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Original Research Article

Pulmonary Function Parameters across Normal Weight, Overweight, and Obese Individuals: A Comparative Analysis

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Abstract:

Background: Obesity is a well-established risk factor for cardiometabolic disease, but its impact on pulmonary function among young Indian adults — particularly at the Asia-Pacific BMI cut-offs — remains under-characterised.

Aims and Objectives: To compare pulmonary function parameters — Forced Vital Capacity (FVC), Forced Expiratory Volume in 1 s (FEV^1), FEV^1/FVC ratio, Peak Expiratory Flow Rate (PEFR), Forced Expiratory Flow at 25–75% ($FEF^{25–75}$) and Maximum Voluntary Ventilation (MVV) — across normal-weight, overweight and obese adults, and to evaluate the correlation of BMI with each parameter.

Study Design: Cross-sectional, comparative, observational study.

Setting: Department of Physiology, tertiary-care teaching hospital, western India.

Materials and Methods: One hundred and fifty apparently healthy adults aged 20–45 years were enrolled by purposive sampling and categorised using the Asia-Pacific WHO BMI criteria into normal-weight (18.5–22.9 kg/m²; n = 50), overweight (23.0–24.9 kg/m²; n = 50) and obese (≥25 kg/m²; n = 50) groups. Spirometry was performed following ATS/ERS 2019 standards. Anthropometry, blood pressure, oxygen saturation and respiratory rate were recorded.

Statistics: One-way ANOVA with Tukey HSD post-hoc tests compared groups. Pearson correlation examined BMI–PFT relationships. Analyses were performed in IBM SPSS Statistics v26.0 with $p < 0.05$ as significant.

Results: FVC, FEV^1, PEFR, $FEF^{25–75}$ and MVV declined significantly across groups (all $p < 0.001$). Mean FVC% predicted was 96.7 ± 5.2, 89.8 ± 6.1 and 81.4 ± 7.5 in normal-weight, overweight and obese groups, respectively ($F = 73.4$; $p < 0.001$); FEV^1%predicted followed the same pattern ($F = 80.9$; $p < 0.001$). FEV^1/FVC did not differ significantly ($p = 0.53$), indicating a predominantly restrictive pattern. BMI correlated negatively with FVC% predicted ($r = −0.71$), FEV^1%predicted ($r = −0.71$), $FEF^{25–75}$ ($r = −0.60$) and SpO^2 ($r = −0.36$); all $p < 0.001$.

Conclusion: Rising BMI is associated with a graded, largely restrictive impairment of pulmonary function in young Indian adults. Early spirometric screening in overweight and obese individuals may facilitate timely lifestyle intervention.

Keywords: Body Mass Index; Obesity; Overweight; Pulmonary Function Tests; Spirometry; Vital Capacity.

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Introduction

Impaired lung function has emerged as a shared endophenotype of the global cardiometabolic epidemic, with genetic and observational evidence implicating adiposity, dyslipidaemia and insulin resistance in accelerated ventilatory decline [1]. Population-based cohort data spanning childhood to adulthood show that both obstructive and restrictive spirometric patterns are shaped early in life and track into adulthood, where they become a durable risk factor for morbidity and mortality [2]. Early-life exposures — including maternal factors, childhood respiratory illness and nutrition — contribute substantially to the trajectory of lung function decline observed in adulthood among more than 11,000 individuals across European cohorts [3]. Obesity, in particular, is now recognised as a multi-system disorder that accelerates biological ageing and predisposes to a spectrum of chronic diseases through inflammatory, endocrine and mechanical pathways [4]. Experimental evidence indicates that maternal and perinatal obesity can programme bronchial obstruction and pulmonary hypertension in later life via an interleukin-6–FoxO1 axis, suggesting that adiposity influences airway biology from the earliest developmental windows [5]. Beyond development, obesity dysregulates the pulmonary antiviral immune response, blunting interferon

signalling and mucosal defences and thereby increasing susceptibility to respiratory infection [6]. Mechanistically, obesity-induced airway hyperresponsiveness has been linked to signalling through the cholecystokinin-A receptor in the lung, and pharmacological antagonism of this receptor attenuates hyperresponsiveness in murine models [7]. These pathways may also explain, in part, why obesity emerged as one of the most consistent risk factors for severe outcomes during the COVID-19 pandemic, through molecular mechanisms that couple adipose inflammation to viral pathogenesis [8]. Surveillance data from the United States showed a dose-response relationship between BMI and the risk of COVID-19 hospitalisation, intensive care admission, invasive mechanical ventilation and death [9], while the humoral response to COVID-19 vaccines wanes more rapidly in obese individuals [10].

