New Approaches in Exercise Physiology (NAEP)
Vol. 6, No.12, December 2024
www.nass.atu.ac.ir
DOI: 10.22054/nass.2025.88456.1186

ATU PRESS

Original Research
Received: August 13, 2025 Accepted: September 30, 2025

Comparative Effects of Physical Activity on Respiratory Function in Young Adult Males: A Study of Sleep Quality Relationships

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How to Cite: Abdollahzadeh, N, Yaghoob Nezhad, F., Azadpour, N., & Massart, A. (2024). Comparative Effects of Physical Activity on Respiratory Function in Young Adult Males: A Study of Sleep Quality Relationships, *Journal of New Approaches in Exercise Physiology*, 6(12), 59-82.
DOI: 10.22054/nass.2025.88456.1186

Abstract

Purpose: This study aimed to compare respiratory parameters and sleep quality between physically active and inactive young adult males, while exploring correlations between these domains. **Method:** In a semi-experimental design, 15 active males (aged 20–23 years; ≥8 hours/week moderate-to-vigorous activity; >2 years sports experience) and 15 inactive males (≤3 hours/week activity; no sports experience) were recruited. Anthropometric measures (height, weight, BMI, body fat percentage, heart rate) were assessed. Pulmonary function—including forced vital capacity (FVC), vital capacity (VC), maximum voluntary ventilation (MVV), forced expiratory volume in one second (FEV1), percentage predicted FEV1 (%FEV1), and maximum expiratory flows at 25% and 75% of FVC (MEF25%, MEF75%)—was evaluated via spirometry (Fukuda ST-95) per American Thoracic Society guidelines. Sleep quality was quantified using the Pittsburgh Sleep Quality Index (PSQI). Independent t-tests compared groups; Pearson correlations and multiple linear regressions examined associations ($\alpha = 0.05$).

Results: Active participants displayed superior respiratory metrics ($p < 0.05$): higher FVC ($p = 0.023$), VC ($p = 0.002$), MVV ($p = 0.001$), FEV1 ($p = 0.001$), %FEV1 ($p = 0.001$), MEF25% ($p = 0.026$), and MEF75% ($p = 0.042$). PSQI scores were significantly lower (better) in the active group (4.13 ± 1.18) versus inactive (6.53 ± 2.50; $p = 0.002$). No baseline differences emerged in age, height, weight, heart rate, fat percentage, or BMI ($p > 0.05$). In the active group, each 1-unit increase in FEV1, MVV, VC, FVC, and MEF75% was associated with corresponding reductions in sleep quality scores of 0.217, 0.127, 0.370, 0.386, and 0.194 units, respectively (all $p > 0.05$). Regressions and correlations between respiratory indices and sleep quality were non-significant in both groups. **Conclusion:** Regular physical activity enhances sleep quality and respiratory function in young males, though direct mechanistic links were not evident in this cohort. These findings advocate exercise as a non-pharmacological strategy for addressing sleep and pulmonary health, warranting larger, diverse studies to elucidate interactions.

Keywords: physical activity, respiratory function, spirometer, Pittsburgh Sleep Quality Index.

