Nutr Res Pract. 2026 Jun;20(3):504-520
https://doi.org/10.4162/nrp.2026.20.3.504
pISSN 1976-1457·eISSN 2005-6168

Nutrition Research and Practice

Research Paper

Check for updates

Vitamin D deficiency modifies the association between ambient $PM_{2.5}$ exposure and possible sarcopenia in Korean adults: evidence from the HEXA study

Jimi Kim §

Department of Food and Nutrition, Changwon National University, Changwon 51140, Korea

OPEN ACCESS

Received: Feb 17, 2026
Revised: Apr 1, 2026
Accepted: Apr 16, 2026
Published online: May 15, 2026

§Corresponding Author:
Jimi Kim
Department of Food and Nutrition, Changwon National University, 20 Changwondaehak-ro, Uichang-gu, Changwon 51140, Korea.
Tel. +82-55-213-3514
Fax. +82-55-213-3510
Email. jmkim206@changwon.ac.kr

ORCID iDs
Jimi Kim
https://orcid.org/0000-0002-3931-4061

Funding
This research was supported by the Junior Faculty Research Support Grant of Changwon National University in 2024, the Glocal University Project supported through the RISE (Regional Innovation System & Education) program funded by the Ministry of Education (MOE), Republic of Korea, and the National Research Foundation of Korea (NRF) grant

ABSTRACT

BACKGROUND/OBJECTIVES: Maintaining muscular power is essential for preventing sarcopenia and ensuring optimal physical performance. Although inadequate vitamin D levels and environmental pollutants are increasingly recognized as threats to musculoskeletal integrity, their synergistic impact has not been fully established. This study examined how the vitamin D status and atmospheric contaminants independently and collectively influence the prevalence of suspected sarcopenia, defined strictly as low handgrip strength.
SUBJECTS/METHODS: This cross-sectional analysis included 20,304 individuals from the Health Examinees cohort using baseline data. Vitamin D insufficiency was categorized as circulating 25-hydroxyvitamin D [25(OH)D] concentrations below 20 ng/mL. Residential address-linked data were used to estimate the annual mean exposure to nitrogen dioxide (NO_2) and particulate matter ($PM_{2.5}$ and PM_{10}). Possible sarcopenia was distinguished from clinically diagnosed sarcopenia by relying solely on gender-specific low grip strength cut-offs, according to the Asian Working Group for Sarcopenia guidelines. Multivariable linear and logistic regression models were used to assess the associations, and the interaction terms were included to evaluate the effect modification according to the vitamin D status.
RESULTS: Suboptimal vitamin D levels were widespread in the cohort (65.0%) and strongly correlated with increased pollutant exposure. After accounting for all potential confounders, elevated $PM_{2.5}$, PM_{10}, and NO_2 levels were associated with reduced serum 25(OH)D levels and a greater likelihood of suspected sarcopenia. Significant interplay was detected between $PM_{2.5}$ exposure and the vitamin D status, suggesting a combined effect on muscle health (P for interaction = 0.005).
CONCLUSION: Vitamin D deficiency and air pollution exposure are independently associated with possible sarcopenia. In particular, $PM_{2.5}$ and a vitamin D deficiency exhibit a synergistic adverse association. These findings suggest that adequate vitamin D levels may be protective against pollution-associated declines in muscle strength, highlighting the need for longitudinal studies to explore integrated nutritional and environmental prevention strategies.

Keywords: Vitamin D deficiency; air pollution; hand strength; sarcopenia

funded by the Korea government (Ministry of Science and ICT [MSIT]) (grant No. RS-2025-16071386), and the funders had no role in the study design, data collection, analysis, or interpretation, manuscript preparation, or the decision to publish.

Conflict of Interest

The author declares no potential conflicts of interests.

INTRODUCTION

Sarcopenia is characterized as a degenerative and advancing disorder involving the loss of skeletal muscle mass alongside declines in strength and physical performance [1,2]. Within the context of a rapidly aging global demographic, this condition has emerged as a pivotal public health concern because of its strong associations with heightened risks of falls, fractures, and overall mortality [3,4]. The development of sarcopenia reflects a multifactorial process in which biological aging interacts with genetic predisposition, lifestyle factors, nutritional status, and environmental exposures [1,5].

Among the nutritional interventions for sarcopenia, vitamin D stands out as a vital factor in preserving the musculoskeletal integrity [6]. The biological impact of this vitamin is mediated by its interaction with vitamin D receptors in muscle cells, a process that stimulates protein synthesis and prevents fiber atrophy [7]. Previous studies have consistently reported that lower serum concentrations of 25-hydroxyvitamin D (25[OH]D) are associated with reduced muscle strength and impaired physical performance, and vitamin D deficiency has been identified as an independent risk factor for sarcopenia [8-10]. Nevertheless, vitamin D deficiency is quite prevalent in contemporary societies, reflecting inadequate dietary intake and insufficient sunlight exposure [11,12].

Accumulating evidence suggests that air pollution may adversely affect musculoskeletal health, extending its impact beyond the respiratory and cardiovascular systems [13]. Prolonged exposure to ambient air pollutants, including particulate matter ($PM_{2.5}$ and PM_{10}) and nitrogen dioxide (NO_2), has been shown to provoke systemic inflammation and oxidative stress, biological processes that can accelerate muscle protein breakdown and impair the muscle regenerative capacity [14]. Among these pollutants, fine $PM_{2.5}$ is particularly concerning. $PM_{2.5}$ can penetrate deeply into the alveolar region and cross the blood-air barrier into the systemic circulation owing to its smaller aerodynamic diameter than larger particles (PM_{10}) or gaseous pollutants (NO_2), exerting more profound systemic toxicity and oxidative stress on the peripheral tissues, such as skeletal muscle. Accordingly, several epidemiological studies have reported significant associations between elevated air pollution levels and reduced handgrip strength and decreased muscle mass among older adults [15-18].

Beyond its immediate toxicological effects on muscle tissue, environmental pollution may also interfere with the natural production of vitamin D by the body. Specifically, atmospheric particulate matter can block or dissipate ultraviolet B (UVB) rays, reducing the efficiency of cutaneous vitamin D synthesis [19,20]. Furthermore, behavioral adaptations to poor air quality, including decreased outdoor activities and increased time spent indoors, further limit natural sun exposure, compounding the risk of vitamin D insufficiency in polluted environments. These combined physical and behavioral mechanisms suggest that air pollution may increase the risk of sarcopenia through direct detrimental effects on skeletal muscle, and indirectly by inducing or exacerbating vitamin D deficiency [21]. Despite these connections, most studies focused primarily on the independent main effects of atmospheric exposure and nutritional status on muscle health, treating them as isolated variables. Consequently, there is a notable lack of evidence regarding whether these 2 factors act synergistically to exacerbate the risk of sarcopenia, particularly regarding diminished muscle strength. Although muscle strength is a primary indicator of sarcopenia and a key determinant of physical function, epidemiological studies formally evaluating the interaction between environmental pollutants and vitamin D status remain highly limited. Addressing

Nutrition Research and Practice

this specific research gap is essential to understanding the compounded risks posed by modern environmental and nutritional challenges.

The present study systematically assessed how vitamin D insufficiency and ambient air pollutants contribute individually and jointly to the development of sarcopenia, leveraging a comprehensive dataset from the Health Examinees (HEXA) cohort. Furthermore, this study explored the potential modifying role of the vitamin D status in the relationship between pollutant exposure and reduced handgrip strength among Korean adults.

SUBJECTS AND METHODS

Study population

This study is a cross-sectional analysis using the baseline data from the HEXA study, a population-based cohort within the Korean Genome and Epidemiology Study (KoGES). The HEXA study was conducted between 2004 and 2013 and enrolled men and women aged 40 yrs or older who visited 38 health examination centers and hospitals nationwide in Korea [22]. Detailed information on the demographic characteristics, lifestyle factors, medical history, and biological samples was collected at the baseline. Among the initial 173,195 participants enrolled in the HEXA cohort, 152,891 individuals were excluded because of missing data on any of the following essential variables: serum 25(OH)D concentrations, valid residential address information required for ambient air pollution linkage, handgrip strength, or key covariates (including demographic characteristics, lifestyle behaviors, and medical history). After applying these exclusion criteria, 20,304 participants, representing approximately 11.7% of the initial cohort, were included in the final analytical sample. **Fig. 1** provides a detailed flowchart of the participant selection process. This study was conducted in accordance with the Declaration of Helsinki. The study protocol was approved by the Institutional Review Board of Changwon National University (No. 7001066-202506-HR-037), and written informed consent was obtained from all participants before enrollment.

