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Beyond the apnea-hypopnea index: clinical phenotypic heterogeneity of obstructive sleep apnea in an Indian tertiary referral cohort using level III polysomnography

Siddhartha Chakraborty, Manu Chopra, Kesu Hari, Rahul Tyagi, Kislay Kishore, Aseem Yadav

Department of Pulmonary Medicine, Critical Care and Sleep Medicine, Army Institute of Cardio-Thoracic Sciences, Pune, India

Correspondence: Siddhartha Chakraborty, Department of Pulmonary Medicine, Critical Care and Sleep Medicine, Army Institute of Cardio-Thoracic Sciences, Pune, India, PIN - 411040. Tel.: - +918827348726. E-mail: doctorsleeves6620@gmail.com

Contributions: Siddhartha Chakraborty, Kesu Hari, Rahul Tyagi: experiments concept and design. Siddhartha Chakraborty, Manu Chopra, Kislay Kishore, Aseem Yadav: analysis and interpretation of experiments results. Ritwik Chakrabarti: radiological analysis of data. All the authors have read and approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

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Abstract

Obstructive sleep apnea (OSA) is usually classified by a single severity index, the Apnea-Hypopnea Index (AHI), which does not capture the clinical heterogeneity described in other populations. To characterize clinically distinct OSA phenotypes by body habitus, symptom–severity relationship, and by respiratory event type in an Indian tertiary referral cohort, using level III polysomnography. Retrospective analysis of 309 patients with OSA ($AHI \geq 5$) from consecutive level III polysomnography studies at a tertiary Indian center. Phenotypes were defined as follows: 1) lean [body mass index (BMI) $< 25 \text{ kg/m}^2$] versus non-lean OSA; 2) symptomatic vs. asymptomatic disease, using an Epworth Sleepiness Scale (ESS) score of 10 as a threshold, crossed with AHI severity; 3) apnea-predominant vs. hypopnea-predominant disease, from the respective event indices. An exploratory unsupervised cluster analysis on physiological variables was performed as a comparator. A lean phenotype was present in 12.3% of patients (95% confidence interval 9.1–16.5) and had lower AHI, less hypoxemia, and fewer daytime symptoms than non-lean OSA, with less moderate-to-severe disease (40.5% vs. 68.4%, $p=0.002$). Symptom burden was poorly matched to severity: 52.7% of severe OSA patients were not sleepy ($ESS < 10$), while 21.5% of mild OSA patients were. Sleepy patients had a greater hypoxemic burden but similar body habitus. Apnea-predominant disease (54.3% of patients with usable indices) occurred in younger, leaner patients but with a higher AHI than hypopnea-predominant disease. Both the lean-phenotype association with less severe disease and the apnea-predominant AHI difference persisted after adjustment for age, sex, and BMI. STOP-Bang score did not reliably discriminate moderate-to-severe disease in this already high-risk referral population (AUC 0.56), and hypertension and diabetes prevalence did not differ between lean and non-lean phenotypes. ODI graded on AHI's own thresholds agreed with the AHI category in 67.4% of patients (quadratic-weighted κ 0.82) and identified moderate-to-severe disease with 89% sensitivity and 92% specificity. Unsupervised clustering on AHI-correlated physiological variables did not identify phenotypes independent of severity. OSA in this referral cohort separates into body-habitus, symptom-discordance and event-type phenotypes that are not interchangeable with AHI severity alone. Recognizing these patterns may help identify lean patients, underrecognized asymptomatic severe disease, and mechanistically distinct event patterns.

Key words: obstructive sleep apnea, Epworth Sleepiness Scale, Apnea-Hypopnea Index.

Introduction

Obstructive sleep apnea (OSA) is conventionally graded by the apnea-hypopnea index (AHI) alone, therefore patients with similar AHI can differ markedly in body habitus, symptom burden and the physiological pattern of their respiratory events. Studies from outside India have described several such patterns: a lean or non-lean OSA phenotype attributed to craniofacial and upper-airway factors rather than adiposity [1,2]; a mismatch between AHI and daytime sleepiness, with some patients having severe disease but minimal symptoms [3]; and differing contributions of frank apneas versus hypopneas to the respiratory disturbance, which may reflect different mechanisms of upper airway collapse [4]. These studies underline that a single severity index may be insufficient to guide the right management.

