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Multi night digital assessment of sleep disordered breathing is associated with accelerated vascular aging

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Abstract

Pulse wave velocity (PWV) is a marker of vascular aging and cardiovascular risk. Obstructive sleep apnea (OSA) may accelerate vascular decline, but evidence from single-night assessments is inconsistent. We examined associations of multi-night OSA severity, night-to-night variability, and snoring with arterial stiffness in a real-world setting. Adults used two in-home digital devices over a ~4y period: an under-mattress sleep sensor to quantify nightly OSA severity and snoring, and a smart scale to measure aortic-leg PWV. Among 29,653 participants from 20 countries (52±12 years; 84% male; BMI 27.3±4.9 kg/m²), increasing OSA severity was associated with higher PWV in a dose-response manner, independent of age, sex, and BMI. Participants with mild OSA but high variability had PWV levels comparable to severe OSA. Higher snoring burden independently predicted higher PWV across OSA severity categories. Multi-night in-home assessments of OSA and snoring may better reflect cardiovascular risk with potential to inform personalized management.

Keywords: Obstructive sleep apnea; night-to-night variability; snoring; arterial stiffness; pulse wave velocity; digital health; digital phenotyping

Introduction

The vascular system is comprised of a complex network of arteries, veins, microvessels, and specialized vessels such as arteriovenous anastomoses that play a vital role in oxygen and nutrient delivery, waste removal, thermoregulation and overall tissue homeostasis. As a natural consequence of aging, blood vessels accumulate a multifaceted array of morphological and functional changes that collectively contribute to progressive circulatory dysfunction and the onset of age-related diseases^{1,2}. Among these changes, arterial stiffening represents a hallmark of early vascular aging and a key determinant of hypertension, organ damage and cardiovascular mortality, independent of traditional risk factors^{3,4}. Carotid-femoral pulse-wave velocity (PWV) is the current gold-standard measure of aortic stiffness⁵, as endorsed by the European Society of Hypertension (ESH) and the European Society of Cardiology (ESC)^{6,7}. PWV is a robust predictor of cardiovascular disease and mortality and independently enhances cardiovascular risk stratification beyond conventional risk models⁸⁻¹¹. Elevated PWV is commonly observed in chronic diseases whereby accelerated vascular aging may represent a shared etiological pathway underlying the increased cardiovascular risk observed in chronic disease populations¹²⁻¹⁴.

Obstructive sleep apnea (OSA) is the most prevalent form of sleep-disordered breathing (SDB), affecting nearly one billion adults globally^{15,16}. This chronic condition is characterized by recurrent interruptions of breathing during sleep, resulting in repetitive cycles of intermittent hypoxia and sleep fragmentation. OSA generates a state of oxidative and endothelial stress and dysfunction, metabolic dysregulation, and chronic low-grade inflammation^{17,18}, all of which are key hallmarks of biological aging and predispose to vascular decline^{19,20}. Despite growing interest

in the potential link between OSA and accelerated vascular aging, existing evidence remains inconsistent²¹⁻²⁵. Prior studies have relied on single-night assessments of OSA severity, typically measured using the apnea-hypopnea index (AHI), which quantifies the average number of breathing pauses per hour of sleep. However, AHI and other OSA severity metrics have substantial night to night variability which leads to diagnosis incertitude in a high proportion of patients²⁶⁻²⁸. Emerging literature indicates that such physiological variability may impose additional strain on the cardiovascular system²⁹⁻³¹. Marked variability could therefore also contribute to accelerated vascular deterioration. Yet, this hypothesis has not been investigated. Additionally, habitual snoring, a major feature of SDB independently associated with hypertension³², may further exacerbate vascular aging by adding mechanical and inflammatory stress on the upper airway during sleep. However, data on the potential association between arterial stiffening and snoring is scarce and mainly limited to self-report³³.

Recent advances in smart health technologies now enable continuous, unobtrusive objective monitoring of sleep, breathing, and cardiovascular function in naturalistic settings. These tools allow for digital phenotyping of physiological processes over extended periods, and capture dynamic fluctuations that traditional, single timepoint assessments overlook³⁴⁻³⁹. In this study, we leverage a unique multi-country consumer dataset of longitudinal SDB metrics derived from an under-mattress sleep sensor and repeated PWV measurements obtained from a smart bathroom scale. By examining data over multiple years in real-time, we sought to explore the strength of longitudinal associations between multi-night OSA severity, night-to-night variability, and snoring with arterial stiffness.

Results

A total of 29,653 participants from 19 countries with more than 300 participants were included (mean±SD age 52 ±12 years; 84% male; mean BMI 27.3 ±4.9 kg/m²) (**Table 1**). Most participants were from Germany (27.2%), the US (16.9%), France (16.3%), the UK (7.0%), and Switzerland (5.6%). The mean PWV was 7.56 ±1.02 m/s, derived from a median [IQR] of 38 [64] measurements per participant per year. There was a progressive increase in PWV with advancing age and increasing BMI. Males had higher PWV values than females (**Figure 1**). Participants had a median [IQR] of 252 [103] nights of sleep data per year. Based on the average AHI across the study period, 40% had no OSA, 35% had mild OSA, 17% had moderate OSA, and 8% had severe OSA. No significant differences in the number of sleep recordings or PWV measurements across OSA severity categories were observed (**Table 1**).

Multi-night OSA severity and PWV

Compared to the non-OSA group, participants with mild OSA had a mean (95% CI) PWV difference of +0.08 (0.06 to 0.10) m/s; those with moderate OSA had +0.14 (0.12 to 0.16) m/s; and those with severe OSA had +0.16 (0.13 to 0.19) m/s (**Figure 2**). There was no evidence of an effect modification by sex (p-for-interaction = 0.34; Supplement **Figure S1a**). However, associations were stronger among younger participants (p-for-interaction < 0.001; **Figure S1b**) and those with lower BMI (p-for-interaction < 0.001; **Figure S1c**). Removing potential outliers did not change the association between OSA severity and PWV (**Figure S2**).