Ambient air pollution and meteorological factors

Exposure to ambient air pollutants was estimated using a high-resolution database derived from the Community Multiscale Air Quality model. The model outputs were assimilated with

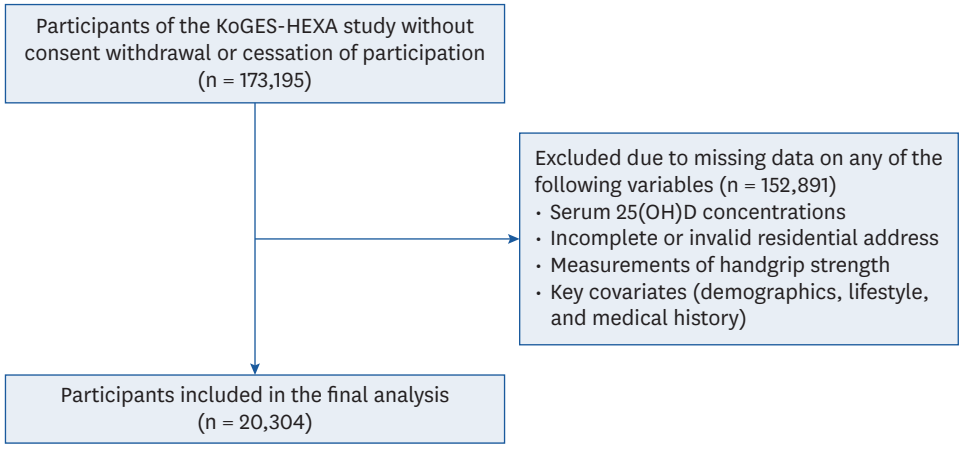

Fig. 1. Flowchart of the study participant selection.
KoGES-HEXA, Korean Genome and Epidemiology Study-Health Examinees.

https://doi.org/10.4162/nrp.2026.20.3.504

Nutrition Research and Practice

ground-level observations and aerosol optical depth (AOD) data obtained from the National Aeronautics and Space Administration Terra and Aqua satellites to ensure high spatial accuracy. Detailed methodological procedures and validation metrics are described elsewhere [23-25]. Data on PM_{10}, $PM_{2.5}$, and NO_2 were used in the present analysis. The integration of satellite-derived AOD data allowed for the refinement of particulate matter estimates to a high-resolution 1 km grid, effectively capturing the spatial variability attributable to aerosol attenuation of solar radiation. In contrast, gaseous pollutants, including NO_2, and meteorological factors such as daily average temperature (°C) and relative humidity (%), were estimated at a 9 km grid resolution. The reliability of the modeled data was validated against ground measurements, yielding root-mean-square errors of 15.16 μg/m^3 for PM_{10}, 8.31 μg/m^3 for $PM_{2.5}$, and 6.9 ppb for NO_2, confirming robust prediction accuracy [24,25]. Individual exposure was assessed by geocoding the residential addresses of participants and spatially matching them to the corresponding air quality grids using ArcGIS software (ESRI, Inc., Redlands, CA, USA). The daily concentrations of each pollutant for the 365 days preceding the baseline examination of each participant were calculated. These daily values were then averaged to determine the annual mean concentration, which served as the primary exposure metric for this study. The 1-yr average was specifically selected as an indicator of chronic exposure because it effectively accounts for seasonal variations in air pollutant levels and reflects stable, long-term environmental conditions. Such long-term exposure metrics are biologically more relevant to the gradual and cumulative pathogenesis of age-related muscle strength decline. Nevertheless, these residential address-based estimates may not fully reflect the actual individual-level exposures. Specifically, this approach does not account for individual daily mobility, such as commuting to workplaces and time spent on outdoor activities, nor does it consider the variations in indoor microenvironments, including indoor air quality and the use of air purifiers. Furthermore, the aforementioned differences in spatial resolution between PM (1 km) and NO_2 (9 km), along with these unmeasured behavioral factors, could introduce a certain degree of exposure misclassification.

Laboratory assays for serum vitamin D

Venous blood specimens were collected from all participants after an overnight fast of at least 8 h. Upon collection, samples were centrifuged to isolate serum, which was subsequently preserved at −80°C until analysis. Quantification of the total serum 25(OH)D concentrations was performed using liquid chromatography-tandem mass spectrometry [26]. The analysis was conducted between 2019 and 2020 at Green Cross Laboratories (Yongin, Korea), using an Acquity UPLC Xevo-TQ system (Waters, Milford, MA, USA). Rigorous quality control (QC) procedures were implemented to ensure analytical validity. Internal QC samples with known concentrations were assayed at regular intervals to monitor method performance. The assay precision was high, with the average coefficients of variation maintained below 5.0%. Based on the measured serum 25(OH)D levels, the vitamin D status was classified as a deficiency (< 20 ng/mL) or normal (≥ 20 ng/mL).

Assessment of possible sarcopenia

Possible sarcopenia was assessed according to the diagnostic algorithm established by the Asian Working Group for Sarcopenia (AWGS) 2019 consensus [2]. Considering that low muscle strength is a primary indicator of sarcopenia, this study evaluated the handgrip strength using a digital hand dynamometer (Lavisen KS-301; Lavisen Co. Ltd., Namyangju, Korea) [27]. Standardized measurements in accordance with the standardized KoGES protocol were ensured by instructing the participants to maintain an upright standing posture with their feet placed shoulder-width apart and arms naturally extended. While

https://doi.org/10.4162/nrp.2026.20.3.504

Nutrition Research and Practice

seated measurements are frequently used to isolate the upper limb strength, the standing position is used widely in epidemiological studies because it captures core stability and lower-body strength alongside localized upper extremity strength, serving as a robust proxy for whole-body muscular fitness [28,29]. They were asked to squeeze the dynamometer with maximum isometric effort. The measurements were obtained twice for each hand in an alternating sequence. Because an individual's true maximum grip strength frequently occurs in the non-dominant hand [30], relying solely on the dominant hand may lead to a systematic underestimation of maximal voluntary contraction. Therefore, following the recommendations of the AWGS, the absolute highest value among the 4 trials, regardless of hand dominance, was recorded as the final handgrip strength (kg) for analysis. Consistent with the AWGS 2019 criteria, possible sarcopenia was identified based on sex-specific cut-off points for low muscle strength. Specifically, men with a handgrip strength of < 28 kg and women with < 18 kg were classified as having possible sarcopenia. Note that possible sarcopenia was defined solely based on low handgrip strength because data on appendicular skeletal muscle mass were unavailable in the baseline cohort. Consequently, this operational definition reflects muscle weakness rather than a definitively diagnosed clinical condition.

Covariates

Information on the demographic characteristics, lifestyle behaviors, and medical history was collected through structured, interviewer-administered questionnaires. The sociodemographic variables included age, sex, educational attainment (less than high school, high school graduate, and college or higher), marital status (married or cohabiting vs. other), and occupation (office, sales, or service; manual labor or agriculture; unemployed or homemaker). The residential area was categorized according to the administrative classification as metropolitan (special cities and metropolitan cities) or non-metropolitan regions. Anthropometric measurements, including height (cm) and weight (kg), were obtained using standardized protocols, and body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). The lifestyle factors assessed included the smoking status (never smoker, former smoker, and current smoker), alcohol consumption (non-drinker or current drinker), and regular physical activity, defined as engagement in exercise inducing perspiration at least once per week. To account for potential seasonal variations, the season of blood collection was categorized as spring (March–May), summer (June–August), fall (September–November), or winter (December–February). Comorbid conditions, including hypertension, diabetes mellitus, cardio-cerebrovascular disease, cancer, and osteoporosis, were defined based on self-reported physician diagnoses and were considered potential confounding variables in the analyses. Information on comorbidities was obtained through self-reported questionnaires, which inherently carry a potential risk of recall bias. Furthermore, specific behavioral factors influencing vitamin D status, such as direct sun exposure, sunscreen use, and detailed dietary vitamin D intake, could not be incorporated into the multivariable adjustment models due to data limitations within the baseline cohort.