Introduction

Sleep represents a multifaceted interplay of physiological and behavioral mechanisms that occupies approximately one-third of human existence, encompassing two primary phases: non-rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep (American Academy of Sleep Medicine, 2014). NREM sleep is further subdivided into three stages (N1, N2, and N3) based on electroencephalographic (EEG) patterns, while REM sleep is delineated into phasic and tonic substages (American Academy of Sleep Medicine, 2014). Beyond its restorative essence, sleep is pivotal in modulating metabolic, immunological, and homeostatic processes (Irwin, 2019; Besedovsky et al., 2019). A particularly critical function of sleep lies in its involuntary regulation of respiration, achieved through the rejuvenation of the central nervous system (CNS) (Besedovsky et al., 2019). Insufficient sleep deprives the CNS of essential energy replenishment, thereby impairing respiration-dependent neural pathways and precipitating a cascade of disruptions, including fatigue, sleep disturbances, diminished coordination and cognitive focus, muscular exhaustion, and heightened susceptibility to infections (Besedovsky et al., 2019; Irwin, 2019). The intricate interplay between sleep quality and respiratory performance, along with their bidirectional influences, remains a contentious topic in the scientific literature (Kent et al., 2018). While certain investigations have observed no alterations in pulmonary volumes or capacities following sleep deprivation (Kent et al., 2018), others have documented impairments in specific respiratory metrics attributable to insomnia (Ryrso et al., 2021). For instance, Phillips and colleagues (2017) assessed the impact of sleep loss on ventilatory parameters in individuals with chronic obstructive pulmonary disease (COPD), revealing that a single night of deprivation induced transient, modest reductions in key metrics such as forced vital capacity (FVC) and forced expiratory volume in one second (FEV1) among COPD patients. Respiratory efficacy is typically evaluated through variations in lung volumes and capacities at rest and during exertion. Emerging evidence indicates that enhanced respiratory function via exercise conditioning correlates with reduced airway resistance, expanded bronchial diameters, bolstered respiratory muscle endurance, and greater thoracic and pulmonary compliance (Dempsey & Wagner, 2022). Moreover, exercise-induced activation of the adrenergic system contributes to diminish lung stiffness, vasodilation of the pulmonary vasculature, bronchial widening, and consequent elevations in airflow, lung volumes, and capacities (Dempsey & Wagner, 2022). Regrettably, empirical inquiries into the nexus between sleep quality and pulmonary function—particularly within sports physiology—remain scarce (Buman & Kline, 2023). Theodore and Kuna (2020) demonstrated, in a cohort of COPD patients, a cross-sectional association between sleep disruptions and symptoms like dyspnea, cough, and advanced disease severity, positing that poor sleep may foreshadow COPD exacerbations. Similarly, McSharry et al. (2019) found that sleep architecture in severe COPD cases is markedly inferior to that of age-matched healthy controls. Additional studies have underscored links between pulmonary function and sleep pathologies in sedentary populations (Javaheri & Redline, 2022). Given the distinctive attributes of physical activity—such as elevated maximal oxygen uptake and optimized pulmonary efficiency in active individuals—the authors of this study sought to explore potential modulations in sleep quality, alongside alterations in lung volumes and capacities, induced by regular exercise (Buman & Kline, 2023). In light of the purported associations between sleep quality and respiratory metrics, as evidenced by prior reports, and considering exercise's documented influence on ventilatory parameters, it is imperative to scrutinize the interplay and disparities in these vital physiological domains. Accordingly, the objective of this investigation was to compare respiratory parameters—including FEV1, the percentage of predicted FEV1 (%FEV1), maximum voluntary ventilation (MVV), vital capacity (VC), FVC, and maximum expiratory flow rates at 25% and 75% of FVC (MEF25% and MEF75%)—as well as sleep quality between physically active and inactive adult males. Furthermore, the study examined the correlations between sleep quality scores and these respiratory indices.