Whether these patterns are present, and how common they are, has not been described in an Indian tertiary referral population undergoing Level III polysomnography testing, the diagnostic pathway most widely used in resource-limited settings [5]. We analysed a consecutive series of Level III sleep studies from this centre to describe three pre-specified OSA phenotypes - by body habitus, by symptom severity relationship, and by respiratory event type.

Materials and Methods

Design and setting

Retrospective analysis of consecutive adults referred for suspected OSA who underwent Level III polysomnography (SOMNOtouch RESP, SOMNOmedics GmbH) at a tertiary respiratory medicine department in western India during 2025, scored according to AASM 2017 criteria. This dataset overlaps with, but is not identical to, the cohort described in our earlier report on the diagnostic yield of Level 3 testing at this centre [6], which was restricted to studies performed between January and December 2025: the present dataset includes additional patients tested subsequently (till Jun 2026)). The present analysis addresses phenotypic heterogeneity, a question not examined in the earlier report. The parent study was approved by the Institutional Ethics Committee (IEC/2025/53); patients consented to use of non-identifying group data. The analysis cohort comprised all unique patients with OSA, defined as AHI of 5 events/h or more ($n = 309$); severity was graded mild (5–14.9) with symptoms or comorbidities, moderate (15–29.9) or severe (30 or more), as per The International Classification of Sleep Disorders, third edition.

Patients were classified as lean ($\text{BMI} < 25 \text{ kg/m}^2$) or non-lean ($\text{BMI} \geq 25 \text{ kg/m}^2$) using the WHO Asian BMI cut-off, on the basis that standard Western thresholds under-classify adiposity-related risk in South Asian populations. Hypertension and diabetes mellitus as comorbidities were also compared between lean and non-lean phenotypes. Moreover, patients were described according to their level of somnolence: with an ESS score of 10 or more, patients were classified as symptomatic, and those with an ESS score below 10 as asymptomatic [7]. This threshold was crossed with AHI severity category to identify symptomatic mild disease and asymptomatic severe disease. In addition, using the obstructive apnea index and hypopnea index recorded for each study, patients were classified as apnea-predominant (obstructive apnea index greater than hypopnea index) or hypopnea-predominant (hypopnea index greater than obstructive apnea index). Central and mixed apnea indices were reviewed separately and were not used for this classification. Because Level III recordings lack electroencephalography, hypopneas in this cohort have been scored only using a desaturation-based criterion without the arousal-based option, consistent with the $\geq 3\%$ threshold used for the oxygen desaturation index [8]. As an additional analysis, the STOP-BANG score, a screening questionnaire widely used to prioritise patients for sleep testing, was related to AHI and to moderate-to-severe OSA by Spearman correlation and ROC analysis, and standard risk categories (low 0–2, intermediate 3–4, high 5–8) were examined for a severity gradient, in order to test whether it retains discriminative value in a population already referred for suspected OSA.

As a further analysis, the AHI severity thresholds (mild 5–14.9, moderate 15–29.9, severe ≥ 30 events/h) were applied directly to ODI to generate an ODI-based severity category, which was compared with AHI-based severity category using exact agreement, linearly and quadratically weighted Cohen's kappa, and the sensitivity and specificity of ODI thresholds for AHI-defined moderate-to-severe (≥ 15) and severe (≥ 30) disease.

As a comparator to the pre-specified phenotypes, k-means clustering ($k = 2\text{--}6$, standardized variables, 25–50 restarts) was performed on BMI, oxygen desaturation index (ODI), minimum SpO_2 , obstructive apnea index and hypopnea index in patients with complete data on these five variables, to test whether an unsupervised, data-driven approach using physiological variables would separate patients along axes independent of AHI. Cluster number was chosen by silhouette score; stability was checked against a second k-means run with a different seed and against agglomerative clustering, using the adjusted Rand index.