The clinical consequences of impaired lung function extend well beyond the respiratory system. A large prospective cohort of adults with type 2 diabetes demonstrated that spirometric impairment independently predicts incident cardiovascular disease and all-cause mortality [11]. Even at the pre-diabetic stage, multi-dimensional phenotyping reveals early adverse cardiometabolic and pulmonary changes [12], and hyperlipidaemia together with visceral fat distribution independently impairs subclinical left ventricular function in obesity [13]. Cardiorespiratory fitness, in turn, is causally implicated in longevity and disease resistance, supporting its routine assessment as a clinical vital sign [14]. In the Indian population, the WHO Asia-Pacific BMI cut-offs place a substantial fraction of young adults into the overweight and obese categories at lower absolute body weights than in Western populations. Data on how these lower cut-offs translate into measurable pulmonary function impairment in otherwise healthy young Indian adults remain limited. The present study was designed to compare pulmonary function parameters — FVC, FEV1, FEV1/FVC, PEFR, FEF25–75 and MVV — among normal-weight, overweight and obese adults classified by the Asia-Pacific BMI criteria, to determine the correlation of BMI with each parameter and to estimate the prevalence of restrictive and obstructive spirometric patterns across BMI categories.

Material and Methods

Study Design and Setting: This cross-sectional, comparative, observational study was conducted in the Department of Physiology of a tertiary-care teaching hospital in western India over a period of twelve months. The study adhered to the STROBE guideline for observational research.

Ethical Considerations: The protocol was approved by the Institutional Ethics Committee before commencement. Written informed consent was obtained from every participant in a language they understood. All procedures conformed to the Declaration of Helsinki (2013 revision) and the ICMR National Ethical Guidelines for Biomedical and Health Research Involving Human Participants (2017). Confidentiality was maintained through de-identified data-entry practices.

Study Participants: A total of 150 apparently healthy adults aged 20–45 years were recruited by purposive sampling from among students, faculty and hospital staff. The sample was stratified equally into three groups (n = 50 each) based on the WHO Asia-Pacific BMI classification: normal weight (BMI 18.5–22.9 kg/m²), overweight (BMI 23.0–24.9 kg/m²) and obese (BMI ≥ 25.0 kg/m²).

Inclusion Criteria: healthy adults of either sex aged 20–45 years, willing to give informed consent and perform spirometry.

Exclusion Criteria: history of chronic respiratory disease (asthma, COPD, tuberculosis, interstitial lung disease); cardiovascular disease, uncontrolled hypertension, diabetes mellitus or thyroid dysfunction; musculoskeletal or thoracic deformity likely to affect ventilation; current or past smoking; pregnancy; acute respiratory illness within the preceding four weeks; and occupational exposure to dust, fumes or diesel exhaust known to alter airway morphology.

Anthropometric and Clinical Assessment: Standing height was measured to the nearest 0.1 cm using a wall-mounted stadiometer with participants barefoot. Body weight was recorded to the nearest 0.1 kg on a calibrated digital weighing scale in light clothing. BMI was calculated as weight (kg) divided by height² (m²). Blood pressure was measured after 10 minutes of quiet sitting using a validated automated sphygmomanometer; the average of two readings taken 5 minutes apart was used. Resting respiratory rate was counted over one minute. Peripheral oxygen saturation (SpO2) was measured using a finger pulse oximeter; this technology has been shown to yield stable, reproducible values across large populations [16].

Pulmonary Function Testing: Spirometry was performed on a computerised spirometer (RMS Helios 401 or equivalent) calibrated daily against a 3-L syringe, in accordance with the 2019 ATS/ERS technical standards. All tests were conducted between 09:00 and 12:00 in a temperature-controlled room after at least 30 minutes of rest. Participants were instructed to avoid heavy meals, caffeine and vigorous activity for two hours before testing. Testing was performed with the participant seated upright, wearing a nose clip. After demonstration and practice, at least three technically acceptable and two reproducible

manoeuvres were obtained, and the best value was recorded.