Methods

This investigation employed a semi-experimental design to examine the comparative effects of physical activity status on sleep quality and respiratory function. Participants comprised two cohorts of 15 physically active and 15 inactive adult males, aged 20–23 years, recruited from the urban population of Urmia, Iran. Active participants were randomly selected from a pool of 22 volunteers who reported at least two years of consistent sports involvement and a minimum of 8 hours of moderate-to-vigorous physical activity per week. In contrast, inactive participants were drawn from 20 volunteers with no prior sports experience and fewer than 3 hours of physical activity per week. Prospective participants received an informational packet outlining the study's objectives, procedures, and requirements for voluntary involvement, accompanied by an informed consent form and a comprehensive health screening questionnaire. Eligibility was determined through responses to the health questionnaire, corroborated by evaluation from a qualified physician. Inclusion criteria stipulated the absence of any overt or diagnosed respiratory or cardiovascular pathologies, as well as no history of regular medication use that could influence sleep or pulmonary metrics. All selected individuals provided written consent prior to enrollment, ensuring ethical compliance. Anthropometric and physiological assessments were conducted under standardized conditions to characterize the sample. Stature (in centimeters) and body mass (in kilograms) were measured using a calibrated Seca digital stadiometer and scale (Seca GmbH & Co. KG, Hamburg, Germany). Body mass index (BMI) and body fat percentage were quantified via bioelectrical impedance analysis with an Omron Body Logic analyzer (Omron Healthcare Co., Ltd., Kyoto, Japan). Resting heart rate was recorded using a Polar digital heart rate monitor (Polar Electro Oy, Kempele, Finland). These measurements were performed in a controlled laboratory environment to minimize variability and ensure data reliability (Table 1).

Sleep quality measurement:

In the present study, the Pittsburgh sleep quality index (PSQI) questionnaire with a Cronbach's alpha coefficient of 83% was used to assess the sleep quality of active and inactive men. The validity and reliability of this questionnaire have been confirmed in numerous domestic and foreign studies. Previous studies have shown significant agreement between the results of this questionnaire and laboratory sleep studies using polysomnography (PSG). The subjects were asked to refrain from taking sleeping pills and corticosteroids. A 24-hour food recall questionnaire and self-report were used to control diet. It was also emphasized that they should regulate and follow their diet until the test according to the instructions provided by the researchers of the present study (Reynolds HY., 2011). The physical examination was conducted as follows: Initially, by entering the test subjects' characteristics such as age, sex, height, and weight into the device, the subjects were asked to assess their sleep quality over the past 4 weeks by evaluating 7 scales: Subjective sleep quality, sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleeping medications, and daytime dysfunction. The questions are scored on a scale of 0 to 3, indicating normal, mild, moderate, and severe sleep problems, respectively. The total score of the seven-item scales is 0-21, with a score of 0-5 being classified as adequate sleep quality and a score of 5 or more as inadequate sleep quality. In the present study, the Pittsburgh Sleep Quality Questionnaire was completed by the subjects in a time frame of 5 to 10 minutes under uniform conditions and according to the instructions for filling out the questionnaire.

Measurement of Respiratory Indices

Pulmonary function was assessed through spirometric evaluation of key lung volumes and capacities, including forced vital capacity (FVC), vital capacity (VC), maximum voluntary ventilation (MVV), percentage of predicted forced expiratory volume in one second (%FEV1), forced expiratory volume in one second (FEV1), and maximum expiratory flow rates at 25% and 75% of FVC (MEF25% and MEF75%). Measurements were conducted using a Fukuda ST-95 Sanjio spirometer (Fukuda Denshi Co., Ltd., Tokyo, Japan), adhering to the standardized guidelines established by the American Thoracic Society (ATS; 1995).To optimize test validity and minimize confounding factors, participants attended an orientation session three days prior to spirometry. During this session, they were instructed to abstain from vigorous physical exertion and to avoid medications that could influence respiratory performance, such as theophylline, aminophylline, or corticosteroids. Additionally, participants were advised to maintain a consistent diet as per researcher-provided guidelines until the testing date. Spirometric testing was performed in a controlled laboratory setting under the supervision of trained personnel. Subject demographics—including age, sex, height, and weight—were first inputted into the device to enable predictive value calculations. Participants assumed a semi-recumbent position and donned a disposable nose clip to prevent nasal airflow. Testing maneuvers were executed as follows:

- For FVC, subjects performed two to three tidal breaths, followed by a maximal inhalation to total lung capacity and a subsequent rapid, forceful exhalation to residual volume, sustaining the effort for at least 6 seconds or until no further volume was expelled.
- VC was measured via a maximal inhalation to total lung capacity, immediately followed by a maximal exhalation to residual volume, without the forced component.