Statistical analyses

The baseline characteristics of the study population were compared according to the vitamin D status. Continuous variables were expressed as means ± SDs and analyzed using Student's *t*-tests. In contrast, the categorical variables are presented as frequencies and percentages (n, %) and compared using χ^2 tests. Multivariable linear regression models were used to investigate the association between the annual average air pollution exposure and serum 25(OH)D concentrations. In addition, multivariable logistic regression analysis was conducted to estimate the odds ratios (ORs) and 95% confidence intervals (CIs) for

https://doi.org/10.4162/nrp.2026.20.3.504

the risk of possible sarcopenia associated with air pollution exposure. Two statistical models were constructed to control for potential confounders. Model I was adjusted for age and sex. Model II was further adjusted for BMI, residential area, education level, marital status, occupation, smoking status, alcohol consumption, physical activity, season of blood collection, comorbidities, and meteorological factors (annual mean temperature and relative humidity). For ambient air pollutants, the primary effect estimates were calculated per standard increments (10 μg/m^3 for $PM_{2.5}$ and PM_{10}, and 1 ppb for NO_2) to facilitate a comparison with the existing literature. Furthermore, the effect estimates were also calculated per interquartile range (IQR) increment for each pollutant to reflect realistic exposure variations within the study population, as presented in **Supplementary Table 1**. Stratified analyses were performed based on the presence of vitamin D deficiency to determine if the association between air pollution and possible sarcopenia was modified by the vitamin D status. The statistical significance of this modification was evaluated by including a multiplicative interaction term in the regression models (*P* for interaction). All statistical analyses were performed using SAS software, version 9.4 (SAS Institute Inc., Cary, NC, USA), and a 2-tailed *P*-value of < 0.05 was considered significant.

RESULTS

Baseline characteristics of the study participants according to vitamin D status

Among the 20,304 individuals included in this study, 65.0% were identified as having a vitamin D deficiency, while the remaining 35.0% maintained normal levels (**Table 1**). A significant disparity in serum 25(OH)D was observed; the deficiency group had an average concentration of 13.7 ± 3.7 ng/mL, whereas the normal group had a concentration of 26.3 ± 6.1 ng/mL ($P < 0.001$). These findings showed that subjects with insufficient vitamin D resided in areas with statistically higher, albeit modestly different, annual mean concentrations of all measured air pollutants. Specifically, although the absolute differences were small, the mean exposure levels for $PM_{2.5}$, PM_{10}, and NO_2 were significantly higher in the deficiency cohort compared to their counterparts with adequate vitamin D status (25.1 ± 2.9 vs. 24.9 ± 2.9 μg/m^3 for $PM_{2.5}$, 49.7 ± 5.6 vs. 48.7 ± 5.7 μg/m^3 for PM_{10}, and 0.024 ± 0.008 vs. 0.022 ± 0.008 ppm for NO_2; all $P < 0.001$). Demographically, the deficiency group had a younger mean age (53.1 ± 8.0 yrs) than the normal group (55.8 ± 7.6 yrs) and a higher proportion of female participants ($P < 0.001$). The socioeconomic characteristics also differed significantly between the groups. The participants in the deficiency group were more likely to reside in metropolitan areas ($P < 0.001$) and possess a higher level of education than the normal group ($P = 0.003$). Regarding occupation, the deficiency group had a higher proportion of office, sales, or service workers, whereas the normal group included a larger percentage of laborers or agricultural workers ($P < 0.001$). Regarding the marital status, the proportion of participants who were married or cohabiting was slightly lower in the deficiency group than in the normal group ($P = 0.002$). Seasonal variations in blood sampling were also observed. The proportion of samples collected during winter and spring was higher in the deficiency group, whereas summer and autumn collections were more frequent in the normal group ($P < 0.001$). Regarding clinical history, the normal group exhibited a significantly higher prevalence of hypertension, cardio-cerebrovascular diseases, cancer, and osteoporosis (all $P < 0.01$). No significant difference in the prevalence of diabetes mellitus was observed between the 2 groups. In terms of lifestyle behaviors, the normal group reported higher rates of multivitamin supplementation, regular physical exercise, non-smoking, and current alcohol consumption compared to the deficiency group (all $P < 0.001$),

https://doi.org/10.4162/nrp.2026.20.3.504

Table 1. Baseline characteristics of the study participants according to their vitamin D status

Variables	Total	Normal (25[OH]D level ≥ 20 ng/mL)	Vitamin D deficiency (25[OH]D level < 20 ng/mL)	*P*-value
No.	20,304 (100.0)	7,126 (35.0)	13,188 (65.0)	
Vitamin D levels				
Serum 25(OH)D levels (ng/mL)	18.1 ± 7.6	26.3 ± 6.1	13.7 ± 3.7	< 0.001
Air pollution				
1-yr average $PM_{2.5}$ (μg/m^3)	25.1 ± 2.9	24.9 ± 2.9	25.1 ± 2.9	< 0.001
1-yr average PM_{10} (μg/m^3)	49.4 ± 5.6	48.7 ± 5.7	49.7 ± 5.6	< 0.001
1-yr average NO_2 (ppm)	0.023 ± 0.008	0.022 ± 0.008	0.024 ± 0.008	< 0.001
Demographics				
Age (yrs)	54.1 ± 8.0	55.8 ± 7.6	53.1 ± 8.0	< 0.001
< 65	18,122 (89.3)	6,104 (85.8)	12,018 (91.1)	< 0.001
≥ 65	2,182 (10.7)	1,012 (14.2)	1,170 (8.9)	
Sex				
Male	6,640 (32.7)	3,072 (43.2)	3,568 (27.0)	< 0.001
Female	13,664 (67.3)	4,044 (56.8)	9,620 (73.0)	
Season of blood draw				
Spring (Mar–May)	4,000 (19.7)	838 (11.8)	3,162 (24.0)	< 0.001
Summer (Jun–Aug)	6,327 (31.2)	2,750 (38.7)	3,577 (27.1)	
Fall (Sep–Nov)	6,437 (31.7)	2,833 (39.8)	3,604 (27.3)	
Winter (Dec–Feb)	3,540 (17.4)	695 (9.8)	2,845 (21.6)	
History of diabetes mellitus				
Yes	2,218 (10.9)	817 (11.5)	1,401 (10.6)	0.06
No	18,086 (89.1)	6,299 (88.5)	11,787 (89.4)	
History of hypertension				
Yes	5,943 (29.3)	2,273 (31.9)	3,670 (27.8)	< 0.001
No	14,361 (95.7)	4,843 (68.1)	9,518 (72.2)	
History of cardio-cerebrovascular disease				
Yes	867 (4.3)	343 (4.8)	524 (4.0)	0.005
No	19,437 (95.7)	6,773 (95.2)	12,664 (96.0)	
History of cancer				
Yes	876 (4.3)	375 (5.3)	501 (3.8)	< 0.001
No	19,428 (95.7)	6,741 (94.7)	12,687 (96.2)	
History of osteoporosis				
Yes	1,177 (5.8)	551 (7.7)	626 (4.7)	< 0.001
No	19,127 (94.2)	6,565 (92.3)	12,562 (95.3)	
Occupation				
Professional or administrative	2,627 (12.9)	893 (12.5)	1,734 (13.1)	< 0.001
Office, sales, or service	5,137 (25.3)	1,612 (22.6)	3,525 (26.7)	
Laborer or agricultural	2,715 (13.4)	1,190 (16.7)	1,525 (11.6)	
Others or unemployed	9,825 (48.4)	3,421 (48.1)	6,404 (48.6)	
Marital status				
Married or cohabiting	18,332 (90.3)	6,489 (91.2)	11,843 (89.8)	0.002
Single, divorced, widowed, or others	1,972 (9.7)	627 (8.8)	1,345 (10.2)	
Education				
Less than middle school	2,777 (13.7)	1,016 (14.3)	1,761 (13.4)	0.003
High school	10,836 (53.4)	3,815 (53.6)	7,021 (53.2)	
College or more	6,630 (32.7)	2,275 (32.0)	4,355 (33.0)	
Metropolitan residence				
Yes	13,351 (65.8)	4,447 (62.5)	8,904 (67.5)	< 0.001
No	6,953 (34.2)	2,669 (37.5)	4,284 (32.5)	
Lifestyle factors				
BMI (kg/m^2)	23.9 ± 2.9	23.9 ± 2.8	23.8 ± 3.0	0.15
Obese (≥ 25 kg/m^2)	6,512 (32.1)	2,322 (32.6)	4,190 (31.8)	0.22
Non-obese (< 25 kg/m^2)	13,792 (67.9)	4,794 (67.4)	8,997 (68.2)	
Multivitamin use				
Yes	4,290 (21.1)	2,083 (29.3)	2,207 (16.7)	< 0.001
No	16,014 (78.9)	5,033 (70.7)	10,981 (83.3)	
Regular exercise				
Yes	11,454 (56.4)	4,374 (61.5)	7,080 (53.7)	< 0.001
No	8,850 (43.6)	2,742 (38.5)	6,108 (46.3)	

