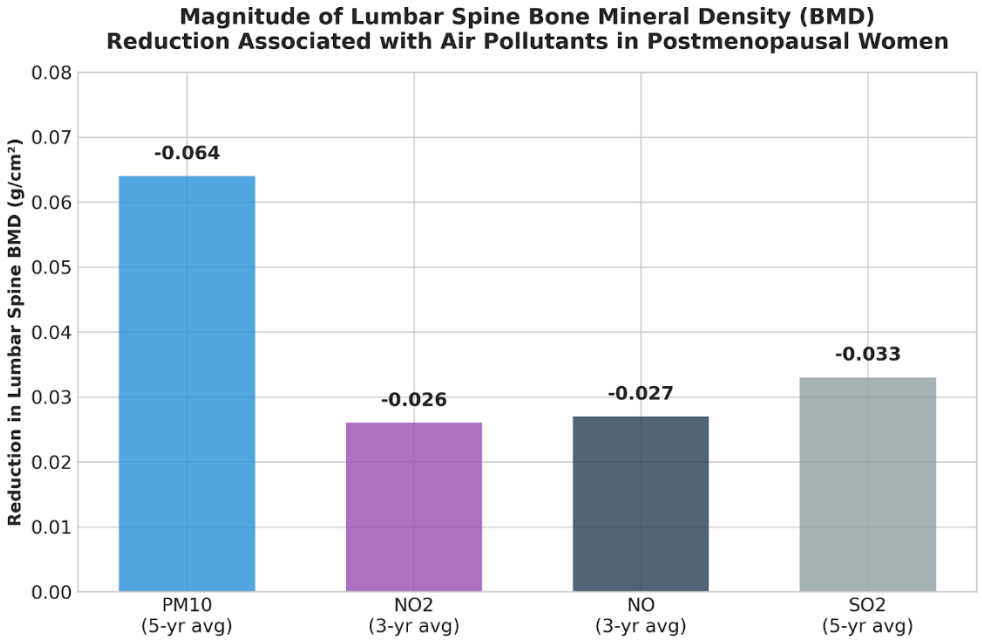

Figure 4. Reduction in Bone Mineral Density (BMD) of the lumbar spine in postmenopausal women associated with exposure to various air pollutants. As illustrated, particulate matter (PM10) exerts the most significant negative impact (23).

Table 1. Summary of characteristics and key findings of the reviewed studies investigating the association between air pollution, vitamin D status, and bone health.

Country	Study Design	Population	Exposure Factor	Key Findings	Reference
Lebanon	Cross-sectional	19,452 Adults	Urban Air Pollution (Beirut)	45% increased risk of vitamin D deficiency in high-pollution areas (OR=1.45).	(5)
Thailand	Cohort	77 Postmenopausal Women	Seasonal PM2.5	18.8% decrease in Vitamin D levels and 3.5-fold increased risk of deficiency during the polluted season.	(16)
USA	Cohort	9,041 Postmenopausal Women	PM10, NOx	Significant reduction in lumbar spine and hip BMD; strong correlation with nitrogen oxides.	(18)
Iran	Meta-analysis	General Population	Metropolitan Pollution	Prevalence of vitamin D deficiency exceeding 50% in Iranian women; associated with living in industrial cities.	(9)
Malaysia	Review	General Population	Particulate Matter (PM)	Confirmation of PM-induced inflammatory mechanisms in increasing risks of osteoporosis and fractures.	(15)
China	Cross-sectional	22,387 Adults	Air Quality Index (AQI)	Significant negative correlation between AQI and serum vitamin D levels.	(11)

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Iran	Cross-sectional	267 Pregnant Women	Season/Location (Tehran)	76.8% prevalence of deficiency; significant association with winter (peak pollution period).	(22)
China	Cohort	3,285 Pregnant Women	PM2.5, PM10	5% decrease in vitamin D levels for every 10 µg/m³ increase in particulate matter.	(27)
Iran	Review	General Population	Dust/Cloud Cover	Direct correlation between polluted/cloudy days and the prevalence of vitamin D deficiency.	(4)

Discussion

The findings of this review consolidate air pollution as an independent, modifiable environmental determinant of vitamin D deficiency and accelerated bone loss in women, particularly during critical physiological windows such as pregnancy and menopause (8,9). Unlike previous studies that focused primarily on respiratory outcomes, the present synthesis reveals a dual pathogenic pathway: pollutants disrupt endocrine control of calcium homeostasis by blocking UVB-mediated vitamin D synthesis, while simultaneously exerting direct pro-resorptive effects on skeletal tissue through oxidative and inflammatory cascades (20,23).