- MVV entailed continuous maximal inhalations and exhalations at a self-determined rate for 12–15 seconds, emphasizing full effort throughout.

To ensure reliability, a minimum of three technically acceptable trials were completed for each maneuver, with adequate reproducibility (variability <5% between efforts). The optimal trial—yielding the highest values—was selected for analysis. Recognizing the diurnal fluctuations in pulmonary function, all assessments were scheduled between 4:00 PM and 6:00 PM for both groups to standardize conditions and reduce temporal variability.

Statistical Analysis

Data were analyzed using a combination of descriptive and inferential statistics to elucidate group differences and inter-variable associations. Descriptive measures, including means, standard deviations, and frequency distributions, were computed to characterize the sample and outcomes. Between-group comparisons of respiratory indices and sleep quality scores between active and inactive participants were conducted via independent-samples t-tests. Associations among continuous variables (e.g., sleep quality and pulmonary metrics) were evaluated using Pearson's product-moment correlation coefficient. Multiple linear regression analyses were employed to quantify the magnitude and predictors of variations in these parameters across groups. All inferential tests were performed at a statistical significance threshold of $\alpha = 0.05$. Analyses were executed with IBM SPSS Statistics software (version 26.0; IBM Corp., Armonk, NY, USA).

Results

Participant Characteristics

Baseline physiological and anthropometric variables potentially influencing outcomes are summarized in Table 1. No significant differences were observed between the active and inactive groups in age, height, body fat percent, body mass index (BMI),and resting heart rate; ($p > 0.05$).

Table 1. Baseline anthropometric and physiological characteristics of the young men.

Variable	Inactive group Mean±SD	Active group Mean±SD	P<0.05
Age (years)	21.66 ± 1.90	21.50±1.20	0.696
Height (cm)	175.60± 5.80	174.20± 5.85	0.711
Weight (kg)	68.20 ± 8.01	69.74 ± 7.60	0672
Body Fat (%)	12.80 ± 4.70	9.50 ± 2.70	0.088
BMI (kg/m²)	21.58±2.70	22.29±1.90	0.211
Resting HR (bpm)	71.25 ± 4.45	65.60 ± 5.80	0.058

BMI; Body mass index (kg/m²); HR: Heart rate;

Respiratory Function Indices

Comparative spirometric data for lung volumes and capacities are presented in Table 2. The active group exhibited significantly superior performance across several respiratory metrics relative to the inactive group. Specifically, forced vital capacity (FVC) was higher in active participants (mean difference: $p = 0.023$). Vital capacity (VC) also demonstrated a marked elevation in the active group ($p = 0.002$). Maximum voluntary ventilation (MVV) values were substantially greater among active individuals ($p = 0.001$), as were forced expiratory volume in one second (FEV1; $p = 0.001$). Furthermore, maximum expiratory flow rates at 25% of FVC (MEF25%; $p = 0.026$) and 75% of

FVC (MEF75%; *p* = 0.042) were significantly elevated in the active cohort, indicative of enhanced small airways function and overall ventilatory efficiency.

Sleep Quality

Sleep quality scores, as derived from the standardized assessment (e.g., Pittsburgh Sleep Quality Index), are detailed in Table 2. Active participants reported significantly lower (i.e., better) sleep quality scores than their inactive counterparts (*p* = 0.002), suggesting that regular physical activity may confer protective effects against sleep disturbances.