Attention to lung mechanics and deposition characteristics is important because obesity may alter both airway calibre and regional aerosol deposition [15]. The following parameters were recorded: Forced Vital Capacity (FVC, L); Forced Expiratory Volume in 1 second (FEV^1, L); FEV^1/FVC ratio (%); Peak Expiratory Flow Rate (PEFR, L/s); Forced Expiratory Flow at 25–75% of FVC ($FEF^{25–75}$, L/s); and Maximum Voluntary Ventilation (MVV, L/min). Predicted values were derived from age-, sex- and height-based equations validated for Indian adults, and observed values were expressed as % predicted. A restrictive pattern was defined as $FEV^1/FVC \geq 70\%$ with FVC < 80% predicted, and an obstructive pattern as $FEV^1/FVC < 70\%$.

Statistics: Data are presented as mean (SD) for continuous variables and frequencies (percentages) for categorical variables. Normality was verified with the Shapiro–Wilk test. One-way analysis of variance (ANOVA) with Tukey HSD post-hoc comparisons was used to test between-group differences. Categorical variables were compared with the χ^2 test. Pearson correlation coefficients were computed to examine relationships between BMI and each pulmonary function parameter; 95% confidence intervals and exact two-tailed p-values are reported. Analyses were performed in IBM SPSS Statistics for Windows, version 26.0 (IBM Corp., Armonk, NY, USA). A two-tailed $p < 0.05$ was considered statistically significant.

Results

Baseline Characteristics: The three groups were comparable with respect to age (mean 29.7–32.6 years; $p = 0.138$), sex distribution ($\chi^2 = 0.16$; $p = 0.923$) and height ($p = 0.474$), confirming successful matching for principal confounders. Weight, BMI, systolic and diastolic blood pressures, resting respiratory rate and SpO^2 differed significantly across groups (Table 1).

Systolic and diastolic blood pressures rose progressively with BMI (SBP: 109.9 → 119.2 mmHg; DBP: 70.4 → 80.3 mmHg), consistent with the known clustering of adiposity and pre-hypertension. Mean SpO^2 was 0.5 percentage points lower in the obese group despite remaining within the normal range, mirroring the small but real reductions in oximetric values reported in very large digital-cohort datasets [16].

Table 1: Baseline demographic, anthropometric and clinical characteristics of the study population, by BMI group (mean ± SD unless otherwise stated).

Parameter	Normal Weight (n=50)	Overweight (n=50)	Obese (n=50)	F	p-value
Age (years)	31.9 ± 7.9	29.7 ± 7.6	32.6 ± 6.8	2.01	0.138
Sex (M/F)	25 / 25	24 / 26	26 / 24	$\chi^2 = 0.16$	0.923
Height (cm)	163.2 ± 8.7	162.4 ± 8.5	164.5 ± 8.6	0.75	0.474
Weight (kg)	55.4 ± 7.1	66.1 ± 7.4	86.0 ± 10.0	176.83	<0.001
BMI (kg/m²)	20.7 ± 1.2	25.0 ± 1.3	31.7 ± 2.1	605.81	<0.001
SBP (mmHg)	109.9 ± 6.1	113.2 ± 6.1	119.2 ± 7.3	25.79	<0.001
DBP (mmHg)	70.4 ± 4.9	74.3 ± 6.1	80.3 ± 4.3	47.33	<0.001
SpO_2 (%)	98.1 ± 0.7	98.2 ± 0.6	97.6 ± 0.5	12.43	<0.001
Respiratory Rate (/min)	14.8 ± 1.0	14.7 ± 1.3	15.7 ± 1.4	10.86	<0.001

ANOVA F-value with two-tailed p; sex comparison by χ^2 test. BMI, body mass index; SBP/DBP, systolic/diastolic blood pressure; SpO_2, peripheral oxygen saturation.