Patients with a recorded central apnea index of 5 events/h or more were described separately, as a phenotypic observation potentially suggestive of a ventilatory-control contribution. This was not treated as a validated endotype measure, and no group comparisons were performed given the small subgroup size.

Each phenotype axis was analysed in the largest subset of patients with complete data for its defining variables; patients could contribute to more than one axis. Continuous variables are reported as mean $\pm$ standard deviation and compared with the Mann–Whitney U test; proportions are reported with Wilson 95% confidence intervals and compared with the chi-square test. Correlations used Spearman's ρ . The two headline comparisons of moderate-to-severe disease by body-habitus phenotype, and AHI by event-type phenotype were repeated in multivariable models (logistic and linear regression respectively) adjusted for age and sex, to test whether unadjusted group differences reflected these potential confounders. Given the number of comparisons made across three phenotype axes, reported p values are nominal and uncorrected for multiplicity, and the analysis as a whole should be read as hypothesis-generating rather than confirmatory. A two-sided p value below 0.05 was considered significant.

Results

Of 371 patients with a valid AHI, 309 (83.3%) had OSA (AHI $\geq$ 5): 109 mild, 79 moderate and 121 severe. The OSA cohort was 77.3% male (239/309), with a mean age of 56.2 ± 13.1 years ($n = 187$) and a mean BMI of 31.2 ± 6.2 kg/m² ($n = 300$); median AHI was 23.0 events/h (IQR 10.6–39.9). Median ODI was 15.8 events/h (IQR 6.1–3.5) and median minimum SpO₂ was 79% (IQR 67–85).

Lean vs. non-lean OSA

Among 300 OSA patients with a recorded BMI, 37 (12.3%; 95% CI 9.1–16.5) were lean (BMI < 25 kg/m²). Lean patients had lower AHI, less hypoxemic burden and fewer daytime symptoms than non-lean patients, with a similar age and sex distribution (Table 1). Moderate-to-severe disease was less frequent in lean patients (40.5% vs 68.4% of non-lean patients, $p = 0.002$).

After adjustment for age and sex, the lean phenotype remained independently associated with lower odds of moderate to severe disease (OR 0.26, 95% CI 0.11–0.65, $p = 0.004$), indicating this is not explained by lean patients being younger or more often female.

Symptom-severity discordance

Among 194 patients with an ESS score, 65 (33.5%) were symptomatic ($ESS \geq 10$). AHI correlated only weakly with ESS ($p = 0.27$, $p < 0.001$). Symptom status and severity were frequently discordant (Table 2): 39 of 74 patients with severe OSA (52.7%; 95% CI 41.5–63.7) were not sleepy, while 14 of 65 patients with mild OSA (21.5%; 95% CI 13.3–33.0) were. Sleepy patients had a greater hypoxemic burden (higher ODI, lower minimum SpO_2) than non-sleepy patients but a similar BMI, age and sex distribution (Figure 1).

Apnea-predominant versus hypopnea-predominant disease

Among 197 patients with usable obstructive apnea and hypopnea events, 107 (54.3%) were apnea-predominant. This group was younger and had a lower BMI than the hypopnea-predominant group, despite having a higher AHI (Figure 2); ESS and sex distribution did not differ. STOP-BANG score correlated only weakly with AHI ($p = 0.14$, $p = 0.069$) and discriminated moderate-to-severe disease poorly (AUC 0.56, 95% CI 0.48–0.64); 86.5% sensitivity at the standard cut-off of 3 or more came with only 20.7% specificity, and there was no significant gradient in OSA severity across the standard low/intermediate/high risk categories ($p = 0.36$). This likely reflects a ceiling effect. In a population already selected for a high pretest probability of OSA, most patients score in the intermediate to high STOP-BANG range regardless of eventual severity.

Hypertension was present in 27.5% and diabetes mellitus in 14.6% of the OSA cohort (Table 1). Neither prevalence differed between the lean and non-lean phenotypes (hypertension, $p = 0.84$; diabetes, $p = 0.87$), suggesting that lean OSA patients, despite milder polysomnographic severity, were not correspondingly free of cardiometabolic comorbidity in this cohort.