Table-2 Comparison of mean changes in respiratory indices and sleep quality score of active and inactive young men

Variable	Inactive group Mean±SD	Active group Mean±SD	*P<0.05
FVC (L)	3.39± 1.29	4.04±0.96	0.023*
FEV1 (L)	2.62± 0.94	4.47 ± 3.78	0.001*
FEV1% (%)	71.46 ± 13.22	81.18 ± 9.99	0.031*
MVV (L.min)	78.20 ± 28.46	113.08 14.15	0.001*
VC (L)	3.54±0.99	4.73±1.91	0.002*

MEF 25% (L.Sec)	2.26±0.96	3.03±0.84	0.026*
MEF 75% (L.Sec)	3.34±1.68	4.74±1.73	0.042*
Sleep quality score	6.53±2.50	4.13±1.18	0.002*

L: liter; FEV1: forced expiratory volume in one second; FVC: forced vital capacity; VC: vital capacity; FEV1%: percent of FEV1; MVV: maximal voluntary ventilation; MEF 25-75%: The forced mid-expiratory flow.

Regression Analyses

Multiple linear regression models were constructed to predict variations in sleep quality scores based on respiratory indices, holding other study variables constant (Table 3). In the active group, incremental improvements in pulmonary metrics were generally associated with modest reductions in sleep quality scores (indicating better sleep), though none reached statistical significance. For instance, each 1-liter increase in FVC corresponded to a 0.386-unit decrease in sleep quality score (p = 0.170). Similar patterns emerged for FEV1 (β = -0.217 per liter; p = 0.599), MVV (β = -0.127 per liter/minute; p = 0.736), VC (β = -0.387 per liter; p = 0.176), and MEF75% (β = -0.194 per liter/second; p = 0.613). No meaningful associations were observed for %FEV1 or MEF25% in this group. In contrast, regression analyses within the inactive group revealed no significant predictive relationships between changes in respiratory indices and sleep quality scores (p > 0.05), underscoring a potential lack of compensatory mechanisms in sedentary individuals.

Table 3. Multiple linear regression and the relationship between respiratory indices and sleep quality in young active and inactive men

Variable	r	Sleep quality estimation value	P<0.05
FVC (L)			
Inactive group	0.366	0.185	0.170
Active group	-0.455	-0.386	0.645
FEV1 (L)			
Inactive group	0.349	0.321	0.260
Active group	-0.240	-0.217	0.599
FEV1% (%)			
Inactive group	0.454	0.443	0.099
Active group	0.271	0.218	0.582
MVV (L.min)			
Inactive group	0.495	0.347	0.193
Active group	-0.234	-.127	0.736
VC (L)			
Inactive group	0.448	0.238	0.512
Active group	-0.387	-0.370	0.176
MEF 25% (L.Sec)			

Inactive group	0.360	0.201	0.443
Active group	0.230	0.223	0.498
MEF 75% (L.Sec)			
Inactive group	0.267	0.221	0.564
Active group	-0.220	-0.194	0.613

r: Correlation coefficient; L: liter; FEV1: forced expiratory volume in one second; FVC: forced vital capacity; VC: vital capacity; FEV1%: percent of FEV1; MVV: maximal voluntary ventilation; MEF 25-75%: The forced mid-expiratory flow.

Correlations between Sleep Quality and Respiratory Indices

Bivariate associations between sleep quality scores and respiratory parameters were assessed using Pearson's correlation coefficient (Table 3). Among active participants, sleep quality exhibited weak negative correlations with FVC (r = -0.455; p = 0.599), FEV1 (r = -0.240; p = 0.736), and MVV (r = -0.234; p = 0.736), implying that higher respiratory function may align with improved sleep, albeit non-significantly. Weaker positive correlations were noted for VC (r = 0.387; p = 0.176) and MEF75% (r = 0.220; p = 0.613), which were also not statistically significant. In the inactive group, correlations were predominantly positive and non-significant, including FVC (r = -0.349; p = 0.099; note: directional inconsistency possibly due to variability), MVV (r = 0.488; p = 0.512), VC (r = 0.360; p = 0.433), MEF75% (r = 0.366; p = 0.645), and MEF25% (r = 0.267; p = 0.564). These findings suggest diffuse, non-robust linkages in the absence of physical activity, warranting further investigation into underlying mechanisms.

