



# Comparison of Maximal Oxygen Uptake and Post-Exercise Recovery Dynamics of Smoking and Non-Smoking Athletes

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## Abstract:

**Background of Study:** Smoking has long been recognized as a risk factor for impaired cardiovascular and respiratory functions, yet its specific effects on athletic performance require further clarity. For athletes, maintaining high aerobic capacity and efficient recovery is essential for endurance, training adaptation, and competitive success. This study aimed to compare maximal oxygen and post-exercise recovery dynamics-  $VO_2$  Max, Heart Rate Recovery, Oxygen Saturation, and Breathing Frequency between smoking and non-smoking athletes..

**Methods:** A comparative cross-sectional design was employed with forty trained athletes aged 25–28 years, equally divided into smoking and non-smoking groups, each with at least four years of competitive training. Participants performed a graded treadmill exercise test followed by standardized recovery assessments under controlled laboratory conditions.

**Result:** Findings revealed that non-smoking athletes achieved significantly higher maximal oxygen uptake, reflecting superior aerobic capacity. While differences in oxygen saturation, heart rate recovery, and breathing frequency were not statistically significant, smokers showed trends toward lower oxygen availability, slower cardiovascular recovery, and less efficient ventilatory adjustment.

**Conclusion:** In conclusion, maximal oxygen uptake emerged as the most sensitive marker distinguishing smokers from non-smokers, underscoring the detrimental influence of tobacco use on endurance and recovery. These results emphasize the importance of discouraging smoking among athletes to safeguard physiological efficiency, support optimal training, & enhance long-term performance.

**Keywords:** Breathing Frequency, Heart Rate Recovery, Maximal Oxygen Uptake, Saturated Oxygen ( $SpO_2$ ), Tobacco

## 1. INTRODUCTION

Aerobic capacity, commonly represented as Maximal Oxygen Uptake ( $VO_2$  max), is a key indicator of cardiovascular health and athletic performance. The combined effectiveness of the respiratory, circulatory, and muscular systems to transport and use oxygen during prolonged exercise is reflected in  $VO_2$  Max, which has a direct impact on stamina and competitive results in endurance sports (Kahmasi, 2025). Higher  $VO_2$  Max values are linked to lower risk of cardiovascular morbidity and all-cause mortality, highlighting their clinical implications beyond performance (Aker et al., 2023).

In addition to improving aerobic power and training responses,  $VO_2$  Max optimization for athletes is a crucial indicator of resilience and long-term health. Sensitive physiological parameters that show how well autonomic regulation and cardiopulmonary adaptation work after exertion include post-exercise recovery dynamics, namely heart rate recovery (HRR), oxygen saturation ( $SpO_2$ ), and breathing frequency (BF). HRR, defined as the rate of drop in heart rate immediately after exercise, is substantially linked with parasympathetic reactivation and cardiovascular fitness (Holt et al., 2019). Similarly, the recovery of  $SpO_2$  indicates the effectiveness of oxygen supply and pulmonary gas exchange, both of which can be hampered by illnesses like smoking-induced hypoxemia (Dugral et al., 2019). Faster normalization during recovery dynamics, better training adaptation, and breathing frequency during recovery offer information on ventilatory control and metabolic demand (Naranjo et al., 2015).

When combined, these indices offer a comprehensive and non-invasive evaluation of the total cardiopulmonary efficiency and recovery state of athletes. Because these measurements represent different but connected elements of cardiopulmonary and autonomic function, studying maximal oxygen uptake ( $VO_2$  Max), heart rate recovery (HRR),

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oxygen saturation (SpO<sub>2</sub>), and breathing frequency together offers an integrated assessment of athlete physiology.

The most frequently accepted measure of aerobic capacity is VO<sub>2</sub> Max, which shows how well oxygen is transported and used during maximal effort (Kahmasi, 2025). As a marker of training state and cardiovascular health, post-exercise HRR represents parasympathetic reactivation and cardiovascular resilience (Lazic et al., 2015). While breathing frequency records ventilatory control and respiratory muscle demand before and after activity, SpO<sub>2</sub> offers information on the efficiency of pulmonary gas exchange (Webster & Karan, 2020). A multifaceted understanding of how training, recuperation, and lifestyle factors—like smoking—affect athlete performance and recovery capacity is made possible by evaluating these variables simultaneously.