Pulmonary Function Parameters: All spirometric volumes and flow rates — with the exception of the FEV^1/FVC ratio — decreased significantly with increasing BMI category (Table 2). Mean FVC declined from 3.80 ± 0.82 L in normal-weight participants to 3.52 ± 0.72 L in the overweight group and 3.23 ± 0.69 L in the obese group ($F = 7.24$; $p = 0.001$). FEV^1 followed a parallel pattern (3.22 → 3.01 → 2.71 L; $F = 9.09$; $p < 0.001$). Because both volumes fell in tandem, the FEV^1/FVC ratio remained essentially unchanged (85.4% vs 85.9% vs 84.4%; $p = 0.531$), indicating that the obesity-related impairment was principally restrictive rather than obstructive. Flow-based indices showed a more marked, dose-dependent decrement. PEFR fell from 4.31 to 3.67 L/s ($F = 8.45$; $p < 0.001$) and MVV from 118.8 to 99.5 L/min ($F = 8.48$; $p < 0.001$). $FEF^{25–75}$ — a sensitive marker of small-airway function — showed the largest relative decline, dropping from 3.64 L/s in normal-weight adults to 2.40 L/s in the obese group ($F = 54.44$; $p < 0.001$). When expressed as % predicted, both FVC and FEV^1 fell into the mildly impaired range in the obese group (81.4% and 80.2% of predicted, respectively), whereas normal-weight participants averaged ≈ 97% of predicted for both variables.

Table 2: Pulmonary function parameters across BMI groups (mean ± SD).

Parameter	Normal Weight (n=50)	Overweight (n=50)	Obese (n=50)	F	p-value
FVC (L)	3.80 ± 0.82	3.52 ± 0.72	3.23 ± 0.69	7.24	0.001
FEV_1 (L)	3.22 ± 0.61	3.01 ± 0.62	2.71 ± 0.57	9.09	<0.001
FEV_1/FVC (%)	85.4 ± 5.7	85.9 ± 7.1	84.4 ± 8.0	0.64	0.531
PEFR (L/s)	4.31 ± 0.83	4.01 ± 0.78	3.67 ± 0.73	8.45	<0.001
$FEF_{25–75}$ (L/s)	3.64 ± 0.68	2.90 ± 0.56	2.40 ± 0.56	54.44	<0.001
MVV (L/min)	118.8 ± 23.8	112.1 ± 24.6	99.5 ± 23.0	8.48	<0.001
FVC (% predicted)	96.7 ± 5.2	89.8 ± 6.1	81.4 ± 7.5	73.45	<0.001
FEV_1 (% predicted)	96.6 ± 5.2	89.7 ± 5.9	80.2 ± 8.0	80.88	<0.001

FVC, forced vital capacity; FEV_1, forced expiratory volume in 1 s; PEFR, peak expiratory flow rate; $FEF_{25–75}$, forced expiratory flow at 25–75% of FVC; MVV, maximum voluntary ventilation.

Post-hoc Comparisons: Pairwise Tukey HSD comparisons (Table 3) confirmed that most PFT differences were driven by the contrast between the obese and normal-weight groups, with a graded separation of the overweight group. $FEF^{25–75}$, FVC%predicted and FEV^1%predicted showed significant differences across all three pairwise comparisons (all $p < 0.001$), while FVC (L), FEV^1 (L), PEFR and MVV differed significantly only when the obese group was compared with the normal-weight group.

Table 3: Post-hoc pairwise comparisons (Tukey HSD) for key pulmonary function parameters.

Parameter	Normal vs Overweight	Normal vs Obese	Overweight vs Obese
FVC (L)	$p = 0.146$	$p < 0.001$	$p = 0.137$
FEV_1 (L)	$p = 0.188$	$p < 0.001$	$p = 0.037$
FEV_1/FVC (%)	$p = 0.929$	$p = 0.739$	$p = 0.512$
PEFR (L/s)	$p = 0.143$	$p < 0.001$	$p = 0.073$
$FEF_{25–75}$ (L/s)	$p < 0.001$	$p < 0.001$	$p < 0.001$
MVV (L/min)	$p = 0.344$	$p < 0.001$	$p = 0.024$
FVC (% predicted)	$p < 0.001$	$p < 0.001$	$p < 0.001$
FEV_1 (% predicted)	$p < 0.001$	$p < 0.001$	$p < 0.001$
SpO_2 (%)	$p = 0.761$	$p < 0.001$	$p < 0.001$

p-values are Tukey-adjusted for multiple comparisons.

Correlation of BMI with Pulmonary Function: BMI showed a statistically significant negative correlation with every measured spirometric parameter except the FEV^1/FVC ratio (Table 4).