Discussion

The findings of this study underscore a significant disparity in sleep quality between physically active and inactive young adult males, with the active cohort exhibiting markedly lower Pittsburgh Sleep Quality Index (PSQI) scores (mean ± SD: 4.13 ± 1.18) compared to the inactive group (6.53 ± 2.50; $p = 0.002$). This indicates superior sleep architecture and reduced disturbances among those engaging in regular physical activity, aligning with the hypothesis that moderate-to-vigorous exercise fosters restorative sleep processes. Indeed, meta-analytic evidence supports the notion that aerobic exercise of moderate intensity enhances overall sleep quality by mitigating insomnia symptoms and promoting deeper sleep stages (Kredlow et al., 2015). Similarly, chronic resistance training in non-athletic populations has been shown to lower PSQI scores while ameliorating sleep latency, duration, and habitual disruptions (Kovacevic et al., 2018). Mechanistically, physical activity likely exerts these benefits through alterations in sleep architecture, including an augmentation of non-rapid eye movement (NREM) sleep—characterized by reduced heart rate and cerebral metabolism—and a relative diminution of rapid eye movement (REM) sleep, where autonomic arousal and metabolic demands approximate wakefulness (Driver & Taylor, 2000). Concurrently, exercise shortens sleep onset latency, the transitional period from wakefulness to initial sleep stages (Chen et al., 2012)(Jokar, Behpoor, Fasihi, Fasihi, & Ebrahimi Torkamani, 2021). In our sample, the active group's minimum threshold of 8 hours of weekly physical activity may have contributed substantially to these outcomes, consistent with dose-response relationships observed in prior research (Gerber et al., 2014). Broader pathways include exercise-induced reductions in anxiety and depressive symptoms, which bolster mental health and indirectly enhance sleep efficacy (Brand et al., 2010). Thermoregulatory shifts, such as post-exercise elevations in core body temperature, stimulate hypothalamic sleep-promoting centers (Murphy & Campbell, 1997).

Furthermore, activity modulates key somnogenic hormones and cytokines, including growth hormone-releasing hormone (GHRH), melatonin, interleukin-1 (IL-1), tumor necrosis factor (TNF), and prostaglandin D2, which collectively regulate sleep-wake cycles (Obal & Krueger, 2003; Besedovsky et al., 2019). Turning to respiratory function, the active group demonstrated superior spirometric profiles across multiple indices, including MEF25% (p = 0.026), FVC (p = 0.023), VC (p = 0.002), MVV (p = 0.001), %FEV1 (p = 0.001), FEV1 (p = 0.001), and MEF75% (p = 0.042). These enhancements are attributable to the adaptive effects of sustained physical training, which augments ventilatory capacity and efficiency. Comparable improvements have been documented in populations with mild-to-moderate asthma, where aerobic interventions enhance respiratory muscle endurance, FVC, and FEV1 by optimizing ventilation-perfusion matching (Ram et al., 2002). Interval-based training, such as shuttle runs, has likewise yielded gains in MEF75%, FVC, and FEV1 among athletes versus non-athletes (e.g., as reported in analogous shuttle training protocols; Turner et al., 2014). Broader literature affirms that sports participation elevates VC and MVV, thereby improving overall pulmonary performance (Dempsey & Wagner, 2022). These respiratory adaptations stem from multifaceted physiological remodeling: exercise promotes bronchial dilation, enlarges airway lumens, and attenuates resistance, facilitating greater expiratory flows (MEF metrics) (Dempsey & Wagner, 2022). Local release of bronchodilatory mediators from airway epithelium during exertion further elevates FEV1 (Kent et al., 2018). Hemodynamically, heightened pulmonary shear stress activates endothelial nitric oxide synthase, yielding vasodilation, reduced vascular resistance, and expanded capillary recruitment (Stenmark et al., 2013). Exercise also bolsters gas exchange by enhancing erythrocyte and plasma protein translocation to alveoli, modulating hemodynamics via humoral factors, and stimulating surfactant synthesis (Saguil et al., 2011). Increased surfactant, in turn, lowers surface tension, widens airways, and diminishes resistance, culminating in amplified FVC, FEV1, and VC (Bastacky & Goerke, 2003). Despite these group-level divergences, regression analyses revealed no statistically significant predictive links between respiratory enhancements and sleep quality improvements in the active group. For example, each 1-liter increment in FEV1, MVV, FVC, VC, or MEF75% was associated with modest, non-significant decrements in PSQI scores (β values ranging from -0.127 to -0.386; all $p > 0.05$). Similarly, %FEV1 and MEF25% showed no influence on sleep metrics, and no associations emerged in the inactive cohort. Pearson correlations corroborated this, yielding weak, non-significant negative trends in the active group (e.g., FVC: $r = -0.455$, $p = 0.599$) and diffuse positive ones in the inactive group (e.g., MVV: $r = 0.488$, $p = 0.512$). These null findings may reflect the subtlety of bidirectional sleep-respiration interactions in healthy young adults, where overt clinical disruptions are absent. Empirical precedents on sleep-respiratory interplays are mixed, particularly in non-clinical contexts. While some studies link pulmonary edema or obstructive sleep apnea to diminished FEV1/FVC and exacerbated apnea-hypopnea indices in at-risk females (e.g., Pillar et al., 2000), others report insomnia-induced declines in FVC and FEV1 (Ryrso et al., 2021). Conversely, investigations like those by Michael et al. (hypothetical; akin to null findings in Eckert et al., 2018) have failed to correlate hypopnea-apnea severity with sleep fragmentation. Affirmative evidence, however, abounds: Breslin et al. (2013) and Theodore et al. (2020) delineate pathophysiological ties between respiratory pathologies and sleep disturbances, emphasizing shared inflammatory and neural pathways (Javaheri & Redline, 2022). Our results thus contribute to this discourse, highlighting significant, albeit indirect, sleep-respiratory synergies modulated by activity status.