Among 322 patients with both AHI and ODI recorded, applying AHI's own severity thresholds to ODI gave exact category agreement in 67.4% and agreement within one category in 97.8%. Weighted kappa rose from 0.56 (unweighted) to 0.71 (linear) to 0.82 (quadratic), indicating that most disagreement was between adjacent categories rather than gross misclassification. ODI graded disease one or more categories lower than AHI in 19.3% of patients and higher in 13.4%. As a threshold test, $ODI \geq 15$ identified AHI-defined moderate-to-severe disease with 89% sensitivity and 92% specificity, and $ODI \geq 30$ identified AHI-defined severe disease with 76%

sensitivity and 94% specificity. Five patients had AHI ≥ 30 (range 30.2–50.7 events/h) with a disproportionately low ODI (0.3–9.8 events/h), illustrating that a minority of patients have frequent respiratory events without a matching degree of desaturation, so ODI-based grading alone would misclassify these patients as having milder disease.

A central apnea event was recorded in 160 of 309 OSA patients (median 0.95 events/h, IQR 0.4–2.3). In 22 of these patients (13.8%), the central apnea index was 5 events/h or more, a pattern described clinically as central-predominant sleep apnea; this subgroup was predominantly male (19/22) with a higher mean BMI (32.5 ± 6.1 kg/m²) and AHI (46.9 ± 23.1 events/h) than the OSA cohort overall. We report this as a phenotypic observation only. An elevated central apnea count is sometimes used as a coarse clinical indicator that ventilatory control instability (loop gain) may be contributing, but it is not a validated measure of loop gain, and should not be read as an endotype characterization. Group comparisons were not performed for this subgroup given its small size and the absence of a specific hypothesis.

Discussion

In this Indian tertiary referral cohort, three clinically defined phenotypes captured heterogeneity that AHI alone did not. These included a lean OSA subtype with milder, less hypoxemic disease; a substantial mismatch between symptom burden and severity in both directions; and an event-type distinction between apnea and hypopnea predominant disease with different demographic and anthropometric correlates.

The lean phenotype is consistent with reports that OSA occurs in non-lean individuals more often in Asian than in Western cohorts, attributed to craniofacial and upper-airway anatomical factors that operate independently of adiposity [1,2]. In our cohort this phenotype was, on average, milder rather than simply "OSA without obesity": lower AHI, less hypoxemia and lower symptom scores accompanied the lower BMI. This does not mean lean OSA is unimportant since over a third of lean patients still had moderate to severe disease, but it does suggest the mechanism, rather than the severity, is what differs.

The symptom-severity mismatch is the phenotype with the most direct clinical relevance. Over half of patients with severe OSA reported little daytime sleepiness, while roughly a fifth of patients with mild OSA reported clinically significant sleepiness. This mirrors the broader recognition that AHI and symptom burden are only loosely coupled [3] and that ESS alone should not be used to

decide who needs testing or treatment. Cultural under-reporting of sleepiness is a further possible contributor in this population since patients in South Asian clinical settings may minimise daytime symptoms for social or occupational reasons independent of true symptom burden, which would inflate the "asymptomatic severe" group beyond what a purely physiological explanation accounts for. This cannot be distinguished from genuine symptom resilience with the data available here. Sleepy patients in our cohort had greater hypoxemic burden, not greater obesity, suggesting that intermittent hypoxemia rather than event frequency or body habitus per se may drive perceived sleepiness which was consistent with the emphasis on hypoxic burden over AHI in recent literature [9] and with earlier reports linking nocturnal hypoxemia to excessive daytime sleepiness [10] and impaired vigilance [11].