Night-to-night OSA variability and PWV

The interaction p value for OSA severity and night-to-night variability for the association with PWV was 0.05. The association between OSA variability and PWV was more pronounced among participants with no, mild, or moderate OSA, but was not significant for severe OSA (**Figure 3a**). Within each OSA severity group, high night-to-night variability in AHI, comparing the 90th to the 10th percentile of OSA variability within that group, was associated with higher PWV values. Specifically, PWV was higher by +0.11 m/s (5.6 vs. 1.3 events/h; 95%CI 0.09 to 0.13) in participants with no OSA, +0.12 m/s (10.1 vs. 5.2 events/h; 95%CI 0.10 to 0.14) in mild OSA, and +0.06 m/s (15.6 vs. 8.2 events/h; 95%CI 0.03 to 0.08) in moderate OSA (**Figure 3b**). Conversely, there was no significant difference in PWV between high and low night-to-night variability percentiles (90th vs. 10th, 22 vs. 12.2 events/h) among participants with severe OSA (mean [95%CI] = 0.01 [-0.03 to 0.04] m/s). Participants with mild OSA and high night-to-night variability (above the 90th percentile; ~10 events/h) had a predicted PWV of 7.72 (7.70 to 7.74) m/s, comparable to those with severe OSA but low variability (below the 10th percentile; ~12 events/h, 7.77 (7.73 to 7.80 m/s). In sensitivity analyses, a model investigating the association between OSA variability and PWV without adjustment for OSA severity showed that high night-to-night variability in AHI, comparing the 90th to the 10th percentile of OSA variability across all participants, was associated with a 0.24 m/s increase in PWV (13.7 vs. 2.4 events/h; 95%CI 0.22 to 0.27; **Figure S3**). Findings did not change after removing outliers (**Figure S4**).

Snoring burden and PWV

The interaction between OSA severity categories and snoring burden was not significant (p-for interaction = 0.17; **Figure 4a**). Higher percentage of time spent snoring (90th vs. 10th) was

consistently associated with elevated PWV across all OSA severity categories: no OSA +0.11 m/s (0.1 vs. 12.1%; 95%CI 0.09 to 0.13), mild OSA +0.12 m/s (0.3 vs. 22.1%; 95%CI 0.10 to 0.14), moderate OSA +0.06 m/s (0.6 vs. 33.7%; 95%CI 0.03 to 0.08), and severe OSA +0.14 m/s (1.2 vs. 46.3%; 95%CI 0.10 to 0.18) (**Figure 4b**). Participants with no OSA but a high snoring burden (above the 90th percentile; ~12%), had a predicted PWV of 7.68 (7.66 to 7.70) m/s, similar to those with severe OSA but minimal snoring (below the 10th percentile; ~1%, 7.66 (7.62 to 7.71) m/s). In sensitivity analyses without adjustment for OSA, higher snoring burden, comparing the 90th to the 10th across all participants, was associated with a 0.24 m/s higher PWV (24.1 vs. 0.1%; 95%CI 0.22 to 0.26; **Figure S5**). Effect estimates were slightly larger when removing outliers, but overall interpretation remained consistent (**Figure S6**).

Discussion

This large, digital technology-enabled, multi-national, real-world study of nearly 30,000 participants monitored at home for ~4 years reveals that SDB is associated with elevated arterial stiffness, a hallmark of vascular decline and an established precursor of cardiovascular disease. Multi-night OSA severity shows a dose-response relationship with PWV, independent of age, sex, and BMI. Beyond average OSA severity, greater night-to-night variability in OSA severity and higher snoring burden are both independently associated with increased arterial stiffness. These findings highlight the importance of multi-night sleep assessments to capture the cumulative and fluctuating burden of disturbed breathing during sleep to better inform personalized cardiovascular risk stratification and clinical management of OSA and CVD.

The mechanical properties of the arterial system are fundamental to circulatory physiology and cardiovascular health. Arterial stiffening often precedes the onset of clinically significant systemic hypertension and independently predicts premature coronary artery disease, atherosclerosis, and cardiovascular mortality⁴⁰. PWV, a well-established proxy of arterial stiffness, ranks among the strongest predictors of cardiovascular disease, independent traditional risk factors such as blood pressure and BMI^{8,9,11}. A recent meta-analysis reported a 15% increase in mortality for each 1 m/s increase in PWV¹⁰. Despite its strong prognostic value, the clinical use of PWV has been limited by the need for specialized equipment and expertise. Recent advances in consumer-grade smart bathroom scales now enable longitudinal, self-administered PWV measurements with clinically validated accuracy⁴¹ and high user acceptability⁴². Together with the growing adoption of home-based sleep sensors, these technologies create an unprecedented opportunity to characterize the long-term interplay between multi-night digital SDB phenotypes and vascular aging in real-world environments, which, to our knowledge, is explored in this study for the first time.

An early study more than two decades ago suggested that people with severe OSA ($n = 16$) had higher PWV and other signs of early vascular aging compared with age-, sex-, and BMI-matched controls ($n = 12$)⁴³. Subsequent studies have yielded inconsistent results^{23,24}. A meta-analysis of five studies ($n = 282$) reported significantly higher carotid-femoral PWV in OSA compared to controls (standardized mean difference [95% CI] = 0.45 [0.21 to 0.69]) m/s²², yet heterogeneity in study populations and PWV assessment methods was considerable. In contrast, a later individual patient data meta-analysis of nine studies ($n = 893$) using standardized PWV technique found no significant association between PWV and AHI or OSA severity categories²¹. A key

limitation of prior studies was their reliance on single time-point assessments of OSA and PWV, which overlooked nightly variability of OSA severity and the temporal evolution of vascular remodeling. In the present study, long-term, technology-enabled, longitudinal measures with nightly monitoring of OSA (median >250 nights per participant per year) and repeated PWV assessments (median ~40 per participant per year), revealed a robust dose-response association between OSA severity and arterial stiffness. Accumulating evidence supports OSA as a potent accelerator of biological aging⁴⁴⁻⁴⁶, particularly in younger individuals⁴⁷. Consistent with this, we observed stronger associations between OSA and arterial stiffness among younger participants.