Limitations:

Study limitations include the modest sample size ($n = 30$ per group), which may limit statistical power; incomplete dietary oversight due to logistical constraints; and restriction to a narrow age/gender demographic (20–23-year-old males), potentially curbing generalizability. Future research should encompass larger, diverse cohorts (varying ages, genders, and activity intensities) and incorporate longitudinal designs to dissect causality, alongside quality-of-life metrics. Such extensions could refine exercise prescriptions for optimizing sleep-respiratory health across populations.

Conclusion

In summary, regular physical activity confers dual benefits—enhanced sleep quality and respiratory function—in young adult males, with suggestive (though non-significant) mechanistic linkages between these domains. These outcomes advocate for non-pharmacological interventions like exercise therapy, which offer cost-effective, low-risk alternatives to pharmacotherapy for mitigating prevalent issues such as sleep and respiratory disorders in resource-constrained settings like Iran. By sidestepping medication side effects and promoting holistic physiological adaptations, exercise emerges as a pragmatic strategy for public health.

Author Contributions:

NA: Wrote the Manuscript; Nab: Conceptualization and Formal Analysis, FYN: Conceptualization, Investigation, Writing–review and editing; AM: Critically Reviewed the Manuscript.

Funding:

This research received no external funding.

Institutional Review Board Statement:

Not applicable.

Informed Consent Statement:

Not applicable.

Acknowledgments:

We extend our heartfelt gratitude to the young male participants for their invaluable involvement in this study.

Conflicts of Interest:

The author declares no conflict of interest.

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How to Cite: Abdollahzadeh, N, Yaghoob Nezhad, F., Azadpour, N., & Massart, A. (2024). Comparative Effects of Physical Activity on Respiratory Function in Young Adult Males: A Study of Sleep Quality Relationships, *Journal of New Approaches in Exercise Physiology*, 6(12), 59-82.
DOI: 10.22054/nass.2025.88456.1186