(continued to the next page)

Table 1. (Continued) Baseline characteristics of the study participants according to their vitamin D status

Variables	Total	Normal (25[OH]D level ≥ 20 ng/mL)	Vitamin D deficiency (25[OH]D level < 20 ng/mL)	*P*-value
Smoking				
Non-smoker	15,021 (74.0)	4,730 (66.5)	10,291 (78.0)	< 0.001
Ex-smoker	3,316 (16.3)	1,587 (22.3)	1,729 (13.1)	
Current smoker	1,967 (9.7)	799 (11.2)	1,168 (8.9)	
Alcohol consumption				
Non-drinker	10,500 (51.7)	3,329 (46.8)	7,171 (54.4)	< 0.001
Ex-drinker	786 (3.9)	326 (4.6)	460 (3.5)	
Current drinker	9,018 (44.4)	3,461 (48.6)	5,557 (42.1)	
Possible sarcopenia				
Handgrip strength (kg)	28.2 ± 9.6	29.7 ± 10.2	27.4 ± 9.2	< 0.001
Low (< 28 kg for men, < 18 kg for women)	2,359 (11.6)	831 (11.7)	1,528 (11.6)	0.86
Normal (≥ 28 kg for men, ≥ 18 kg for women)	17,945 (88.4)	6,285 (88.3)	11,660 (88.4)	

Values are presented as the mean ± SD for continuous variables and number (%) for categorical variables.
The vitamin D status was defined based on serum 25(OH)D concentration (normal: ≥ 20 ng/mL; deficiency: < 20 ng/mL). Possible sarcopenia was defined according to the Asian Working Group for Sarcopenia 2019/2023 criteria as low handgrip strength (< 28 kg for men and < 18 kg for women). The differences between groups were assessed using a Student's *t*-test for the continuous variables and a χ^2 test for the categorical variables. Annual average concentrations of air pollutants were calculated based on 1-yr average exposure prior to blood sampling.
25(OH)D, 25-hydroxyvitamin D; PM, particulate matter; NO_2, nitrogen dioxide; BMI, body mass index.

while the BMI and obesity rates did not differ significantly. Regarding the physical function, the mean handgrip strength was significantly lower in the deficiency group (27.4 ± 9.2 kg) than in the normal group (29.7 ± 10.2 kg) ($P < 0.001$). On the other hand, the prevalence of possible sarcopenia, defined as low handgrip strength, was comparable between the deficiency (11.6%) and normal groups (11.7%).

Associations between annual average air pollution exposure and serum vitamin D levels

Table 2 lists the associations between annual average exposure to air pollutants and serum 25(OH)D concentrations. For linear regression analyses, the serum 25(OH) D levels were natural log-transformed to achieve a normal distribution. Therefore, the presented β coefficients represent a proportional shift; specifically, an exponentiated coefficient [exp(β) - 1] × 100 approximates the percentage change in geometric mean 25(OH) D concentrations per specified unit increase in each air pollutant (i.e., per 10 μg/m³ for PM and 1 ppb for NO_2). In model I, higher exposures to $PM_{2.5}$ and PM_{10} were significantly associated with lower serum vitamin D levels. Specifically, a 10 μg/m³ increase in $PM_{2.5}$ was associated with a decrease in the serum 25(OH)D concentration (log-transformed β = −4.95; 95% CI, −6.77 to −3.10), while a corresponding increase in PM_{10} was associated with a β coefficient of −8.17 (95% CI, −9.07 to −7.26) (both $P < 0.001$). These inverse associations

Table 2. Associations between the annual average air pollution exposure and serum vitamin D levels

Air pollution	Model I		Model II	
	β (95% CI)	*P*-value	β (95% CI)	*P*-value
$PM_{2.5}$	−4.95 (−6.77, −3.10)	< 0.001	−4.93 (−6.77, −3.04)	< 0.001
PM_{10}	−8.17 (−9.07, −7.26)	< 0.001	−3.89 (−5.06, −2.71)	< 0.001
NO_2	−0.49 (−0.56, 0.42)	0.18	−0.25 (−0.34, −0.16)	< 0.001

The values are presented as log-transformed β coefficients (95% CIs) for the association between annual average air pollutant exposure and serum vitamin D concentration. The β coefficients represent the estimated difference in natural log-transformed serum 25-hydroxyvitamin D levels. To interpret these values as a percentage change on the original scale, the formula [exp(β) − 1] × 100 can be applied. The effect estimates are scaled per 10 μg/m³ for PM and 1 ppb (0.001 ppm) for NO_2 to maintain comparability with previous large-scale epidemiological studies. Model I: adjusted for age and sex. Model II: adjusted for age, sex, season, metropolitan area status, comorbidities (diabetes mellitus, hypertension, cardio-cerebrovascular disease, cancer, and osteoporosis), occupation, marital status, educational level, obesity status, vitamin or dietary supplement use, physical activity, smoking status, alcohol consumption, and annual average ambient temperature and relative humidity.
CI, confidence interval; PM, particulate matter; NO_2, nitrogen dioxide.

https://doi.org/10.4162/nrp.2026.20.3.504

remained robust after further adjustments for potential confounding factors in model II. In the fully adjusted model, each 10 μg/m^3 increase in $PM_{2.5}$ and PM_{10} was associated with decreases in the serum vitamin D levels of −4.93 (95% CI, −6.77 to −3.04) and −3.89 (95% CI, −5.06 to −2.71), respectively (both $P < 0.001$). In contrast, exposure to NO_2 was not significantly associated with serum 25(OH)D concentrations in model I, but in model II, a 1 ppb increase in NO_2 exposure was significantly associated with lower serum vitamin D levels (β = −0.25; 95% CI, −0.34 to −0.16; $P < 0.001$).

Associations between annual average air pollutant exposure and low grip strength

Table 3 lists the associations between annual average exposure to air pollutants and low grip strength, indicative of possible sarcopenia. In model I, increased exposures to PM_{10} and NO_2 were significantly associated with higher odds of low grip strength ($P < 0.001$), whereas no significant association was observed for $PM_{2.5}$. After further adjustment for potential confounders in model II, $PM_{2.5}$ exposure showed a modest but significant positive association with the likelihood of low grip strength. Specifically, a 10 μg/m^3 increase in $PM_{2.5}$ was associated with a 22% higher odds of low grip strength (OR, 1.22; 95% CI, 1.03 to 1.45; $P = 0.020$). Similarly, PM_{10} exposure showed a significant positive association with a low grip strength (OR, 1.37; 95% CI, 1.23 to 1.52; $P < 0.001$). For NO_2, consistent associations with low grip strength were observed across both models for each 1 ppb increase (OR, 1.04; 95% CI, 1.03 to 1.05; $P < 0.001$). In addition, a significant interaction with the vitamin D status was uniquely detected for the association between $PM_{2.5}$ exposure and low grip strength (P for interaction = 0.005), whereas no such significant interactions were observed for PM_{10} or NO_2.