Beyond mean OSA severity, we found that night-to-night variability in OSA severity is associated with arterial stiffening, particularly among people with mild OSA (based on average multi-night AHI). Participants with mild OSA but high nightly variability have PWV values comparable to those with severe OSA and low variability. This observation is clinically relevant, given that people with mild OSA are often not considered for treatment despite growing evidence supporting that fluctuating nightly exposure to respiratory disturbance increases the risk of adverse cardiovascular outcomes, including hypertension^{29,31}, atrial fibrillation⁴⁸, and major cardiovascular and cerebrovascular events. A variety of factors can influence AHI variability, including body and head position, variations in nasal resistance, ambient environmental conditions, and behavioral and lifestyle factors⁴⁹⁻⁵³. Our findings reinforce the need to move beyond current single-night diagnostic tests towards multi-night sleep assessments, which more accurately capture real-world patterns of disease risk exposure. Given the importance of selecting effective long-term treatment and ensuring sustained adherence, multi-night testing

with posture detection may better inform individualized therapeutic decisions, as positional OSA and posture-related variability in disease severity can be missed with single-night assessments.

Few studies have investigated the relationship between snoring and arterial stiffness. In the Brisighella Heart Study, self-reported snoring, both with and without concomitant self-reported OSA, was associated with higher PWV compared to non-snorers³³. Conversely, another study using objective measures of snoring frequency found no significant association with PWV after adjusting for measured confounders⁵⁴. However, single-night snoring assessments may not reliably reflect typical snoring, as snoring duration and intensity may change from night to night due to different factors such as sleep position or alcohol consumption. Our analysis, leveraging measurements over multiple years to capture cumulative snoring burden, showed that habitual snoring was associated with increased arterial stiffness independent of the presence and severity of OSA. This finding adds to the growing literature linking snoring, independently of OSA severity, with cardiovascular risk^{32,55-57}, and highlights the importance of considering snoring within clinical sleep care.

Several pathophysiological pathways may explain the observed associations between SDB and arterial stiffening, including oxidative stress, systemic inflammation, hypercoagulability, and endothelial dysfunction, which are established mediators of vascular remodeling⁵⁸. Respiratory event-triggered heart rate surges during sleep may play a key role in this relationship, as suggested by a recent mediation analysis in a large Japanese cohort of OSA patients⁵⁹. Highly variable OSA severity from night to night may prevent potential compensatory mechanisms, potentially further contributing to progressive cardiovascular function decline. However, this

hypothesis needs further investigation. Additionally, the persistent mechanical stress of snoring vibrations can induce local inflammation and microvascular damage in upper airway tissues^{60,61}, which may contribute to systemic vascular decline. In a rabbit model, reduced baroreflex sensitivity with external upper airway compression-induced snoring, but not peri-carotid tissue vibration, supports that chronic partial upper airway obstruction and exposure to more negative intrathoracic pressures may also play an important role in chronic arterial stiffening and hypertension⁶². Further mechanistic studies are needed to better understand these pathways and identify potential therapeutic targets.

Arterial stiffness is considered a modifiable process and therefore represents a promising clinical target for therapeutic intervention for reducing cardiovascular risk⁶³. Continuous positive airway pressure (CPAP) therapy lowers PWV in people with OSA in a recent meta-analysis of randomized trials (9 studies; n = 685; mean change [95%CI] -0.44 m/s [-0.76 to -0.12])⁶⁴, consistent with findings from an earlier meta-analysis of randomized and observational data (15 studies; n = 615)⁶⁵. These results suggest that the vascular consequences of OSA may at least be partially reversible, aligning with previous evidence that epigenetic aging-related acceleration in OSA can be reversed with adherent CPAP treatment⁴⁶. Similarly, the detrimental vascular effects of snoring, independent of OSA, may also be reversible, although evidence from interventional studies is lacking. Lifestyle modifications have demonstrated clinical efficacy in preventing and treating arterial stiffening⁶⁶, which is particularly relevant given the high prevalence of obesity and sedentarism in SDB populations. Remote monitoring of PWV may therefore serve as a useful tool to assess treatment effects to mitigate cardiovascular risk and promote healthier longevity in people with SDB⁶⁵.

This study has several unique strengths, including its unprecedented scale, multi-year follow-up, and use of novel validated, consumer-grade digital tools to enable objective, repeated measurements of SDB and PWV in naturalistic home environments. However, there are several potential limitations. First, participation was self-selected, resulting in overrepresentation of male sex and likely people of higher socio-economic status and those more conscious about their health. Second, OSA and snoring metrics were derived from a consumer sleep monitor, precluding analysis of other physiological measures such as oxygen desaturation, arousals, or snoring intensity. Nevertheless, multi-night home-based measures and robust relationships with PWV support their utility and may better capture cumulative exposure than single-night laboratory settings. Similarly, arterial stiffness was estimated using a smart scale without clinical assessments of PWV or other measures of vascular function and structure. Third, data on potential confounders or effect mediators, such as alcohol intake, smoking, diet, exercise, comorbidities, medications and treatments including continuous positive airway pressure (CPAP) were unavailable, which may result in residual confounding. Fourth, although annual averaging of exposures and outcomes was used to mitigate potential short-term and seasonal variability, some seasonal factors such as temperature, changes in physical activity, or respiratory infections may have influenced the observed associations.

