QUALITY IN SPORT
eISSN 2450-3118 · Open Access · Peer-reviewed
apcz.umk.pl/QS Nicolaus Copernicus University in Toruń

Cite as: CIEŚLEWICZ, Karolina, BRAKSAL, Barbara, CZĘCZEK, Kacper, MINIK, Bartosz, MIRKOWSKI, Krzysztof, SZUREK, Zuzanna Wanda, CHYŁA, Zofia Julia, MIROŃSKI, Adam, NOWACZEK, Patrycja and SZYMAŃSKA-ZDYB, Kinga. The Effect of Conventional Cigarette Smoking on Submaximal and Maximal Heart Rate Responses and Post-exercise Heart Rate Recovery- A Narrative Review. Quality in Sport. 2026;63:73028. https://doi.org/10.12775/QS.2026.63.73028

ARTICLE TIMELINE
Received: 03.06.2026. Revised: 10.07.2026. Accepted: 12.07.2026. Published: 12.07.2026.

The journal has been awarded 20 points in the parametric evaluation by the Polish Ministry of Higher Education and Science (Annex to the announcement of 05.01.2024, No. 32553). Unique Journal Identifier: 201398. Scientific disciplines assigned: Economics and finance (Field of social sciences); Management and Quality Sciences (Field of social sciences).
Punkty Ministerialne z 2019 – aktualny rok 20 punktów. Załącznik do komunikatu Ministra Szkolnictwa Wyższego i Nauki z dnia 05.01.2024 Lp. 32553. Posiada Unikatowy Identyfikator Czasopisma: 201398. Przypisane dyscypliny naukowe: Ekonomia i finanse (Dziedzina nauk społecznych); Nauki o zarządzaniu i jakości (Dziedzina nauk społecznych).

The Effect of Conventional Cigarette Smoking on Submaximal and Maximal Heart Rate Responses and Post-exercise Heart Rate Recovery- A Narrative Review

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Abstract

Background. Conventional cigarette smoking remains a major modifiable risk factor for cardiovascular and respiratory disease. Exercise testing provides a practical framework for assessing how smoking may influence submaximal heart rate, peak heart rate, and post-exercise heart rate recovery.

Aim. The aim of this narrative review was to summarize current evidence on the effect of conventional cigarette smoking on submaximal, maximal and recovery heart rate responses during physical exercise.

Material and methods. A narrative literature review was conducted using the PubMed and Scopus databases. Publications from January 2016 to April 2026 were considered, including full-text original studies, systematic reviews, and meta-analyses addressing cigarette smoking, exercise response, heart rate, autonomic regulation, physical fitness, or cardiopulmonary function.

Results. The reviewed evidence suggests that smoking is associated with altered cardiac autonomic regulation, reduced cardiorespiratory fitness, impaired pulmonary function, and delayed post-exercise recovery. Differences during moderate submaximal exercise may be subtle, whereas limitations become more apparent at ventilatory or lactate thresholds, during peak exercise, and during recovery.

Conclusions. Conventional cigarette smoking may impair the integrated cardiovascular, respiratory, and metabolic response to physical exercise, even in young and physically active individuals. Smoking status and post-exercise heart rate recovery should be considered in sport, clinical, and rehabilitation settings.

Keywords: cigarette smoking, tobacco smoking, heart rate, exercise test, heart rate recovery, physical exertion, cardiorespiratory fitness, autonomic nervous system.

1. Introduction

Conventional cigarette smoking remains a major global health problem and an important modifiable risk factor for premature morbidity and mortality. According to the World Health Organization, tobacco kills more than seven million people each year, and approximately 80% of the world's 1.3 billion tobacco users live in low- and middle-income countries. This epidemiological context is relevant to sport and exercise science because tobacco exposure affects organs and regulatory systems that determine tolerance of physical effort, including the cardiovascular, respiratory, and autonomic nervous systems (World Health Organization 2025; Gallucci et al. 2020).

The physiological response to exercise depends on the rapid integration of cardiac output, pulmonary ventilation, oxygen transport, skeletal muscle metabolism, and autonomic regulation. Heart rate is one of the most accessible indicators of this integration. Submaximal heart rate reflects the cardiovascular cost of a given workload, peak heart rate contributes to the interpretation of maximal exercise capacity, and heart rate recovery reflects the restoration of autonomic balance after exercise. Abnormal heart rate recovery has also been associated with a higher risk of cardiovascular events and all-cause mortality, supporting its clinical value beyond sport performance assessment (Qiu et al. 2017; Makhoul et al. 2020).

Smoking may influence heart rate responses through several interacting mechanisms. Nicotine acutely stimulates sympathetic activity and increases cardiovascular load, whereas chronic exposure to cigarette smoke is associated with altered heart rate variability and less favorable autonomic modulation. In addition, carbon monoxide reduces oxygen transport capacity, and smoking-related oxidative stress and endothelial dysfunction may impair vascular adaptation during exercise. These mechanisms indicate that the effect of smoking cannot be reduced to a single variable, such as resting heart rate or maximal heart rate alone (Dimitriadis et al. 2022; Gallucci et al. 2020; Makhoul et al. 2020; Murgia et al. 2019).