Associations between air pollution exposure and sarcopenia stratified by vitamin D status

Table 4 lists the associations between air pollution exposure and possible sarcopenia, stratified according to the vitamin D status. Among the participants with normal vitamin D levels, there were 7,126 individuals, including 831 cases of possible sarcopenia, whereas among those with vitamin D deficiency, there were 13,188 individuals, including 1,528 cases. In the population with normal vitamin D levels, no significant correlation was observed between $PM_{2.5}$ exposure and possible sarcopenia (OR, 0.95; 95% CI, 0.71 to 1.28). Conversely, among participants with a vitamin D deficiency, each 10 μg/m^3 increment in $PM_{2.5}$ was significantly linked to an elevated risk of possible sarcopenia (OR, 1.39; 95% CI, 1.12 to 1.71; $P = 0.002$). Although there was partial overlap in the 95% CIs between the 2 strata (1.12 to 1.28), the formal multiplicative interaction remained statistically significant. In contrast to $PM_{2.5}$, exposure to PM_{10} and NO_2 had significant positive associations with possible sarcopenia in both subgroups, irrespective of the vitamin D levels. Specifically, the ORs for

Table 3. Associations between the annual average air pollutant exposure and possible sarcopenia

Air pollution	Model I		Model II		*P*-value for interaction
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value	
$PM_{2.5}$	1.11 (0.96, 1.30)	0.17	1.22 (1.03, 1.45)	0.020	0.005
PM_{10}	1.39 (1.29, 1.50)	< 0.001	1.37 (1.23, 1.52)	< 0.001	0.41
NO_2	1.04 (1.03, 1.05)	< 0.001	1.04 (1.03, 1.05)	< 0.001	0.71

The values are presented as ORs and 95% CIs for low grip strength in relation to annual average air pollutant exposure. Effect estimates are scaled per 10 μg/m^3 for $PM_{2.5}$ and PM_{10}, and per 1 ppb (0.001 ppm) for NO_2 to maintain comparability with previous large-scale epidemiological studies. ORs indicate the relative odds of possible sarcopenia associated with an increase in air pollutant exposure. Model I: adjusted for age and sex. Model II: adjusted for age, sex, season, metropolitan area status, comorbidities (diabetes mellitus, hypertension, cardio-cerebrovascular disease, cancer, and osteoporosis), occupation, marital status, educational level, obesity status, vitamin or dietary supplement use, physical activity, smoking status, alcohol consumption, and annual average ambient temperature and relative humidity.
OR, odds ratio; CI, confidence interval; PM, particulate matter; NO_2, nitrogen dioxide.

https://doi.org/10.4162/nrp.2026.20.3.504

Table 4. Associations between air pollution exposure and possible sarcopenia stratified by vitamin D status

Air pollution	Normal (25[OH]D level ≥ 20 ng/mL)		Vitamin D deficiency (25[OH]D level < 20 ng/mL)	
	OR (95% CI)	*P*-value	OR (95% CI)	*P*-value
No. of participants/events	7,126/831		13,188/1,528	
$PM_{2.5}$	0.95 (0.71, 1.28)	0.74	1.39 (1.12, 1.71)	0.002
PM_{10}	1.37 (1.14, 1.64)	< 0.001	1.36 (1.19, 1.56)	< 0.001
NO_2	1.03 (1.02, 1.05)	< 0.001	1.04 (1.03, 1.05)	0.001

The values are presented as ORs and 95% CIs for sarcopenia associated with annual average air pollutant exposure, stratified by vitamin D status. The vitamin D status was defined based on the serum 25(OH)D concentration (normal: ≥ 20 ng/mL; deficiency: < 20 ng/mL). The effect estimates were scaled per 10 μg/m^3 for $PM_{2.5}$ and PM_{10}, and per 1 ppb (0.001 ppm) for NO_2 to maintain comparability with previous large-scale epidemiological studies. ORs indicate the relative odds of sarcopenia associated with an increase in air pollutant exposure within each vitamin D subgroup. All models were adjusted for age, sex, season of blood draw, metropolitan area status, comorbidities (diabetes mellitus, hypertension, cardio-cerebrovascular disease, cancer, and osteoporosis), occupation, marital status, educational level, obesity status, vitamin or dietary supplement use, physical activity, smoking status, alcohol consumption, and annual average ambient temperature and relative humidity.
25(OH)D, 25-hydroxyvitamin D; OR, odds ratio; CI, confidence interval; PM, particulate matter; NO_2, nitrogen dioxide.

PM_{10} were comparable between the normal group (OR, 1.37; 95% CI, 1.14 to 1.64) and the deficiency group (OR, 1.36; 95% CI, 1.19 to 1.56) (both $P < 0.001$). Similarly, NO_2 exposure exhibited a consistent adverse relationship with possible sarcopenia across both strata; the ORs were 1.03 (95% CI, 1.02 to 1.05; $P < 0.001$) for the normal group and 1.04 (95% CI, 1.03 to 1.05; $P = 0.001$) for the deficiency group.

DISCUSSION

This large-scale study using data from the Korean HEXA cohort investigated the complex interrelationships among air pollution exposure, vitamin D status, and the risk of possible sarcopenia. A high prevalence of vitamin D deficiency (65.0%) was observed in this population, which was significantly correlated with elevated exposure to ambient air pollutants. These results revealed a clear inverse association between the annual average concentrations of air pollutants ($PM_{2.5}$, PM_{10}, and NO_2) and the serum 25(OH)D levels. Furthermore, air pollution exposure was independently associated with an increased risk of possible sarcopenia. A notable finding was the significant interaction between vitamin D deficiency and $PM_{2.5}$ exposure (P for interaction = 0.005). Specifically, an adverse association between $PM_{2.5}$ and grip strength was observed in individuals with a vitamin D deficiency, whereas this association was not significant in those with sufficient vitamin D levels. These findings suggest that a vitamin D deficiency may exacerbate the susceptibility to $PM_{2.5}$-associated low grip strength, highlighting the potential protective role of adequate vitamin D status against specific particulate matter exposure.

The magnitude and significance of the associations for $PM_{2.5}$ and NO_2 shifted considerably between the unadjusted and fully adjusted models. This fluctuation strongly indicates the presence of negative confounding by sociodemographic and environmental factors. For example, high NO_2 and $PM_{2.5}$ concentrations are typically clustered in highly urbanized metropolitan areas. Urban residents often engage in different occupational patterns, such as predominantly indoor non-manual work, and may exhibit different seasonal behaviors or possess better access to healthcare infrastructure. These urban-associated lifestyle factors could inherently protect against muscle strength decline, initially masking the harmful effects of the pollutants. Consequently, adjusting for covariates such as residential area, occupation, and seasons was crucial to unmasking the true, independent deleterious effects of these pollutants on muscle health.

https://doi.org/10.4162/nrp.2026.20.3.504

Nutrition Research and Practice

The strong inverse association between air pollution and serum vitamin D suggests that air pollutants may directly contribute to muscle strength decline, a hallmark of sarcopenia, and indirectly increase the risk of muscle dysfunction by depleting vitamin D, a key protective factor for muscle health [6,31]. The serum 25(OH)D levels were inversely associated with incremental increases in $PM_{2.5}$, PM_{10}, and NO_2, which aligns with the hypothesized UVB-blocking effect whereby airborne particulates and nitrogen oxides may attenuate cutaneous vitamin D synthesis by reducing UVB radiation at the skin surface [32,33]. This UVB attenuation is particularly pronounced in heavily polluted environments, where an atmospheric curtain of pollutants can markedly lower the erythemal UV dose and may be especially detrimental for vulnerable groups [33]. In older adults, whose 7-dehydrocholesterol levels are already reduced, such additional UVB blocking is likely to entrench chronic vitamin D deficiency and compromise musculoskeletal health [34]. In parallel, behavioral and seasonal factors amplify these effects because individuals in polluted urban areas tend to limit outdoor activity, particularly during seasons with low solar radiation, while predominantly indoor lifestyles are consistently associated with higher vitamin D insufficiency, even in sunny regions [35,36]. Collectively, these findings suggest that air pollution interacts with the lifestyle and seasonal determinants of sun exposure, potentially increasing the risk of chronic vitamin D deficiency and its downstream consequences for muscle function [6,35].