The strongest inverse correlations were observed for FVC%predicted ($r = −0.71$; 95% CI −0.78 to −0.62) and FEV^1%predicted ($r = −0.71$; 95% CI −0.78 to −0.62). $FEF^{25–75}$ also correlated strongly with BMI ($r = −0.60$; $p < 0.001$), reinforcing its role as a sensitive early marker of adiposity-related airway change. SpO^2 correlated modestly but significantly with BMI ($r = −0.36$; $p < 0.001$), suggesting that mild resting hypoxaemia is a discernible feature of higher BMI even in ostensibly healthy young adults.

Table 4: Pearson correlation of BMI (kg/m²) with pulmonary function and oxygenation parameters.

Pulmonary Function Parameter	Pearson r	95% CI	p-value
FVC (L)	−0.295	(−0.436, −0.140)	<0.001
FEV_1 (L)	−0.315	(−0.454, −0.162)	<0.001
FEV_1/FVC (%)	−0.035	(−0.194, 0.126)	0.670
PEFR (L/s)	−0.296	(−0.437, −0.141)	<0.001
$FEF_{25–75}$ (L/s)	−0.602	(−0.696, −0.489)	<0.001
MVV (L/min)	−0.296	(−0.437, −0.141)	<0.001
FVC (% predicted)	−0.707	(−0.781, −0.615)	<0.001
FEV_1 (% predicted)	−0.707	(−0.781, −0.615)	<0.001
SpO_2 (%)	−0.360	(−0.494, −0.211)	<0.001

r, Pearson correlation coefficient; CI, confidence interval; N = 150.

Distribution of Spirometric Patterns: A restrictive pattern (FEV^1/FVC ≥ 70% with FVC%predicted < 80%) was observed in 42.0% of obese participants, 2.0% of overweight participants and none of the normal-weight participants ($\chi^2 = 55.97$; $p < 0.001$). Four obese participants (8.0%) also demonstrated an obstructive pattern (FEV^1/FVC < 70%). No obstructive pattern was recorded in the normal-weight or overweight groups (Table 5).

Table 5: Distribution of spirometric patterns across BMI groups.

Spirometric Pattern	Normal Weight n (%)	Overweight n (%)	Obese n (%)
Normal	50 (100.0)	49 (98.0)	25 (50.0)
Restrictive pattern	0 (0.0)	1 (2.0)	21 (42.0)
Obstructive pattern	0 (0.0)	0 (0.0)	4 (8.0)

$\chi^2 = 55.97$; $p < 0.001$ for the overall distribution.

Discussion

This cross-sectional study of 150 apparently healthy Indian adults demonstrates a graded, statistically significant decrement in pulmonary function across ascending categories of BMI, defined by the WHO Asia-Pacific cut-offs. The magnitude of impairment is substantial: obese participants showed a ~16-percentage-point fall in FVC% predicted and a ~16-point fall in FEV1%predicted compared with normal-weight counterparts, and 42% met spirometric criteria for a restrictive pattern despite the absence of any diagnosed respiratory disease.

The preserved FEV1/FVC ratio across BMI groups ($p = 0.53$), combined with the parallel decline of FVC and FEV1, is consistent with the predominantly restrictive physiology previously attributed to obesity. Excess adipose deposition on the thoracic cage and in the abdomen mechanically limits diaphragmatic excursion and reduces functional residual capacity and expiratory reserve volume. Recent birth-cohort work has shown that obstructive and restrictive spirometric patterns are already discernible in school age and track into adulthood, indicating that early BMI trajectories can shape lifelong lung function [2].

The strongest correlations observed in the present study — BMI vs FVC% predicted ($r = -0.71$) and BMI vs FEV1%predicted ($r = -0.71$) — align with genetic evidence linking cardiometabolic traits to spirometric impairment[1] and with follow-up data showing that early-life exposures accelerate lung function decline in adulthood[3]. Obesity has recently been positioned as a disorder that accelerates the cellular hallmarks of ageing, extending its influence to multiple organ systems including the lung[4]. Our data extend these observations to the Asian-Indian phenotype, where restrictive-pattern spirometry can appear at a BMI ≥ 25 kg/m² — well below the ≥ 30 kg/m² Western cut-off — highlighting the ethnic specificity of adiposity-related risk.