The relationship between smoking and exercise response is particularly relevant in young adults and athletes. Aerobic training may improve selected markers of cardiac autonomic regulation in habitual smokers, but this does not prove that training neutralizes tobacco-related harm. Recent athlete-focused evidence also indicates that sport participation does not fully protect athletes from the adverse effects of smoking. Studies conducted in professional or elite athletes have reported associations between tobacco use and less favorable pulmonary function, cardiopulmonary exercise parameters, and aerobic capacity. Therefore, smoking should be considered a relevant health and performance issue even in physically trained populations (Kim et al. 2017; Chaabane et al. 2016; Jang et al. 2017; Đorđević Šaranović et al. 2019; Di Gioia et al. 2026).

The existing literature also shows that the negative influence of smoking may not be equally visible at all exercise intensities. Some studies suggest that basic responses during moderate submaximal work may remain relatively preserved, whereas abnormalities become clearer when physiological reserve is challenged at higher intensity or when post-exercise recovery is assessed. This distinction is important because it prevents overgeneralization and allows more precise interpretation of findings from different exercise protocols (Borrelli et al. 2025; Borrelli et al. 2026; Sumartiningsih et al. 2019).

Research Objective. The objective of this narrative review was to synthesize current evidence on the influence of conventional cigarette smoking on submaximal and maximal heart rate responses and post-exercise heart rate recovery, with particular attention to autonomic regulation, cardiorespiratory fitness, pulmonary function, athletic and physically active populations, and the potential effects of smoking cessation or sustained nicotine withdrawal on exercise-related outcomes (Bao et al. 2024; Ho et al. 2022; Borrelli et al. 2025; Cosgun and Oren 2026; Di Gioia et al. 2026).

Research Problems. The review addressed the following questions: how does cigarette smoking affect heart rate response during submaximal exercise; how does it influence peak exercise response and cardiorespiratory fitness; how is smoking related to post-exercise heart rate recovery; and do training status, smoking cessation, or sustained nicotine withdrawal modify these relationships (Erat et al. 2016; Pepera and Panagiota 2021; Ho et al. 2022; Kim et al. 2017; Bao et al. 2024).

Research Hypotheses. Because this is a narrative review, no statistical hypotheses were tested. The synthesis was guided by the following working assumptions: conventional cigarette smoking is associated with less favorable autonomic and cardiopulmonary responses to exercise; these effects are more evident during peak exercise and recovery than during simple submaximal work; sustained nicotine withdrawal may be associated with better exercise-related physical ability and sport performance; and smoking cessation may be associated with broader cardiovascular and selected physical fitness benefits (Bao et al. 2024; Gallucci et al. 2020; Ho et al. 2022; Qiu et al. 2017; Sumartiningsih et al. 2019).

2. Research materials and methods

2.1. Participants

This article is a narrative literature review and did not involve the recruitment of human participants or the collection of new experimental data. The populations described in the included studies were human participants, including young adults, habitual smokers, heavy smokers, male Taiwanese adults, military males, professional or elite athletes, and physically active adults. Participant characteristics were interpreted according to the original study designs and inclusion criteria reported by the authors (Borrelli et al. 2025; Ho et al. 2022; Jang et al. 2017; Su et al. 2020).

2.2. Procedure / Test protocol / Measure / Instruments

A narrative literature search was conducted using the PubMed and Scopus databases. The search focused on publications from January 2016 to April 2026. The following keywords and combinations were used: cigarette smoking, tobacco smoking, smokers, conventional cigarette smoking, heart rate, exercise heart rate, heart rate response, heart rate recovery, chronotropic response, exercise test, cardiopulmonary exercise testing, submaximal exercise, maximal exercise, physical fitness, cardiorespiratory fitness, autonomic regulation, and heart rate variability. When necessary, the search was supplemented by screening the reference lists of relevant open-access articles.

The inclusion criteria comprised English-language, full-text publications involving humans and addressing the relationship between conventional cigarette smoking or smoking status and exercise response, heart rate response, heart rate recovery, physical fitness, pulmonary function, autonomic regulation, or cardiopulmonary exercise variables. Original studies, systematic reviews, and meta-analyses were eligible. Reports from public health institutions were used only as epidemiological background and were not included in the thematic synthesis of scientific evidence.

The exclusion criteria included animal studies, conference abstracts, opinion pieces without empirical evidence, articles focused exclusively on electronic cigarettes or heated tobacco products, publications without sufficient relevance to exercise response or heart rate regulation, and studies outside the predefined period. Studies involving mixed nicotine products were interpreted cautiously and were included only when they provided mechanistic information relevant to conventional tobacco smoking (Dimitriadis et al. 2022; Sumartiningsih et al. 2019).