Numerous physiological systems are negatively impacted by smoking, especially those that affect how well and how quickly one recovers from exercise. Because carbon monoxide has a stronger affinity for hemoglobin than oxygen, it can create carboxyhemoglobin, which lowers tissue delivery and oxygen transport capacity (Oikonomou & Siasos, 2019). Furthermore, persistent inflammation and increased airway resistance brought on by tobacco smoke exposure impair pulmonary function and ventilatory efficiency (Flor et al., 2024). Smoking causes oxidative stress at the systemic level by producing too many reactive oxygen species, which damages mitochondrial efficiency and vascular function (Rahman et al., 2025). Additionally, by increasing sympathetic activity, raising resting heart rate, and decreasing autonomic recovery after exertion, nicotine and other components change cardiovascular control (Middlekauff, 2020).

When combined, these processes impair aerobic capacity and cause athletes' post-exercise recovery to be delayed. In comparison to non-smokers, smokers have a decreased aerobic capacity, a delayed cardiovascular recovery, and impaired oxygen transport, as several studies repeatedly show. Daily tobacco use has a negative correlation with VO<sub>2</sub> Max, even in young, healthy guys, both in relation to lean body mass ( $r = -0.50$ ) and body weight ( $r = -0.37$ ) (Voutilainen et al., 2020). Smokers have a reduced heart-rate recovery after exercise in athletic groups; for example, female smokers on treadmill testing demonstrated a noticeably slower HR fall than comparable non-smokers (Pepera & Panagiota, 2021).

The recovery kinematics of trained soccer players who smoke are also changed, with a noticeably slower second-phase heart-rate normalization (Tsai & Ho, 2017). Moreover, during vigorous exercise, habitual smokers' oxygen transport efficiency decreases by about 10%, and their airway resistance is increased by more than twice that of non-smokers (Bao et al., 2024). All of these results highlight the detrimental effects of smoking on maximal oxygen absorption and post-exercise recovery physiology. Even minor physiological abnormalities can have a significant impact on performance and recovery in athletes. Small declines in oxygen transport, ventilatory efficiency, or autonomic regulation can result in quantifiable decreases in endurance, recovery speed, and overall competitive outcomes for elite and competitive athletes, who operate close to the upper limits of their cardiopulmonary and metabolic capacities (Baumert et al., 2021).

Smoking can impair maximal oxygen uptake and the rate of post-exercise recovery, two processes necessary for maintaining repeated episodes of high-intensity activity. These changes include decreased arterial oxygen saturation, elevated carboxyhemoglobin, and attenuated parasympathetic reactivation (Adatia et al., 2021). The potential effects of smoking on athletes extend beyond short-term performance limitations to long-term athletic development and resilience, as recovery efficiency is crucial for both injury prevention and adaptation, as well as for the quality of subsequent training (Aydin, 2023).

### 1.1 Statement of the Problem

Although smoking has been shown to have detrimental impacts on lung and cardiovascular health, nothing is known about how smoking affects athletic performance. To maintain training quality and succeed in competition, athletes rely significantly on maximal oxygen uptake (VO<sub>2</sub> Max) and effective post-exercise recovery dynamics, such as heart rate recovery, oxygen saturation, and breathing frequency. Even little physiological deficits can reduce endurance, hinder recovery, and make a person more prone to injury or exhaustion. Few studies have directly compared smoking and non-smoking athletes who engage in intense physical exercise, even though previous data show smokers in general populations have lower VO<sub>2</sub> Max and recover more slowly. This dearth of data leaves a significant gap in sports science, where knowledge of the precise effects of smoking is crucial for directing preventive measures, performance enhancement, and athlete health care.



## 1.2 Objectives of the Study

1. To compare the difference in maximal oxygen uptake (VO<sub>2</sub> Max) between smoking and non-smoking athletes.
2. To compare the difference in heart rate recovery (HRR) between smoking and non-smoking athletes.
3. To compare the difference in oxygen saturation (SpO<sub>2</sub>) responses during post-exercise recovery in smoking and non-smoking athletes.
4. To compare the difference in breathing frequency recovery patterns of smoking and non-smoking athletes.