Mechanistic pathways: Beyond UVB attenuation

The inverse dose-response relationship between particulate matter and serum 25(OH)D levels is now well-established across diverse geographical settings (8,9). However, the mechanistic interpretation requires careful distinction between two phenomena: true vitamin D depletion versus reduced cutaneous synthesis. The finding that approximately 70% of PM-induced vitamin D reduction is mediated by decreased UVB radiation (9) suggests that air pollution creates a functional "solar barrier." This is conceptually distinct from seasonal variation alone, because pollution can override even high solar angles—as observed in Beirut and Tehran, where sunny climates co-exist with endemic deficiency (14,16).

Clinically, this implies that sunlight exposure recommendations, which form the backbone of public health guidelines in many sunny countries, are no longer sufficient in polluted megacities. Health practitioners should therefore consider local Air Quality Index (AQI) data when advising women about sun exposure, rather than relying solely on latitude or season (8).

Direct skeletal toxicity: Inflammation without vitamin D mediation

Perhaps the more clinically significant finding is that air pollution harms bone independently of vitamin D status. The cohort study by Prada et al. (23) demonstrated that nitrogen oxides (NOx) and PM10 are associated with reduced lumbar spine BMD even after adjusting for serum vitamin D levels, physical activity, and socioeconomic status. This indicates a direct osteotoxic effect. The proposed mechanism—systemic elevation of TNF-α and IL-6 following inhalation of fine particles—is biologically plausible (20). These cytokines activate NF-κB signaling in osteoclast precursors, promoting differentiation and bone resorption (20).

Importantly, this pathway overlaps with the pathophysiology of postmenopausal osteoporosis, where estrogen withdrawal already increases baseline inflammatory tone. Thus, women living in high-pollution areas may experience a "double hit": estrogen deficiency plus pollutant-induced inflammation. This synergy explains why the risk of deficiency increased 3.5-fold in perimenopausal women during haze episodes (15), a magnitude far exceeding the effect of either factor alone.

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Vulnerable subpopulations and critical windows

Three distinct vulnerable groups emerge from this review:

Pregnant women: The fetus requires substantial calcium for skeletal mineralization, and maternal vitamin D is the sole source. The reduction in 25(OH)D per increase in PM concentration observed by Zhao et al. (9) translates into a clinically meaningful increase in risk of neonatal hypocalcemia and impaired fetal bone development. Moreover, pregnancy is characterized by physiological hemodilution and increased glomerular filtration rate, which may further lower circulating 25(OH)D levels independently of synthesis (14).

Postmenopausal women: This group faces the highest absolute risk of osteoporotic fracture. The combination of age-related decline in cutaneous vitamin D synthesis (due to skin thinning and reduced 7-dehydrocholesterol content), estrogen loss, and chronic pollution exposure creates a perfect storm. The finding that NOx specifically targets the lumbar spine (23)—a site prone to fragility fractures—has direct implications for screening protocols.

Women in seasonal pollution hotspots: Biomass burning episodes (15) and winter inversions (14) produce acute, high-intensity PM exposure. Unlike chronic urban pollution, these events are predictable and short-term, offering a window for targeted prophylaxis.

Comparison with existing literature

Our findings align with the emerging field of "environmental osteology." Previous systematic reviews (e.g., Pang et al. (20)) have reported similar associations but did not specifically address gender-disaggregated effects. The present review fills this gap by focusing exclusively on women, who bear a disproportionate burden due to hormonal factors, longer life expectancy, and higher prevalence of osteoporosis globally.