In this large, multi-national, real-world study involving nearly 30,000 participants monitored longitudinally at home, multi-night OSA severity, night-to-night variability, and snoring burden were each independently associated with a significant increase in arterial stiffness. These novel findings suggest that accelerated vascular system decline may represent a key mechanistic pathway linking SDB with cardiovascular pathology and emphasize the clinical importance of

multi-night sleep assessments to enhance cardiovascular risk stratification and inform personalized management of SDB.

Methods

Study population

Data were acquired from adults who used two FDA-cleared connected health devices: an under-mattress sleep sensor and a smart bathroom scale, between January 2021 and November 2024. Participants self-reported their sex and age at the time of device setup. All participants provided written consent for the use of their deidentified data for research. The study performed in accordance with the Declaration of Helsinki and was approved by the Flinders University Human Research Ethics Committee (Project number: 4291). Clinical trial number: not applicable.

SDB assessments

Nightly SDB metrics, including OSA severity (AHI) and snoring time, were derived from the under-mattress sleep sensor (Withings Sleep Analyser/Sleep Rx). This wearable integrates a built-in microphone to record sound with a high-sensitivity pressure sensor to assess ballistocardiographic signals of body movement, heart rate, and respiratory motion. These signals are processed via AI-based proprietary algorithms to estimate sleep parameters, including total sleep time, AHI, and total snoring time. Nights with total sleep time <5 hours were excluded from AHI computation. Independent validation studies comparing this device against gold-standard polysomnography (PSG) have shown minimal systemic bias and high accuracy for OSA classification: mild (sensitivity = 89%, specificity = 75%), moderate-to-severe (88%, 88%), and

severe (86%, 91%)^{26,67}. These performance metrics are comparable to other wearable and nearable devices that have received regulatory clearance for home-based OSA monitoring⁶⁸. Validation of snoring duration estimated from the under-mattress sensor have also shown a close agreement with PSG ($r^2 = 0.76$)⁶⁹.

Multi-night OSA severity was calculated as the yearly mean AHI for each participant, excluding years with <52 nights of data. Standard clinical thresholds were used to categorize OSA severity: <5 (no OSA), 5-15 (mild), 15-30 (moderate), and ≥ 30 events/h (severe)^{70,71}. Night-to-night variability in OSA severity was quantified annually as the root mean square of successive nightly AHI differences²⁹. Snoring burden was defined as the percentage of total sleep time spent snoring, averaged yearly across the monitoring period³².

PWV and weight assessments

Aortic-leg PWV measurements were obtained using the smart bathroom scale (Withings Body Cardio), with recordings performed in the standing position. This device employs impedance plethysmography and ballistocardiography to estimate the pulse transit time between the onset of systolic ejection and pulse arrival at the foot. PWV is derived using proprietary AI-based algorithms that incorporate user height and BMI. Validation against carotid-femoral PWV assessed via applanation tonometry (SphygmoCor) using three independent datasets has shown good agreement with the clinical gold standard⁴¹. Repeated weight measurements were obtained from the same scale, from which the yearly mean BMI per user was calculated.

The primary outcome was yearly mean PWV, selected to minimize seasonal variation⁷². Years with <12 PWV recordings per participant were excluded from analysis.

Statistical analysis

Multivariable linear mixed-effect models were used to examine associations between SDB metrics and PWV, adjusting for age, sex, and BMI (time-varying). Participant ID and country of residence were included as random intercepts. The primary model investigated associations between OSA severity (categorical) and mean PWV. Two additional models assessed interactions between OSA severity (categorical) and OSA variability (continuous; modelled using a cubic spline with 4 degrees of freedom), and between OSA severity (categorical) and snoring burden (continuous; modelled similarly). Interactions with sex, age, and BMI were also explored. Estimated marginal means were used to summarize the model-predicted PWV values across exposure percentiles (10th, 25th, 50th, 75th, and 90th percentile)⁷³. For contextual interpretation, associations of age (5-year bins), sex, and BMI (3-kg/m² bins) with PWV were also evaluated. Data are presented as mean±SD unless specified otherwise. For each of the main analyses, sensitivity analyses were conducted excluding potential outliers, defined as values above the 99th percentile for exposures (mean OSA severity, OSA variability, snoring burden) and outcome (PWV), as well as values below the 1st percentile for PWV. This resulted in the exclusion of ~4.7% of data points. $p < 0.05$ was considered statistically significant.

Data Availability: The dataset associated with this study is stored in a proprietary repository (Withings) and cannot be shared publicly due to concern for privacy, ethical, and legal reasons. The investigator team accessed the data through an application process to Withings, and a formal data sharing agreement designed to safeguard user confidentiality, as outlined in the terms and conditions and privacy policy documentation. Queries for data access can be directed to Withings (data_compliance@withings.com) with a timeframe for response of four weeks. Specific de-identified raw data that support the findings of this study, including individual data, are available from the corresponding author (lucia.pinilla@flinders.edu.au) upon request subject to ethical and data custodian (Withings) approval described above. The timeframe for response to requests will be up to four weeks.

Code Availability: The underlying code for this study is not publicly available but may be made available to qualified researchers on reasonable request from the corresponding author (lucia.pinilla@flinders.edu.au).

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Author Contributions: LP, BL, and DJE conceived and designed the study. BL performed data extraction. BL, LP, KS, and DJE conducted the data analysis and interpretation. LP drafted the

manuscript. BL, KS, PL, AV, AM, AH, PE, JLP, RA, PC, and DJE provided critical input on data interpretation and contributed to revision of the manuscript. All authors had full access to the data, reviewed and approved the final manuscript, and accept responsibility for its submission for publication.