The apnea-predominant phenotype was unexpected in direction: these patients were younger, and leaner than the hypopnea-predominant group, despite more severe disease by AHI. Hypopnea events, but not obstructive apnea events, correlated with BMI, suggesting hypopnea-predominant events may be more closely tied to soft-tissue loading of the airway, while apnea-predominant events in younger, leaner patients may reflect greater airway collapsibility or neuromuscular factors independent of adiposity [4]. This pattern held after adjustment for age and BMI, arguing against simple confounding by these variables, though it remains a single-cohort, hypothesis-generating observation given the Level III setting. A competing, non-mechanistic explanation is also plausible: younger patients may present later in the course of disease, whether through lower perceived risk, later recognition of symptoms by themselves or their families, or referral patterns particular to this centre, so that apnea-predominant patients are younger not because of a different airway mechanism but because they reach diagnosis with more advanced, if differently patterned, disease. The two explanations are not mutually exclusive and cannot be separated without longitudinal or drug-induced sleep endoscopy data.

A minority of patients had a central apnea index of 5 events/h or more, a pattern that is clinically recognizable but that we deliberately describe as a phenotype observation rather than an endotype finding. True endotyping requires quantifying loop gain, collapsibility (Pcrit), arousal threshold and upper-airway muscle responsiveness using either a CPAP dial-down protocol or validated analysis of raw flow and effort waveforms, and typically EEG-defined arousals [12,13]; none of these are available from routine Level III summary data. We raise this distinction explicitly

because the terms phenotype and endotype are sometimes used loosely in the OSA literature, and a central apnea count, however suggestive, is not a substitute for a measured physiological trait. STOP-BANG performed poorly as a severity discriminator in this cohort, in contrast to its validated performance in general surgical and community populations [14,15]. This is best read as a ceiling effect of applying a screening tool to a population already selected for a high pretest probability of OSA, rather than evidence against STOP-BANG itself; its role here, if any, would be in the primary-care or community settings for which it was designed, not in a tertiary sleep referral population. The similar hypertension and diabetes prevalence in lean and non-lean phenotypes also argues against using body habitus alone to triage patients for cardiometabolic risk assessment, a finding consistent with evidence that sleep-disordered breathing itself, independent of obesity, contributes to incident hypertension and glucose intolerance [16,17].

Grading ODI on AHI's own thresholds agreed with AHI category reasonably well overall (quadratic-weighted kappa 0.82), and performed well as a binary screen for moderate-to-severe disease (89% sensitivity, 92% specificity). This supports oximetry-only indices as a reasonable triage step where full Level III testing capacity is limited, consistent with evidence that ODI and hypoxic burden track cardiovascular outcomes in their own right [9]. However, the five patients with severe AHI but a low ODI are a clinically important exception: frequent respiratory events without proportionate desaturation would be missed, or under-graded, by ODI alone. An oximetry-only pathway in this population should therefore be positioned as a triage step that can rule disease in, not a substitute for event-based testing when ruling disease out, particularly in patients with a high clinical suspicion despite reassuring oximetry, in keeping with reports that oximetry-derived ODI identifies moderate-to-severe OSA accurately but may under-detect events that are not accompanied by desaturation [18,19].

Our unsupervised analysis can also be set against previously published cluster analyses. Most have clustered clinical variables such as symptoms, comorbidity and sleepiness together with sleep-study data: symptom-based subtypes have been described [20], as have four groups differing in age, sex, comorbidity and severity in a Spanish cohort [21], which included a non-obese group with milder disease that parallels our lean phenotype. A cluster analysis restricted to polysomnographic variables, as ours was, identified three groups, one characterised by obstructive-apnea predominance, that did not fully map onto AHI categories [22]. Because our clustering used only physiological variables strongly correlated with AHI, and did not include

symptoms or comorbidity, it is unsurprising that it recovered severity rather than the a priori clinical phenotypes; the two approaches are better seen as complementary than as competing.