Compared to studies from high-income countries with lower baseline PM levels, the effect sizes observed in Iran (6,14), China (8,9), and Lebanon (16) are substantially larger. This suggests a threshold effect: the health impact of pollution on bone may accelerate exponentially once PM2.5 exceeds certain levels, values routinely seen in Tehran, Beirut, and Beijing (14,16).

Limitations of the review

Several limitations should be acknowledged. First, the included studies are predominantly cross-sectional or cohort designs, which cannot establish causality despite rigorous adjustment for confounders. Second, measurement of personal exposure to pollutants remains imprecise; most studies relied on area-level monitoring data, which introduces non-differential misclassification and likely underestimates true effect sizes. Third, there is heterogeneity in vitamin D assay methods and deficiency definitions across studies (6,14), limiting meta-analysis. Fourth, few studies controlled for dietary vitamin D intake, calcium supplementation, or outdoor physical activity—all potential confounders or effect modifiers. Finally, the exclusion of non-English language studies (except Persian) may introduce language bias.

Clinical and policy implications

From a clinical perspective, the evidence supports revising current vitamin D supplementation guidelines. For women residing in areas with high pollution burden, empirical supplementation may be justified without routine testing, particularly during winter or haze episodes (14,15). This is a departure from the current "test-and-treat" approach, which is often impractical in low-resource settings.

From a public health perspective, reducing traffic-related NOx emissions—which showed the strongest bone toxicity (23)—should be prioritized alongside PM reduction. Low-emission zones, promotion of electric public transport, and stricter vehicle emissions standards are not merely respiratory health interventions but also skeletal health interventions.

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Future research directions

Prospective cohort studies with personal PM monitors, repeated measures of 25(OH)D and bone turnover markers, and long-term fracture follow-up are urgently needed. Additionally, randomized controlled trials of higher vitamin D supplementation doses in polluted regions versus standard doses would provide high-quality evidence (15). Finally, epigenetic and metabolomic studies could identify biomarkers of susceptibility, enabling targeted prevention for genetically vulnerable women.

Conclusion

The comprehensive review of scientific evidence in this study demonstrates that urban air pollution has emerged as a serious and silent threat to the metabolic and skeletal health of women, extending far beyond its well-known respiratory impacts. Convergent findings from epidemiological and clinical studies confirm that atmospheric pollutants particularly particulate matter (PM2.5 and PM10) and nitrogen oxides compromise women's health through a dual and synergistic mechanism: first, by creating a physical barrier against UVB radiation and disrupting natural vitamin D synthesis; and second, by inducing oxidative stress and systemic inflammation that directly accelerates bone resorption.

This study highlights that women residing in polluted metropolises and industrial zones face an alarming prevalence of vitamin D deficiency, despite living in sunny latitudes. This condition is exacerbated during sensitive physiological stages such as pregnancy and menopause, when the body's demand for calcium and vitamin D increases. Strong evidence suggests that even seasonal and short-term exposure to high pollutant concentrations (such as during thermal inversion or biomass burning events) can lead to a rapid depletion of vitamin D stores and increase the risk of early-onset osteoporosis, particularly in the spine and hip.

Consequently, air pollution should be recognized as an independent and modifiable risk factor in screening guidelines for osteoporosis and vitamin D deficiency in women. Given the rising trend of urbanization and air pollution in developing countries, relying solely on sunlight to meet vitamin D requirements is no longer a safe scientific recommendation in these regions. To mitigate the burden of these diseases, it is proposed that health policymakers and women's healthcare providers prioritize the following strategies:

1. Revision of Supplementation Protocols: Increasing prophylactic doses of vitamin D for pregnant and menopausal women living in areas with unhealthy Air Quality Index (AQI) levels.
2. Targeted Monitoring: Periodic assessment of bone mineral density and vitamin D levels in women residing in pollution hotspots, regardless of chronological age.
3. Urban Management: Focusing on the reduction of traffic-related pollutants (NOx), which have shown the strongest correlation with bone mass loss in women.

This multilateral approach can serve as an effective step toward preserving skeletal health and preventing debilitating complications in future generations of women.

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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