Competing Interests:

PL receives partial PhD funding from Withings. PE is consultant for Withings. JLP reports income related to medical education from RESMED, SEFAM, Philips, Zoll-Respicardia, Eli Lilly, Idorsia, Pharmanovia, Biosency and Bioprojet. Outside the submitted work, DJE has had research grants from Bayer, Apnimed, Takeda, Invicta Medical (now Restera), and Eli Lilly. DJE currently serves as a scientific advisor/consultant for Apnimed, Invicta Medical (now Restera), Restora, Takeda, SleepRes, Mosanna and humanity Medtech. LP, KS, AV, AM, AH, RA, PC, and BL declare no financial or non-financial competing interests.

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FIGURES

Figure 1. Distribution of yearly mean pulse wave velocity (PWV) values across the study population and across demographic categories. **a)** Histogram and density plot showing the distribution of PWV values across the overall study population. The red line represents the density estimate and the dashed red line indicates the median PWV. **b)** Yearly mean PWV according to age (5-year bins). **c)** Yearly mean PWV according to sex (male/female). **d)** Yearly mean PWV according to body mass index (BMI) (3-kg/m² bins). Results are presented as estimated marginal means and 95% CIs from multivariable linear mixed-effect models including participant ID and country of residence as random intercepts.

Figure 2. Association between obstructive sleep apnea (OSA) severity categories and yearly mean pulse wave velocity (PWV). OSA severity was categorized according to the yearly mean apnea-hypopnea index (AHI) using standard clinical thresholds: <5 (no OSA), 5-15 (mild), 15-30 (moderate), and ≥30 events/h (severe). Results are presented as estimated marginal means and 95% CIs from multivariable linear mixed-effect models adjusted for age, sex, and time-varying body mass index (BMI) and including participant ID and country of residence as random intercepts.

Figure 3. Association between night-to-night variability in obstructive sleep apnea (OSA) severity and yearly mean pulse wave velocity (PWV) across OSA severity categories. **a)** Night-to-night variability was quantified as the yearly root mean square of successive nightly differences (RMSSD) in the apnea-hypopnea index (AHI). OSA severity was categorized according to the yearly mean AHI using standard clinical thresholds: <5 (no OSA; green), 5-15 (mild; orange), 15-30 (moderate; purple), and ≥30 events/h (severe; pink). Solid lines represent fitted regression curves, and shaded areas indicate 95% CIs. Markers (triangle, square, and circle) correspond to the 10th, 50th, and 90th percentiles of night-to-night variability, respectively. **b)** Mean PWV and 95% CI at the 10th, 50th, and 90th percentiles of night-to-night variability in AHI for each OSA severity category, estimated from multivariable linear mixed-effect models adjusted for age, sex, and time-varying body mass index (BMI) and including participant ID and country of residence as random intercepts.

Figure 4: Association between snoring burden and yearly mean pulse wave velocity (PWV) across obstructive sleep apnea (OSA) severity categories. **a)** Snoring burden was quantified as the yearly percentage of total sleep time (TST) spent snoring. OSA severity was categorized according to the yearly mean apnea-hypopnea index (AHI) using standard clinical thresholds: <5 (no OSA; green), 5-15 (mild; orange), 15-30 (moderate; purple), and ≥30 events/h (severe; pink). Solid lines represent fitted regression

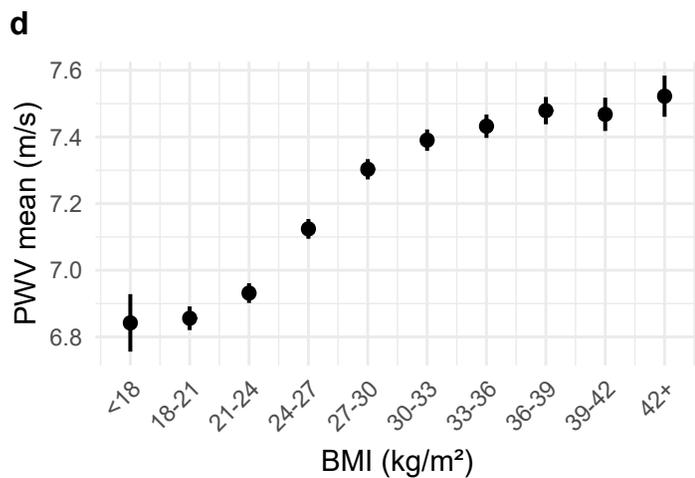
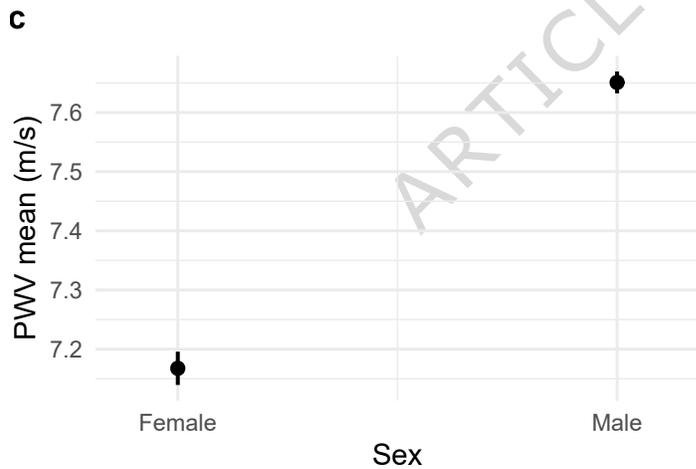
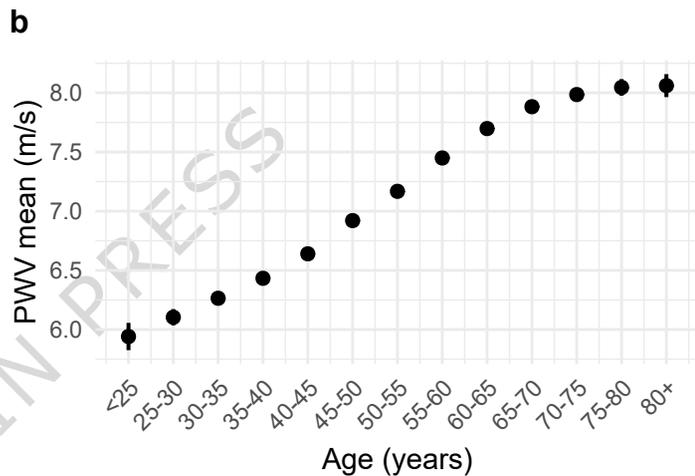
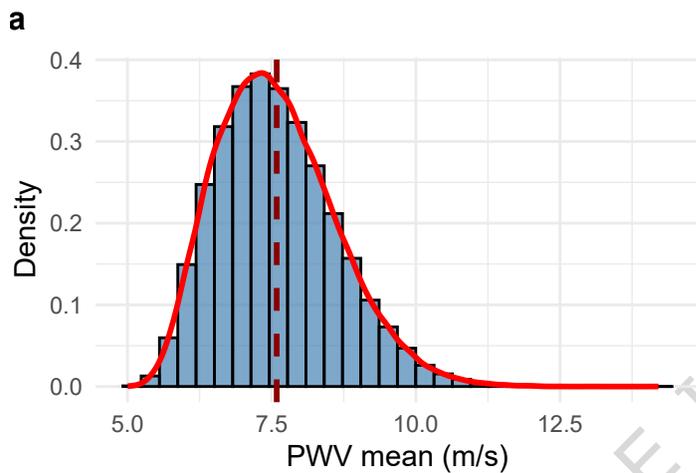
curves, and shaded areas indicate 95% CIs. Markers (triangle, square, and circle) correspond to the 10th, 50th, and 90th percentiles of snoring burden, respectively. **b)** Mean PWV and 95% CI at the 10th, 50th, and 90th percentiles of snoring burden for each OSA severity category, estimated from multivariable linear mixed-effect models adjusted for age, sex, and time-varying body mass index (BMI) and including participant ID and country of residence as random intercepts.