## 1.3 Significance of the Problem

The significance of this study lies in its potential to clarify how smoking compromises the physiological mechanisms that directly support athletic performance. The hidden costs of smoking on sport-specific fitness are demonstrated by comparing smoking and non-smoking athletes. This is because VO<sub>2</sub> Max, heart rate recovery, oxygen saturation, and breathing frequency are important markers of cardiovascular efficiency and recovery capacity. Athletes themselves can benefit from this information, as can coaches, sports doctors, and legislators who aim to create targeted initiatives that discourage smoking and promote healthier lives among competitive populations. Additionally, by connecting the dots between clinical studies on tobacco and its real-world effects on athletic performance, recuperation, and long-term development, the findings will advance the area of exercise science.

## 2. MATERIAL AND METHOD

### 2.1 Research Design

The present study adopted a comparative cross-sectional design to investigate differences in maximal oxygen uptake and post-exercise recovery dynamics between smoking and non-smoking athletes. This design was chosen to allow simultaneous assessment of the two groups under standardized conditions, thereby enabling meaningful comparisons of physiological outcomes.

### 2.2 Participants

The sample includes a total of 40 athletes (20 each) trained athletes having a minimum of 4 years of training experience, aged between 25 to 28 years. To minimize misclassification, participants were divided into two groups according to their self-reported smoking status, which was confirmed by an exhaled carbon monoxide measurement. The athletes in the

smoking group had smoked for at least a year, while the athletes in the non-smoking group had never used tobacco. Active engagement in competitive sports and at least three training sessions per week were prerequisites for inclusion. People who were taking medications that affected their heart rate or respiratory function, or who had been diagnosed with cardiovascular, pulmonary, or metabolic diseases, were not included.

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### 2.3. Instrument and Measurement

#### 2.3.1 Maximum Oxygen Uptake (VO<sub>2</sub> Max)

Maximal oxygen uptake was assessed using a treadmill integrated with a breath-by-breath metabolic gas analyzer, Cosmed Quark CPET. Before each test, the metabolic cart was carefully calibrated using standard reference gases for oxygen and carbon dioxide, along with a 3-L syringe for flow-volume calibration, to ensure measurement accuracy and reliability. Participants then performed a graded exercise test (GXT) involving incremental workload increases until volitional exhaustion. VO<sub>2</sub> Max was considered achieved when at least two of the following verification criteria were satisfied: the occurrence of a plateau in oxygen uptake despite an increase in workload, a respiratory exchange ratio (RER) of  $\geq 1.10$ , and attainment of at least 90% of the age-predicted maximum heart rate.

#### 2.3.2 Heart Rate Recovery (HRR)

Heart rate recovery (HRR) was assessed using continuous ECG monitoring. Peak heart rate (HR<sub>peak</sub>) was determined at the point of exercise termination, after which participants entered a passive seated recovery phase. Heart rate was recorded at 60 seconds (HR<sub>60</sub>) and 120 seconds

(HR<sub>120</sub>) post-exercise, and HRR was subsequently calculated as the difference between HR<sub>peak</sub> and HR<sub>60</sub>, and HR<sub>peak</sub> and HR<sub>120</sub>, respectively.

### 2.3.3 Oxygen Saturation (SpO<sub>2</sub>)

Oxygen saturation (SpO<sub>2</sub>) was measured using a medical-grade fingertip pulse oximeter with high sampling accuracy, the Masimo Radical-7. Measurements were obtained during the recovery phase, with particular attention to recovery values at 1- and 3-minute post-exercise for analysis. To ensure reliability and accuracy.

### 2.3.4 Breathing Frequency

Breathing frequency (BF) was obtained from the ventilatory flow signals of the metabolic cart, recorded on a breath-by-breath basis. Measurements were taken at baseline, at peak exercise, and during the recovery phase at 1-, 2-, and 3-minute post-exercise. To reduce variability and enhance measurement precision, automated averaging over 10-second intervals was applied for all BF data points.

### 2.4 Procedure

After refraining from alcohol, caffeine, and severe exercise over the previous 24 hours, the participants

arrived at the lab refreshed and fasting. The following protocol was introduced. After completing a standardized warm-up, each athlete ran an incremental treadmill test until they were exhausted. Participants went through a sitting recovery phase right after stopping, during which time their BF, SpO<sub>2</sub>, and HRR were continually recorded. To minimize variability, all trials were conducted in the same standardized testing environment, which included the same room temperature and time of day.

### 2.4 Statistical Analysis

The IBM SPSS Version 27 was used for statistical analysis of the data. First, the nature of the data was explained through descriptive statistics (Mean & Standard Deviation). Then the normality of the data was checked through the Shapiro-Wilk Test at 0.05. Later, based on the p-value of the Shapiro-Wilk test, the Independent Sample t-test or Mann-Whitney U test was used for further analysis at the 0.05 significance level.