Limitations

This is a retrospective, single-centre analysis of a referred high-risk population, so phenotype frequencies describe this cohort's diagnostic yield rather than community prevalence, and referral bias may inflate or deflate any of the three subgroups. Each phenotype axis used a different, incomplete subset of patients with the relevant variables recorded (BMI in 300, ESS in 194, apnea/hypopnea indices in 197), so the three analyses are not fully overlapping and some estimates, particularly for the lean and apnea-predominant subgroups, are imprecise. ESS captures subjective sleepiness only and does not represent the full range of OSA-related symptoms or quality of life; other recognized symptom subtypes (for example, insomnia-related presentations) could not be assessed. Level III studies lack electroencephalography, so arousal-based hypopnea scoring and sleep-stage-specific analysis were not possible, and central and mixed apnea events were too sparse to support formal statistical comparison, though their frequency is described. No in-laboratory polysomnography comparison was available, and no validation study specific to the SOMNOtouch RESP device against polysomnography was identified for this analysis; general Level 3 validation evidence was relied on instead. The lean-phenotype association and the apnea-predominant AHI difference were corroborated in age and sex adjusted models, which partly mitigates this concern for those two results specifically.

Conclusions

In this Indian tertiary referral cohort, obstructive sleep apnea separated into a lean phenotype with generally milder disease, a substantial symptom-severity discordance in both directions, and an apnea-predominant subtype occurring in younger, leaner patients with more severe disease than hypopnea-predominant disease. These patterns were not recovered by unsupervised clustering on physiological variables alone, indicating they arise from clinically meaningful, non-redundant dimensions rather than from AHI severity restated. Clinical assessment of OSA in similar settings should not rely on AHI or symptom score alone, and lean and minimally symptomatic patients with severe disease warrant particular attention. STOP-BANG score added little discriminative value once patients had already reached this referral population, and hypertension and diabetes

were equally prevalent regardless of body habitus, reinforcing that neither a screening score nor BMI alone should guide clinical risk assessment at this stage of the diagnostic pathway. ODI tracked AHI severity well overall and may support oximetry-based triage where full testing capacity is limited, provided clinicians remain alert to the minority of patients whose event frequency outpaces their desaturation burden. In practice, describing OSA by body habitus, symptom–severity relationship and event type alongside the AHI can sharpen bedside decisions: lean patients, and patients with severe disease but little sleepiness, should still be tested, risk-assessed and offered treatment rather than being deprioritized on the basis of BMI or ESS alone. Reporting oximetric indices (ODI, minimum SpO₂) and the apnea- versus hypopnea-predominant pattern next to the AHI may help flag patients with a heavier hypoxemic burden who merit closer follow-up, and sets the stage for phenotype-directed therapy. As these findings are hypothesis-generating and come from a single referral center, they need confirmation in larger prospective cohorts before they alter treatment pathways.

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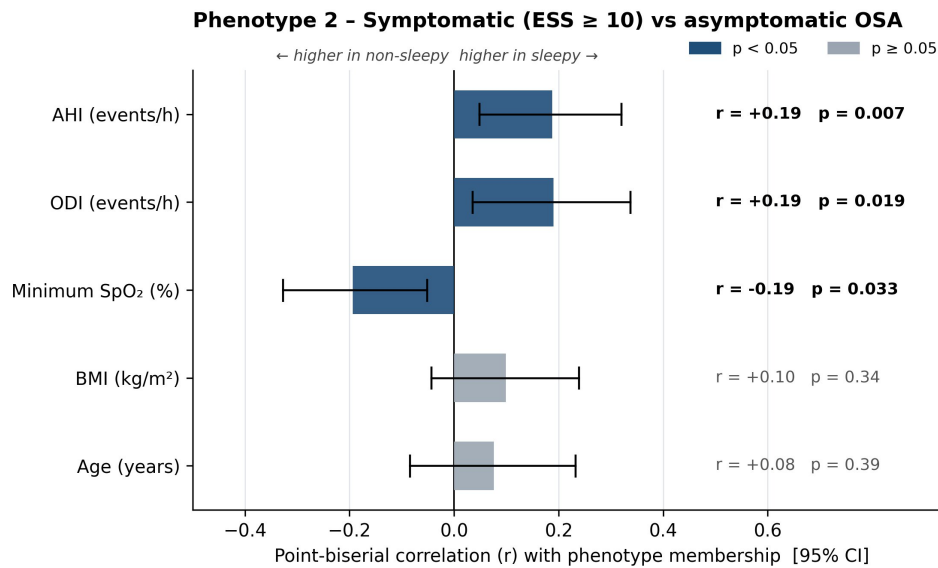


Figure 1. Point-biserial correlation of physiological and anthropometric variables with the symptom-severity phenotype. The plot illustrates the correlation (r) and 95% confidence intervals for variables distinguishing symptomatic from asymptomatic OSA. Statistically significant correlations ($p < 0.05$) are shown in dark blue, while non-significant correlations are in grey. Values extending to the right indicate higher measures in sleepy patients, while values to the left indicate higher measures in non-sleepy patients.