The key finding of this study, namely the interaction between $PM_{2.5}$ exposure and vitamin D deficiency, is likely to reflect shared or interrelated biological pathways that jointly contribute to sarcopenia [37]. In particular, these 2 factors may influence common molecular targets or engage in bidirectional cross-talk within endocrine, inflammatory, and oxidative stress pathways that are relevant to muscle homeostasis [38,39]. The available data cannot definitively establish the underlying mechanisms because this epidemiological study did not measure specific inflammatory markers or biological intermediates. The following hypotheses regarding shared biological pathways are proposed to guide future experimental studies. The first proposed mechanism involves a loss of endogenous defense against oxidative stress and systemic inflammation. $PM_{2.5}$ exposure provokes systemic inflammation and oxidative stress, which can promote muscle protein breakdown [40-43]. Vitamin D normally functions as an anti-inflammatory buffering system [44,45]. Therefore, its deficiency may compromise this immunomodulatory capacity, potentially leaving the body more vulnerable to $PM_{2.5}$-induced inflammatory muscle damage [46]. The second proposed mechanism is a double hit on the balance of muscle protein turnover [47]. $PM_{2.5}$ exposure is primarily associated with accelerated catabolic protein degradation [48,49], whereas a vitamin D deficiency is linked to impaired anabolic protein synthesis [50,51]. When these 2 detrimental effects occur concurrently, the net loss of muscle mass may increase disproportionately, which could, in theory, account for the heightened risk of sarcopenia observed in this study [49,51]. These speculative mechanisms warrant confirmation in future experimental and longitudinal research.

The significant interactions observed specifically with $PM_{2.5}$, rather than PM_{10} or NO_2, warrant discussion. This finding might stem from differences in aerodynamic diameter and bioavailability [52]. $PM_{2.5}$ particles, with diameters ≤ 2.5 μm, can penetrate deep into the alveoli and cross the blood-air barrier more readily to enter systemic circulation, unlike larger PM_{10} particles that primarily affect the upper airways or localized lung regions [39]. Consequently, $PM_{2.5}$ can deliver oxidative stress directly to the peripheral tissues, including skeletal muscle, beyond mere respiratory inflammation [39,53]. In contrast to the predominantly local effects of PM_{10}, the potential systemic toxicity of $PM_{2.5}$ might heighten

https://doi.org/10.4162/nrp.2026.20.3.504

the demand for protective hormonal mechanisms such as those mediated by vitamin D. Therefore, the vitamin D status could serve as a potential effect modifier, whereby a deficiency may increase the risk of sarcopenia-related muscle weakness under exposure to highly penetrative toxicants like $PM_{2.5}$ [54,55]. Methodologically, the discrepancy in spatial modeling resolutions could also contribute to this differential interaction. The NO_2 estimates might be subject to greater non-differential exposure misclassification, which frequently attenuates the statistical signals of complex interaction terms, because NO_2 was estimated at a coarser 9 km resolution than the 1 km resolution for $PM_{2.5}$. This study highlights the specific interactive effect of $PM_{2.5}$ and vitamin D deficiency on sarcopenia-related muscle weakness, emphasizing its potential public health relevance in aging populations.

This study highlights the unique role of $PM_{2.5}$ in aggravating sarcopenia-related muscle weakness, a key functional component of sarcopenia, particularly when a vitamin D deficiency is present, emphasizing its public health relevance in aging populations. Although these public health implications are drawn from observational associations and do not imply direct causal intervention effects, they indicate that future approaches to sarcopenia prevention might benefit from moving beyond individual lifestyle approaches to incorporate environment–nutrition interactions, recognizing air quality as a potential determinant of muscle health and viewing a vitamin D deficiency in highly polluted areas as an indicator of heightened vulnerability rather than simple lack of nutrient lack [56,57]. Consequently, future longitudinal and intervention studies are needed to determine if routine vitamin D screening and supplementation would be beneficial for older adults in high-pollution regions.
In addition, further research should investigate whether conventional advice promoting outdoor exercise for musculoskeletal health needs to be refined so that, during periods of poor air quality, indoor physical activity is encouraged and vitamin D is secured through diet or supplements using environment-adapted behavioral guidance [58,59].

In addition to these public health implications, several methodological considerations should be acknowledged when interpreting these findings. This study also had notable strengths. It was based on a large sample of more than 20,000 Korean adults and used objectively measured serum 25(OH)D concentrations and handgrip strength, thereby reducing the likelihood of self-report bias and ensuring adequate statistical power. Furthermore, this study simultaneously evaluated multiple air pollutants ($PM_{2.5}$, PM_{10}, and NO_2). It formally tested the interaction terms, thereby providing one of the first empirical assessments of the combined effects of vitamin D deficiency and specific $PM_{2.5}$ exposure on the sarcopenia risk within a single analytic framework. This study had several limitations that should be considered when interpreting the findings. First, the analysis was based on baseline data with a cross-sectional design, which prevents establishing a temporal sequence between air pollution, vitamin D status, and sarcopenia and leaves room for reverse causation.

Second, regarding the assessment of air pollution exposure, the derived estimates may not fully capture the true individual-level exposure because the analysis relied on the annual average outdoor concentrations at the participants' residential addresses. This residential-based approach does not account for individual daily mobility, such as commuting to workplaces and time spent outdoors, or variations in indoor microenvironments, including indoor air quality and the use of air purifiers. Furthermore, the discrepancy in spatial resolution, with PM estimated at a 1 km resolution and NO_2 at a coarser 9 km resolution, could introduce varying degrees of non-differential exposure misclassification, potentially attenuating the observed associations.

https://doi.org/10.4162/nrp.2026.20.3.504

Third, although the AWGS 2019 criteria were used for possible sarcopenia based on the handgrip strength, data on appendicular skeletal muscle mass were lacking. Consequently, a diagnosis of sarcopenia could not be confirmed. Nevertheless, a low grip strength is a well-established proxy for overall muscle quality and a robust predictor of adverse health outcomes, justifying its use as a primary endpoint. In addition, a discrepancy was observed wherein the mean handgrip strength differed significantly between the groups, but the prevalence of possible sarcopenia did not. This inconsistency highlights a methodological caveat associated with dichotomizing continuous variables based on clinical cut-offs. Converting a continuous measure, such as the handgrip strength, into a binary outcome inevitably results in a loss of statistical information and power. Consequently, subtle but meaningful shifts in the overall distribution of muscle strength may be obscured when relying solely on the categorical prevalence rates.

Fourth, despite adjusting for multiple covariates, residual confounding is possible because factors, such as detailed diet, sun-avoidant behaviors, sunscreen use, skin characteristics, socioeconomic disadvantage, and comorbidities, could not be fully accounted for. Furthermore, variations in specific exercise intensity and exact outdoor activity durations might not have been perfectly controlled because physical activity was adjusted as a broad dichotomous variable due to a high rate of missing values in detailed questionnaire responses.

Fifth, the serum 25(OH)D was measured only once, limiting assessment of long-term vitamin D status and seasonal dynamics, potentially leading to misclassification of chronic deficiency. Therefore, future longitudinal studies incorporating repeated measurements are warranted to reflect the long-term vitamin D status more accurately.

Sixth, the influence of the substantial sample size should be considered when interpreting the baseline characteristics. Even minute and practically negligible absolute differences between the groups, such as the small variations in ambient air pollution concentrations, resulted in highly significant *P*-values because of the high statistical power. Therefore, the statistical significance in these baseline comparisons does not necessarily imply clinical or environmental relevance. Furthermore, the paradoxically higher prevalence of certain chronic diseases observed in the vitamin D sufficient group is likely confounded by age. Older adults inherently carry a higher burden of age-related comorbidities, but they frequently exhibit higher serum 25(OH)D levels, potentially due to the higher rates of dietary supplement use or increased outdoor leisure time after retirement. Thus, these unadjusted baseline differences should be interpreted with caution. Finally, generalizability to other ethnicities and settings is limited because this study included only Korean adults, and confirmation in diverse, preferably prospective, cohorts is needed.

In conclusion, a vitamin D deficiency and exposure to air pollution may be independently associated with possible sarcopenia, defined as low muscle strength. Furthermore, the current findings reveal a specific interactive association between $PM_{2.5}$ exposure and vitamin D deficiency, with an amplified risk. In environments with high $PM_{2.5}$ exposure, a vitamin D deficiency may be associated with vulnerabilities in muscle inflammation control and metabolic homeostasis, potentially contributing to adverse musculoskeletal outcomes. These findings reaffirm that the deterioration of muscle health is not merely a byproduct of aging but a multifactorial process shaped by the interplay of nutritional status and specific environmental exposures. Future prospective studies and clinical trials will be needed to determine if prevention strategies combining nutritional

https://doi.org/10.4162/nrp.2026.20.3.504

interventions to optimize the vitamin D levels with environmental policies aimed at reducing air pollution can effectively preserve muscle strength in older adults.