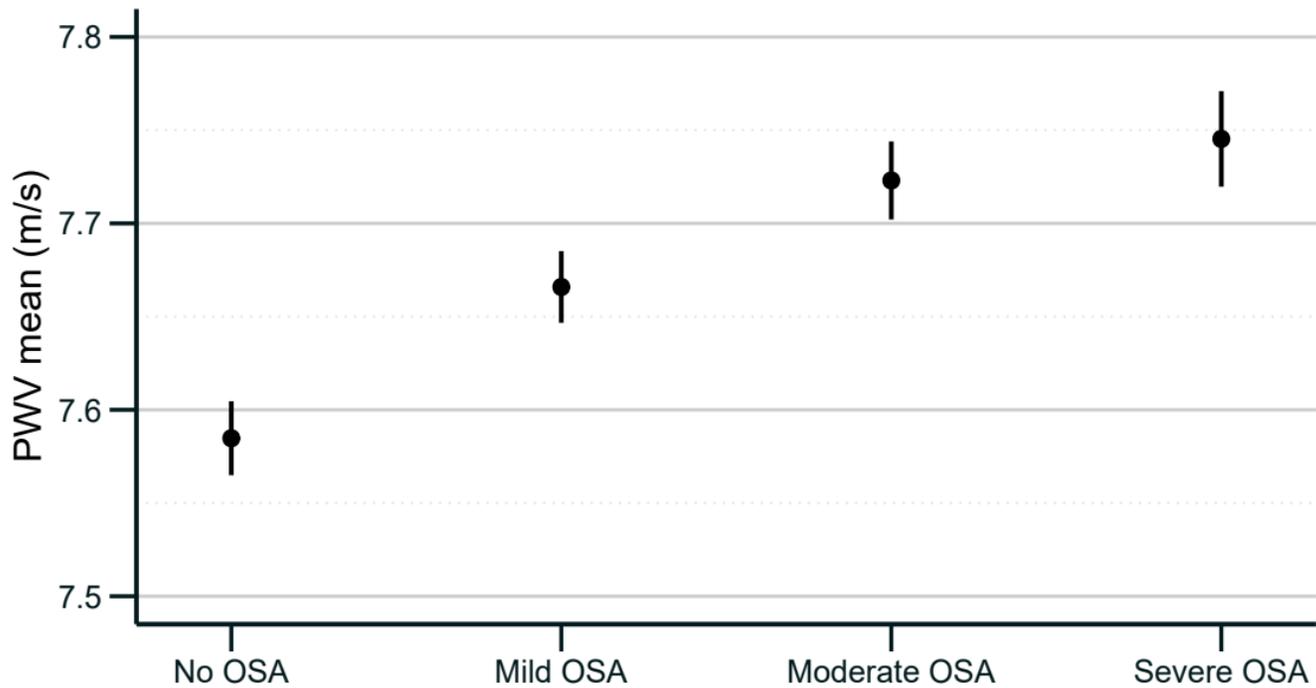
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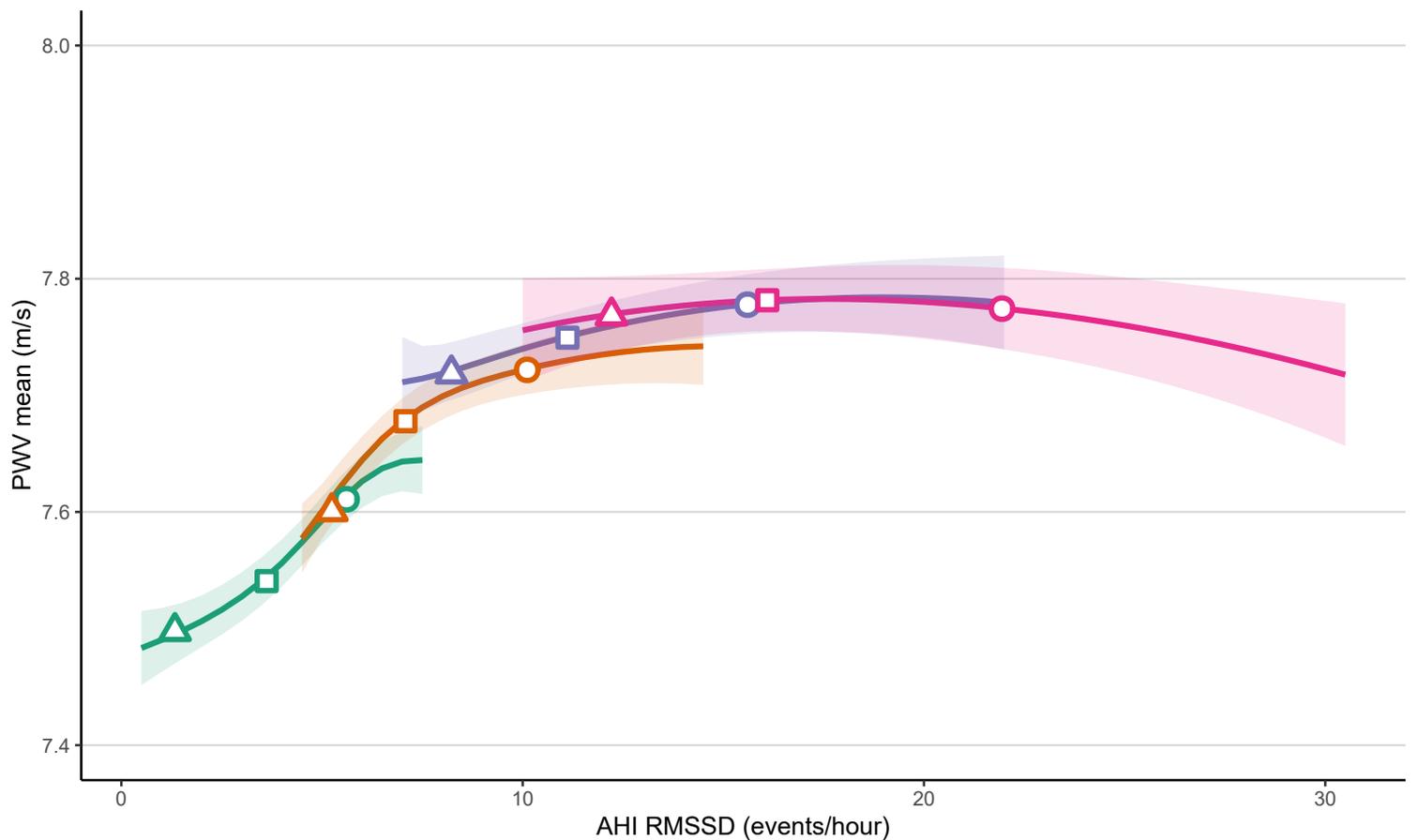
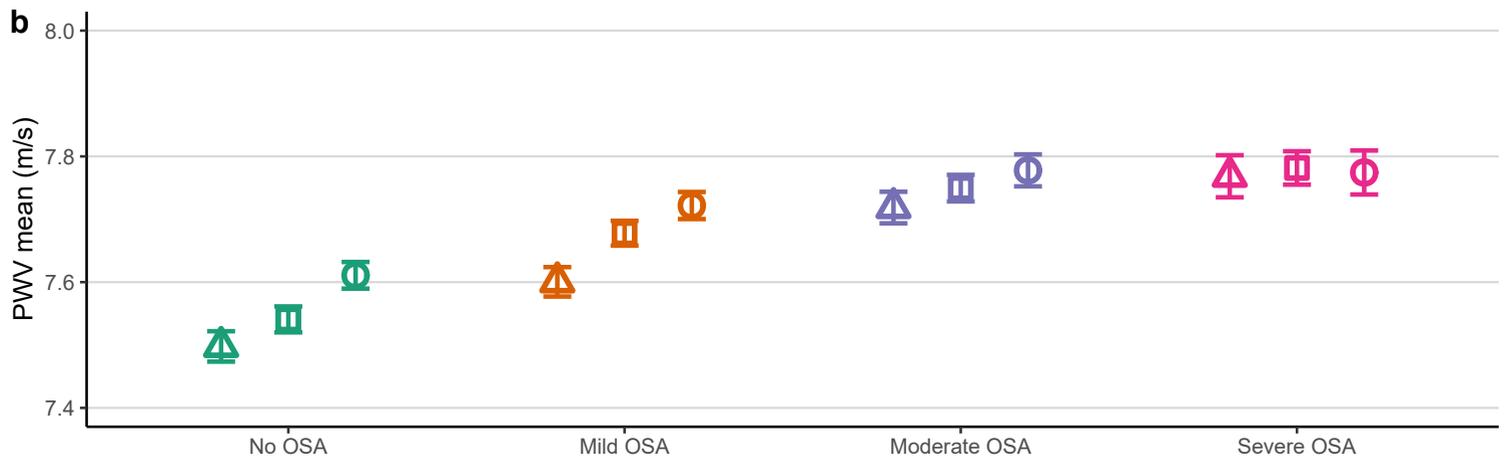
TABLES