## 3. RESULT AND DISCUSSION

### 3.1 Result

**Table 1.** Descriptive Statistics of Smoking and Non-Smoking Athletes Across Maximal Oxygen Uptake and Post-Exercise Recovery Dynamics

Variables	Smoking Athletes (n = 20) (Mean ± SD)	Non-Smoking Athletes (n = 20) (Mean ± SD)
<b>Demographics &amp; Anthropometrics</b>		
Age (years)	23.65 ± 2.77	25.35 ± 4.05
Body Mass (kg)	67.79 ± 7.61	65.08 ± 6.13
Height (cm)	172.47 ± 7.96	174.05 ± 7.66
Body Mass Index (kg/m <sup>2</sup> )	22.92 ± 3.32	21.59 ± 2.81
<b>Study related variables</b>		
VO <sub>2</sub> Max (mL/kg <sup>-1</sup> /min <sup>-1</sup> )	46.15 ± 4.61	56.50 ± 4.93
SpO <sub>2</sub> Rest (%)	97.99 ± 0.97	98.38 ± 0.84
HRR (Rest Phase)	63.90 ± 6.29	62.80 ± 6.57
Breathing Frequency	14.15 ± 1.84	14.95 ± 1.79

The results indicate that non-smoking athletes demonstrated a higher maximal oxygen uptake (VO<sub>2</sub> Max = 56.50 ± 4.93 mL/kg/min) compared to their smoking counterparts (46.15 ± 4.61 mL/kg/min), highlighting the detrimental impact of smoking on cardiorespiratory efficiency and overall aerobic performance. Similarly, peripheral oxygen saturation at rest (SpO<sub>2</sub>) was superior in the non-smoking group (98.38 ± 0.84%) relative to smokers (97.99 ± 0.97%), suggesting more effective pulmonary gas exchange and oxygen transport among athletes without a

history of smoking. Heart rate recovery (HRR), an established marker of autonomic function and cardiovascular resilience, was slightly better in the non-smoking athletes (62.80 ± 6.57) compared to smokers (63.90 ± 6.29); although the difference appears marginal, the slower recovery in smokers may point toward reduced parasympathetic reactivation post-exercise. In terms of breathing frequency, non-smoking athletes exhibited a relatively higher rate (14.95 ± 1.79 breaths/min) than smokers (14.15 ± 1.84 breaths/min), which may



reflect more efficient ventilatory control during recovery.

To assess whether the mean differences shown in Table 1 are statistically significant or not at the 0.05

significance level, the Independent Sample t Test or Mann-Whitney U Test will be used based on the normality of the data across each variable for both groups.

**Table 2.** Result of Test of Normality

Variables	Group	Shapiro-Wilk Statistics	df	p-value	Normality Assumption
VO2 Max	Smoking	.944	20	.281	Normally Distributed
	Not-Smoking	.963	20	.607	Normally Distributed
SpO2 Rest	Smoking	.953	20	.421	Normally Distributed
	Not-Smoking	.981	20	.945	Normally Distributed
HRR	Smoking	.908	20	.058	Normally Distributed
	Non-Smoking	.889	20	.025	Non-Normal Distribution
Breathing	Smoking	.868	20	.011	Non-Normal Distribution
Frequency	Non-Smoking	.862	20	.008	Non-Normal Distribution

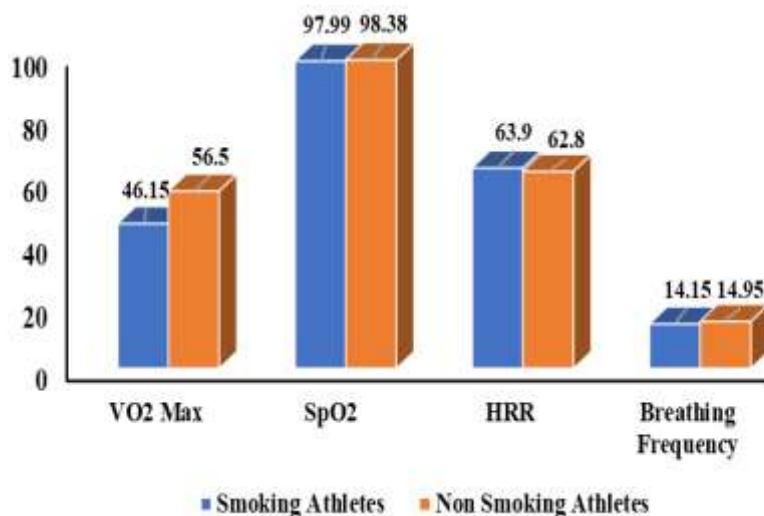
Note:  $p > 0.05$  indicates data are normally distributed;  $p < 0.05$  indicates violation of normality