ACKNOWLEDGMENTS

This study used data from the HEXA study, which was provided as a biomedical and research resource, containing genetic and health information from CODA (Clinical & Omics Data Archive), the Agency for Disease Control and Prevention, Republic of Korea (CODA_S2600011E-01).

SUPPLEMENTARY MATERIAL

Supplementary Table 1

Associations between the annual average air pollutant exposure and possible sarcopenia (estimated per interquartile range increase)

REFERENCES

1. Cruz-Jentoft AJ, Bahat G, Bauer J, Boirie Y, Bruyère O, Cederholm T, Cooper C, Landi F, Rolland Y, Sayer AA, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing 2019;48:16-31. PUBMED | CROSSREF
2. Chen LK, Woo J, Assantachai P, Auyeung TW, Chou MY, Iijima K, Jang HC, Kang L, Kim M, Kim S, et al. Asian working group for sarcopenia: 2019 consensus update on sarcopenia diagnosis and treatment. J Am Med Dir Assoc 2020;21:300-307.e2. PUBMED | CROSSREF
3. Beaudart C, Zaaria M, Pasleau F, Reginster JY, Bruyère O. Health outcomes of sarcopenia: a systematic review and meta-analysis. PLoS One 2017;12:e0169548. PUBMED | CROSSREF
4. Yeung SSY, Reijnierse EM, Pham VK, Trappenburg MC, Lim WK, Meskers CGM, Maier AB. Sarcopenia and its association with falls and fractures in older adults: a systematic review and meta-analysis. J Cachexia Sarcopenia Muscle 2019;10:485-500. PUBMED | CROSSREF
5. Narici MV, Maffulli N. Sarcopenia: characteristics, mechanisms and functional significance. Br Med Bull 2010;95:139-59. PUBMED | CROSSREF
6. Remelli F, Vitali A, Zurlo A, Volpato S. Vitamin D deficiency and sarcopenia in older persons. Nutrients 2019;11:2861. PUBMED | CROSSREF
7. Girgis CM, Mokbel N, Cha KM, Houweling PJ, Abboud M, Fraser DR, Mason RS, Clifton-Bligh RJ, Gunton JE. The vitamin D receptor (VDR) is expressed in skeletal muscle of male mice and modulates 25-hydroxyvitamin D (25OHD) uptake in myofibers. Endocrinology 2014;155:3227-37. PUBMED | CROSSREF
8. Visser M, Deeg DJ, Lips P; Longitudinal Aging Study Amsterdam. Low vitamin D and high parathyroid hormone levels as determinants of loss of muscle strength and muscle mass (sarcopenia): the Longitudinal Aging Study Amsterdam. J Clin Endocrinol Metab 2003;88:5766-72. PUBMED | CROSSREF
9. Houston DK, Cesari M, Ferrucci L, Cherubini A, Maggio D, Bartali B, Johnson MA, Schwartz GG, Kritchevsky SB. Association between vitamin D status and physical performance: the InCHIANTI study. J Gerontol A Biol Sci Med Sci 2007;62:440-6. PUBMED | CROSSREF
10. Kim MK, Baek KH, Song KH, Il Kang M, Park CY, Lee WY, Oh KW. Vitamin D deficiency is associated with sarcopenia in older Koreans, regardless of obesity: the Fourth Korea National Health and Nutrition Examination Surveys (KNHANES IV) 2009. J Clin Endocrinol Metab 2011;96:3250-6. PUBMED | CROSSREF
11. Amrein K, Scherkl M, Hoffmann M, Neuwersch-Sommeregger S, Köstenberger M, Tmava Berisha A, Martucci G, Pilz S, Malle O. Vitamin D deficiency 2.0: an update on the current status worldwide. Eur J Clin Nutr 2020;74:1498-513. PUBMED | CROSSREF
12. Holick MF. The vitamin D deficiency pandemic: approaches for diagnosis, treatment and prevention. Rev Endocr Metab Disord 2017;18:153-65. PUBMED | CROSSREF

13. Lee JJ, Woo HD, Kim JH, Jung EJ, Lee K. Association of sarcopenia, ambient air pollution and cognitive function in a community-dwelling middle-aged and elderly Korean population: a community-based cohort study. BMJ Open 2024;14:e092448. PUBMED | CROSSREF
14. Rom O, Kaisari S, Aizenbud D, Reznick AZ. Lifestyle and sarcopenia-etiology, prevention, and treatment. Rambam Maimonides Med J 2012;3:e0024. PUBMED | CROSSREF
15. Chen CH, Huang LY, Lee KY, Wu CD, Pan SC, Guo YL. Air pollution exposure associated with decline rates in skeletal muscle mass and grip strength and increase rate in body fat in elderly: a 5-year follow-up study. Environ Health Prev Med 2025;30:56. PUBMED | CROSSREF
16. Shi W, Li Y, Zhao JV. Long-term exposure to ambient air pollution with sarcopenia among middle-aged and older adults in China. J Nutr Health Aging 2024;28:100029. PUBMED | CROSSREF
17. Trevisan C, Gregorio C, Welmer AK, Volpato S, Eneroth K, Bellander T, Vetrano DL, Rizzuto D. Air pollution exposure and muscle mass and strength decline in older adults: results from a Swedish population-based study. J Cachexia Sarcopenia Muscle 2025;16:e70111. PUBMED | CROSSREF
18. Liu X, Li Y, Li F, Yang W, Ji W, Chen N, Cui J. Exposure to air pollution, genetic susceptibility, and prevalence of sarcopenia in the UK. Ecotoxicol Environ Saf 2024;285:117143. PUBMED | CROSSREF
19. Manicourt DH, Devogelaer JP. Urban tropospheric ozone increases the prevalence of vitamin D deficiency among Belgian postmenopausal women with outdoor activities during summer. J Clin Endocrinol Metab 2008;93:3893-9. PUBMED | CROSSREF
20. Mousavi SE, Amini H, Heydarpour P, Amini Chermahini F, Godderis L. Air pollution, environmental chemicals, and smoking may trigger vitamin D deficiency: evidence and potential mechanisms. Environ Int 2019;122:67-90. PUBMED | CROSSREF
21. Hosseinpanah F, Pour SH, Heibatollahi M, Moghbel N, Asefzade S, Azizi F. The effects of air pollution on vitamin D status in healthy women: a cross sectional study. BMC Public Health 2010;10:519. PUBMED | CROSSREF
22. Kim Y, Han BG; KoGES group. Cohort profile: the Korean Genome and Epidemiology Study (KoGES) consortium. Int J Epidemiol 2017;46:e20. PUBMED | CROSSREF
23. Woo HD, Song DS, Choi SH, Park JK, Lee K, Yun HY, Choi DR, Koo YS, Park HY. Integrated dataset of the Korean Genome and Epidemiology Study cohort with estimated air pollution data. Epidemiol Health 2022;44:e2022071. PUBMED | CROSSREF
24. Kim JH, Woo HD, Choi S, Song DS, Lee JH, Lee K. Long-term effects of ambient particulate and gaseous pollutants on serum high-sensitivity C-reactive protein levels: a cross-sectional study using KoGES-HEXA data. Int J Environ Res Public Health 2022;19:11585. PUBMED | CROSSREF
25. Kim JH, Woo HD, Lee JJ, Song DS, Lee K. Association between short-term exposure to ambient air pollutants and biomarkers indicative of inflammation and oxidative stress: a cross-sectional study using KoGES-HEXA data. Environ Health Prev Med 2024;29:17. PUBMED | CROSSREF
26. Song S, Woo HD, Lyu J, Song BM, Lim JY, Park HY. Serum 25-hydroxyvitamin D levels and risk of overall and site-specific cancers in Korean adults: results from two prospective cohort studies. Nutr J 2025;24:84. PUBMED | CROSSREF
27. Kim GS, Lee JJ, Park MK, Kim L, Kwon S, Park EJ, Yang H, Yang S. Possible sarcopenia and associated factors in community-dwelling older adults with a history of falls in the Republic of Korea: a cross-sectional study. BMC Geriatr 2025;25:575. PUBMED | CROSSREF
28. Park JH, Lim NK, Park HY. Relative handgrip strength is inversely associated with hypertension in consideration of visceral adipose dysfunction: a nationwide cross-sectional study in Korea. Front Physiol 2022;13:930922. PUBMED | CROSSREF
29. Lawman HG, Troiano RP, Perna FM, Wang CY, Fryar CD, Ogden CL. Associations of relative handgrip strength and cardiovascular disease biomarkers in U.S. adults, 2011–2012. Am J Prev Med 2016;50:677-83. PUBMED | CROSSREF
30. Petersen P, Petrick M, Connor H, Conklin D. Grip strength and hand dominance: challenging the 10% rule. Am J Occup Ther 1989;43:444-7. PUBMED | CROSSREF
31. Golastani B, Poursafa P, Zarean M, Yazdi M, Heidari-Beni M, Kelishadi R. Relationship between air pollution and serum vitamin D levels: a systematic review and meta-analysis. Adv Biomed Res 2024;13:96. PUBMED | CROSSREF
32. Yang C, Li D, Tian Y, Wang P. Ambient air pollutions are associated with vitamin D status. Int J Environ Res Public Health 2021;18:18. PUBMED | CROSSREF
33. Rahman A, Elmi A. Air pollutants are negatively associated with vitamin D-synthesizing UVB radiation intensity on the ground. Sci Rep 2021;11:21480. PUBMED | CROSSREF