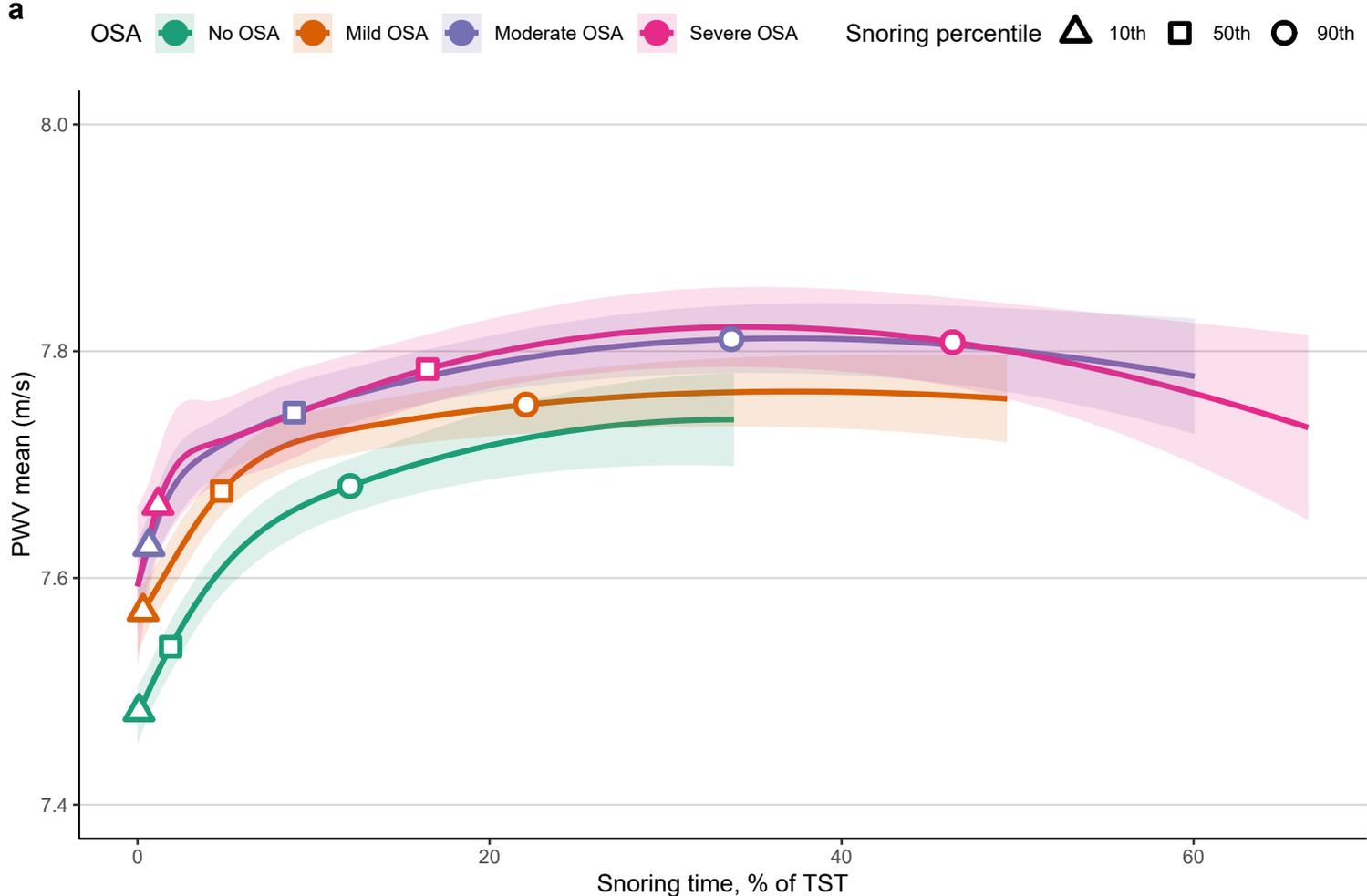
Table 1. Participant characteristics by categories of obstructive sleep apnea (OSA) severity, categorized according to the yearly mean apnea-hypopnea index (AHI) using standard clinical thresholds: <5 (no OSA), 5-15 (mild), 15-30 (moderate), and ≥ 30 events/h (severe). Data are presented as mean (SD) or median [p_{25} ; p_{75}] unless otherwise specified. For participants with multiple years of data, the last year of data was used to generate the table.