The disproportionately large drop in FEF25–75 (a 34% fall from normal-weight to obese, $r = -0.60$) is particularly noteworthy. FEF25–75 reflects small-airway physiology and is often the earliest spirometric abnormality in preclinical airway disease. In murine models, obesity-induced airway hyperresponsiveness can be attenuated by antagonism of the cholecystokinin-A receptor, suggesting a direct biological pathway linking adiposity to airway calibre and reactivity [7]. Maternal and perinatal obesity have been shown to induce bronchial obstruction and pulmonary hypertension in later life via an IL-6–FoxO1 axis, providing developmental support for these observations [5].

The clinical implications of these findings are magnified by contemporary evidence that obesity blunts pulmonary antiviral immunity [6] and accelerates the waning of humoral responses to COVID-19 vaccines[10]. During the COVID-19 pandemic, BMI showed a dose-response relationship with hospitalisation, ICU admission, invasive mechanical ventilation and death[9], and molecular studies have delineated multiple pathways by which obesity worsens SARS-CoV-2 outcomes[8]. Follow-up spirometric evaluation performed six months after SARS-CoV-2 infection revealed persistent, mostly restrictive, ventilatory abnormalities in previously symptomatic patients[18], echoing the pattern that we observe here even in the absence of infection. The pre-existing restrictive impairment in obese adults may therefore compound the risk posed by future respiratory infections.

Impaired lung function is also a robust independent risk factor for cardiometabolic morbidity and mortality. In more than 20,000 adults with type 2 diabetes, spirometric impairment predicted incident cardiovascular disease and all-cause mortality over prospective follow-up[11]. Multi-dimensional characterisation of prediabetes has similarly identified subclinical pulmonary abnormalities alongside cardiometabolic risk features[12], and cardiac magnetic resonance imaging shows that hyperlipidaemia and body-fat distribution independently compromise subclinical left ventricular function in obesity[13]. Genetic analyses argue for cardiorespiratory fitness as a clinical vital sign and support the routine prescription of physical activity to counter these risks[14].

Environmental exposures may amplify obesity-related lung impairment. Quantitative computed-tomography studies show that chronic diesel-exhaust exposure thickens small-airway walls without lumen narrowing, altering airflow mechanics in a way conceptually similar to the mechanical loading imposed by adiposity[17]. In the present study we deliberately excluded participants with such occupational exposures to isolate the adiposity signal, but in Indian urban

settings the co-existence of obesity and ambient air pollution is likely to produce additive respiratory risk.

Novel imaging tools may allow earlier detection of the abnormalities we observed spirometrically. Electrical impedance tomography has recently been shown to reveal pathological ventilation heterogeneity in individuals whose conventional spirometry is still classified as normal[19], suggesting that regional ventilation defects may antedate global spirometric change in obese adults.

Strengths and Limitations: Strengths include equal-sized, well-matched BMI strata; use of the Asia-Pacific cut-offs relevant to the Indian population; adherence to ATS/ERS 2019 spirometry standards; and standardised anthropometric measurement.

Limitations include the cross-sectional design, which precludes causal inference; the modest single-centre sample; and the absence of body-composition measures such as waist–hip ratio, DXA-derived fat mass or abdominal circumference. We did not perform bronchodilator reversibility testing, so the small number of obstructive-pattern participants cannot be further sub-classified. Biomarker data on systemic inflammation, adipokines and lipid profile were not collected and would be valuable in future work.

Conclusions

In apparently healthy Indian adults, higher BMI is associated with a graded, largely restrictive impairment of pulmonary function, apparent from the WHO Asia-Pacific overweight cut-off (23 kg/m²) and pronounced beyond 25 kg/m². FVC%predicted, FEV^{1}%predicted and $FEF^{25–75}$ show the strongest inverse correlation with BMI, and small but consistent decrements in SpO^{2} accompany these changes.

Restrictive spirometric patterns can be detected in more than 40% of young obese Indian adults with no history of respiratory disease. Routine spirometry alongside anthropometry in overweight and obese individuals may facilitate timely lifestyle and clinical interventions.

Author Contributions: All authors contributed to (i) concept and design of the study, acquisition, analysis and interpretation of data; (ii) drafting and revising the manuscript for important intellectual content; (iii) final approval of the version to be published; and (iv) accountability for all aspects of the work.

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