34. Zhang F, Li W. Vitamin D and sarcopenia in the senior people: a review of mechanisms and comprehensive prevention and treatment strategies. Ther Clin Risk Manag 2024;20:577-95. PUBMED | CROSSREF

35. Barrea L, Savastano S, Di Somma C, Savanelli MC, Nappi F, Albanese L, Orio F, Colao A. Low serum vitamin D-status, air pollution and obesity: a dangerous liaison. Rev Endocr Metab Disord 2017;18:207-14. PUBMED | CROSSREF

36. Shin S, Kim MJ. Associations between vitamin D deficiency and sarcopenia in South Korean adults: based on the 2022 Korea National Health and Nutrition Examination Survey. Nutrients 2025;17:3292. PUBMED | CROSSREF

37. Hu P, Wei S, Tian F, Yang W, Ai Z, Liu X, Huang Z, Lin H. Association of air pollution with frailty and sarcopenia among middle-aged and older adults: a systematic review and meta-analysis. Atmos Environ 2025;358:121350. CROSSREF

38. Chen CH, Huang LY, Lee KY, Wu CD, Chiang HC, Chen BY, Chin WS, Pan SC, Guo YL. Effects of PM(2.5) on skeletal muscle mass and body fat mass of the elderly in Taipei, Taiwan. Sci Rep 2019;9:11176. PUBMED | CROSSREF

39. Ding Y, Wan Q, Liu W. Effects of atmospherically relevant $PM_{2.5}$ on skeletal muscle mitochondria: a review of damage mechanisms and potential of exercise interventions. Front Public Health 2025;13:1615363. PUBMED | CROSSREF

40. Li N, Xia T, Nel AE. The role of oxidative stress in ambient particulate matter-induced lung diseases and its implications in the toxicity of engineered nanoparticles. Free Radic Biol Med 2008;44:1689-99. PUBMED | CROSSREF

41. Barbier E, Carpentier J, Simonin O, Gosset P, Platel A, Happillon M, Alleman LY, Perdrix E, Riffault V, Chassat T, et al. Oxidative stress and inflammation induced by air pollution-derived $PM_{2.5}$ persist in the lungs of mice after cessation of their sub-chronic exposure. Environ Int 2023;181:108248. PUBMED | CROSSREF

42. Visser M, Pahor M, Taaffe DR, Goodpaster BH, Simonsick EM, Newman AB, Nevitt M, Harris TB. Relationship of interleukin-6 and tumor necrosis factor-alpha with muscle mass and muscle strength in elderly men and women: the Health ABC Study. J Gerontol A Biol Sci Med Sci 2002;57:M326-32. PUBMED | CROSSREF

43. Costamagna D, Costelli P, Sampaolesi M, Penna F. Role of inflammation in muscle homeostasis and myogenesis. Mediators Inflamm 2015;2015:805172. PUBMED | CROSSREF

44. Ceglia L. Vitamin D and skeletal muscle tissue and function. Mol Aspects Med 2008;29:407-14. PUBMED | CROSSREF

45. Chen Y, Zhang J, Ge X, Du J, Deb DK, Li YC. Vitamin D receptor inhibits nuclear factor κB activation by interacting with IκB kinase β protein. J Biol Chem 2013;288:19450-8. PUBMED | CROSSREF

46. Tao S, Zhang H, Xue L, Jiang X, Wang H, Li B, Tian H, Zhang Z. Vitamin D protects against particles-caused lung injury through induction of autophagy in an Nrf2-dependent manner. Environ Toxicol 2019;34:594-609. PUBMED | CROSSREF

47. Gordon BS, Kelleher AR, Kimball SR. Regulation of muscle protein synthesis and the effects of catabolic states. Int J Biochem Cell Biol 2013;45:2147-57. PUBMED | CROSSREF

48. Su R, Jin X, Zhang W, Li Z, Liu X, Ren J. Particulate matter exposure induces the autophagy of macrophages via oxidative stress-mediated PI3K/AKT/mTOR pathway. Chemosphere 2017;167:444-53. PUBMED | CROSSREF

49. Powers SK, Ji LL, Kavazis AN, Jackson MJ. Reactive oxygen species: impact on skeletal muscle. Compr Physiol 2011;1:941-69. PUBMED | CROSSREF

50. Dzik KP, Kaczor JJ. Mechanisms of vitamin D on skeletal muscle function: oxidative stress, energy metabolism and anabolic state. Eur J Appl Physiol 2019;119:825-39. PUBMED | CROSSREF

51. Bollen SE, Bass JJ, Fujita S, Wilkinson D, Hewison M, Atherton PJ. The vitamin D/vitamin D receptor (VDR) axis in muscle atrophy and sarcopenia. Cell Signal 2022;96:110355. PUBMED | CROSSREF

52. Xing YF, Xu YH, Shi MH, Lian YX. The impact of PM2.5 on the human respiratory system. J Thorac Dis 2016;8:E69-74. PUBMED | CROSSREF

53. Liu W, Wang Z, Kim MH, Gu Y, Sim HJ, Lee JC, Kook SH, Kim SH. Exposure of young mice to atmospherically relevant $PM_{2.5}$ has sex-dependent long-lasting impacts on the skeletal muscle system. Aging Dis 2024;16:3690-705. PUBMED | CROSSREF

54. Lu M, Zhan Z, Li D, Chen H, Li A, Hu J, Huang Z, Yi B. Protective role of vitamin D receptor against mitochondrial calcium overload from $PM_{2.5}$-induced injury in renal tubular cells. Redox Biol 2025;80:103518. PUBMED | CROSSREF

55. Bose S, Diette GB, Woo H, Koehler K, Romero K, Rule AM, Detrick B, Brigham E, McCormack MC, Hansel NN. Vitamin D status modifies the response to indoor particulate matter in obese urban children with asthma. J Allergy Clin Immunol Pract 2019;7:1815-1822.e2. PUBMED | CROSSREF

56. Whyand T, Hurst JR, Beckles M, Caplin ME. Pollution and respiratory disease: can diet or supplements help? A review. Respir Res 2018;19:79. PUBMED | CROSSREF

57. Giustina A, Bilezikian JP, Adler RA, Banfi G, Bikle DD, Binkley NC, Bollerslev J, Bouillon R, Brandi ML, Casanueva FF, et al. Consensus statement on vitamin D status assessment and supplementation: whys, whens, and hows. Endocr Rev 2024;45:625-54. PUBMED | CROSSREF

58. Martin L, Nasir H, Bagheri R, Ugbolue UC, Laporte C, Baker JS, Gu Y, Zak M, Duclos M, Dutheil F. Physical activity, air pollution, and mortality: a systematic review and meta-analysis. Sports Med Open 2025;11:35. PUBMED | CROSSREF

59. Tainio M, Jovanovic Andersen Z, Nieuwenhuijsen MJ, Hu L, de Nazelle A, An R, Garcia LMT, Goenka S, Zapata-Diomedi B, Bull F, et al. Air pollution, physical activity and health: a mapping review of the evidence. Environ Int 2021;147:105954. PUBMED | CROSSREF