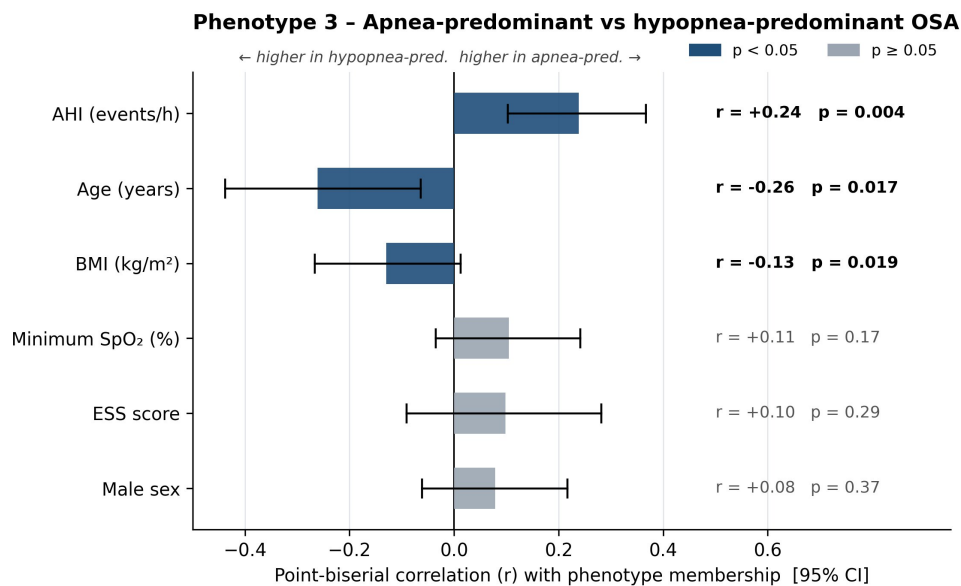


Figure 2. Point-biserial correlation of physiological and anthropometric variables with the event-type phenotype. The plot illustrates the correlation (r) and 95% confidence intervals for variables distinguishing apnea-predominant from hypopnea-predominant OSA. Statistically significant correlations ($p < 0.05$) are shown in dark blue, while non-significant correlations are in grey. Values extending to the right indicate higher measures in apnea-predominant patients, while values to the left indicate higher measures in hypopnea-predominant patients.

Table 1. Lean vs. non-lean OSA.

Variable	Lean (n=37)	Non-lean (n=263)	p
Age (years)	55.9±18.8 (n=24)	56.1±12.0 (n=160)	0.84
Male sex, n (%)	29 (78.4)	203 (77.2)	1.00
AHI, events/h	16.8±12.3	30.2±22.3	< 0.001
ODI, events/h	12.7±12.3 (n=30)	30.1±23.1 (n=225)	< 0.001
Minimum SpO ₂ (%)	81.8±9.9	70.3±15.7 (n=256)	< 0.001
ESS score <10	26 (83.9%)	137 (65.9%)	
ESS score ≥10	5 (16.1%)	71 (34.1%)	
ESS Score	4.8±3.5 (n=31)	8.5±5.1 (n=239)	<0.001
Moderate to severe OSA (%)	40.5	68.4	0.002

Table 2. AHI severity by symptom status (ESS), n=194.

Severity	Not sleepy (ESS < 10)	Sleepy (ESS ≥ 10)	%
Mild	51	14	21.5
Moderate	39	16	29.1
Severe	39	35	47.3