**Table 3.** Between-Group Comparison Based on Test of Normality

Variable	Test	t-value	z-value	df	p-value	Interpretation
VO <sub>2</sub> Max	Independent sample-t	-6.830	N/L	38	.001	Significant
SpO <sub>2</sub> Rest	Independent Sample-t	-1.336	N/L	38	.190	Not Significant
HRR	Mann-Whitney U	N/L	-0.556	-	.578	Not Significant
Breathing Frequency	Mann-Whitney U	N/L	-1.386	-	.166	Not Significant

The results of the between-group comparison presented in Table 3 highlight that only VO<sub>2</sub> Max demonstrated a statistically significant difference between the groups. Specifically, the independent sample t-test revealed a robust difference, indicating that aerobic capacity varied meaningfully across the groups. In contrast, SpO<sub>2</sub> at rest did not reach statistical significance, suggesting comparable resting oxygen saturation levels. Similarly, the

Mann-Whitney U test results for HRR and breathing frequency showed no significant differences, implying that both groups exhibited similar physiological responses in terms of heart rate recovery and breathing regulation. Taken together, these findings underscore that VO<sub>2</sub> Max serves as the primary differentiating variable between the groups, while other physiological markers remained largely consistent



**Figure 1.** Graphical Representation of Mean Value of Study Variables across Smoking and Non-Smoking Athletes.

### 3.2 Discussion

The current study highlights the negative effects of tobacco use on aerobic efficiency and endurance performance by showing that smoking athletes had much lower maximum oxygen uptake than their non-smoking counterparts. This result is consistent with previous findings that smoking reduces mitochondrial function, lowers ventilatory efficiency, and raises carboxyhemoglobin levels, all of which affect oxygen transport and consumption (Oikonomou & Siasos, 2019; Rahman et al., 2025).

The slightly slower recovery in smokers may indicate mild impairments in parasympathetic reactivation, which is consistent with previous research showing delayed autonomic recovery among smokers (Hassan, 2019). Additionally, although there was no statistically significant difference in resting SpO<sub>2</sub>, smokers' slightly lower values are suggestive of pulmonary gas exchange efficiency impairment, which has been extensively documented in smoking populations (Dugral et al., 2019). When taken as a whole, these findings demonstrate that VO<sub>2</sub> Max is the most sensitive physiological indicator that can differentiate smokers from non-smokers, illustrating the long-term effects of tobacco on respiratory and cardiovascular function. Crucially, for athletes functioning close to physiological limitations, even small declines in oxygen transport and recovery dynamics can significantly affect training adaptation and competitive results (Yoda et al., 2024). Thus, this study not only reinforces the adverse consequences of smoking on aerobic performance but also emphasizes the need for targeted preventive interventions in athletic populations to safeguard both health and sport-specific performance.

### 4. CONCLUSION

The findings of this study demonstrate that smoking exerts a detrimental effect on athletic physiology, most notably through a significant reduction in maximal oxygen uptake (VO<sub>2</sub> Max), which serves as a critical determinant of endurance capacity and overall aerobic performance. While differences in heart rate recovery, oxygen saturation, and breathing frequency were not statistically significant, the observed trends suggest subtle impairments in recovery and pulmonary efficiency among smokers. These results underscore that even minor disruptions in cardiopulmonary function can compromise

training adaptation and long-term performance, particularly in athletes already operating near their physiological limits. By highlighting VO<sub>2</sub> Max as the most sensitive marker distinguishing smoking from non-smoking athletes, this study emphasizes the importance of discouraging tobacco use within competitive populations and reinforces the role of lifestyle factors in shaping both performance outcomes and athletic longevity.

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### 6. AUTHOR CONTRIBUTION STATEMENT

The author, Dr. Rahul Dev Choudhury, designed the study and interpreted the findings, and Manish Acharjee collected and analyzed the data and prepared the manuscript. All aspects of the work have been completed under the author's responsibility, and the final version has been approved for submission.

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