	OSA severity category				
	Overall	No OSA	Mild OSA	Moderate OSA	Severe OSA
n, (%)	29653	11850 (40)	10284 (35)	5143 (17)	2376 (8)
Age, years	52.3 (12.0)	47.3 (11.3)	53.3 (11.1)	58.1 (10.8)	59.6 (10.7)
Females, n (%)	4842 (16.3)	2659 (22.4)	1508 (14.7)	538 (10.5)	137 (5.8)
Body mass index, kg/m²	27.3 (4.9)	25.8 (4.5)	27.5 (4.6)	28.8 (4.8)	31.0 (5.3)
Pulse wave velocity measurements, n/year	38.0 [20.0, 84.0]	37.0 [21.0, 84.0]	38.0 [21.0, 85.0]	38.0 [20.0, 87.0]	35.0 [19.8, 74.0]
Pulse wave velocity mean, m/s	7.6 (1.0)	7.2 (0.9)	7.7 (1.0)	8.0 (1.0)	8.1 (1.0)
Sleep recordings, n/year	252.0 [189.0, 292.0]	252.0 [189.0, 292.0]	252.0 [190.0, 290.0]	252.0 [187.0, 292.0]	249.0 [186.8, 294.0]
AHI mean, events/h	7.0 [2.6, 15.2]	1.9 [0.8, 3.3]	8.8 [6.7, 11.5]	20.1 [17.3, 23.9]	40.0 [34.2, 50.5]
AHI root mean square of successive nightly differences, events/h	7.4 (4.8)	3.6 (1.7)	7.5 (2.2)	11.7 (3.3)	16.9 (4.5)
Snoring mean, % of TST	4.1 [1.0, 12.4]	1.9 [0.4, 5.9]	4.9 [1.4, 12.5]	9.0 [2.7, 20.6]	17.1 [5.6, 33.2]





aOSA  No OSA  Mild OSA  Moderate OSA  Severe OSA AHI RMSSD percentile  10th  50th  90th**b**

a**b**