2.3. Data collection and analysis / Statistical analysis

Data were extracted narratively from the included publications and organized into thematic categories: physiological mechanisms, submaximal and moderate exercise response, maximal exercise response, post-exercise heart rate recovery, physical fitness, athlete-specific implications, and cessation- or training-related considerations. The synthesis emphasized consistencies and differences between findings rather than pooled effect estimates. Particular attention was paid to avoiding unsupported interpretation; therefore, each substantive conclusion in the review is linked to specific included publications.

2.3.1. Statistical Software

No statistical software was used because this review did not include quantitative pooling, reanalysis of participant-level data, or formal meta-analysis. Numerical results were interpreted only as reported in the original studies.

2.3.2. AI

Artificial intelligence tools were used only for language refinement, structural organization, and editorial assistance during manuscript preparation. Source selection, interpretation of scientific evidence, assessment of relevance, and final conclusions were controlled by the authors. AI tools did not replace human judgment and were not used to generate independent scientific findings.

2.3.3. Statistical Methods

No inferential statistical tests were performed for the present review. The results were synthesized using a narrative thematic approach. The review does not claim statistical causality beyond the conclusions reported in the included original studies, systematic reviews, and meta-analyses.

3. Research results

3.1. Physiological significance of heart rate response during physical exercise

Heart rate response during exercise is a practical marker of cardiovascular adjustment to increasing metabolic demand. During dynamic exercise, heart rate rises in response to parasympathetic withdrawal and increasing sympathetic activation, allowing cardiac output to increase in proportion to skeletal muscle oxygen demand. In smokers, this regulatory process may be modified by acute nicotine exposure and chronic autonomic imbalance, which justifies the use of heart rate response as an outcome in studies of tobacco-related exercise impairment (Makhoul et al. 2020; Murgia et al. 2019; Sumartiningsih et al. 2019).

Submaximal heart rate is important because it reflects the physiological cost of workloads similar to daily activity, recreational training, and many rehabilitation sessions. A higher heart rate at a given workload may suggest reduced efficiency, whereas a blunted increase may indicate altered chronotropic or autonomic regulation. The literature on smoking suggests that submaximal responses may be heterogeneous: some studies show altered heart rate regulation after cigarette smoke exposure, whereas others indicate that moderate workloads may not reveal obvious between-group differences in basic cardiorespiratory and metabolic variables (Borrelli et al. 2025; Borrelli et al. 2026; Sumartiningsih et al. 2019).

Peak heart rate and peak exercise variables provide information about the upper limit of integrated cardiovascular, respiratory, and muscular function. In the context of smoking, maximal or peak exercise testing may reveal impairments that remain less apparent at lower intensities, including reduced aerobic capacity, altered ventilatory thresholds, or lower mechanical output. This pattern is consistent with research in young physically active adults and athletes, in whom smoking-related differences were more evident during peak exercise or cardiopulmonary exercise testing than during simple resting assessment (Borrelli et al. 2025; Di Gioia et al. 2026; Jang et al. 2017).

Heart rate recovery is a particularly informative marker because it reflects the transition from exertion to autonomic restoration. A rapid decline in heart rate after exercise is associated with efficient parasympathetic reactivation and withdrawal of sympathetic drive. Conversely, slower recovery is considered a less favorable autonomic profile and has been associated with cardiovascular risk. In smokers, impaired recovery has been observed in heavy smokers, athletes, and apparently healthy individuals without known cardiovascular disease (Cosgun and Oren 2026; Erat et al. 2016; Pepera and Panagiota 2021; Qiu et al. 2017).

3.2. Mechanisms linking cigarette smoking with altered cardiovascular regulation

The mechanisms linking cigarette smoking to exercise-related heart rate changes are multifactorial. Acute tobacco cigarette smoking can increase sympathetic nerve activity, blood pressure, and cardiovascular load. These responses may influence exercise readiness and recovery because the heart may begin exercise, or enter recovery, under greater autonomic and hemodynamic stress. Evidence comparing tobacco and electronic cigarette exposure indicates that tobacco smoking has acute effects on sympathetic activity and blood pressure, supporting a direct autonomic pathway (Dimitriadis et al. 2022).

Chronic smoking is associated with less favorable cardiac autonomic modulation. Cross-sectional data show that smokers may present altered cardiac autonomic responses, while population-based data indicate associations between smoking status, smoking history, smoking intensity, and heart rate variability. These findings suggest that cigarette smoke exposure may contribute to reduced parasympathetic modulation or relative sympathetic predominance, both of which are relevant to exercise heart rate response and recovery (Makhoul et al. 2020; Murgia et al. 2019).

Gallucci et al. emphasize several biological pathways through which smoking increases cardiovascular risk, including oxidative stress, carbon monoxide exposure, endothelial dysfunction, inflammation, and autonomic modulation. Carbon monoxide reduces oxygen-carrying capacity by binding to hemoglobin, whereas oxidative stress and endothelial dysfunction may impair vascular adaptation. These mechanisms are relevant to exercise because oxygen transport and vascular responsiveness are required to maintain adequate muscle perfusion at higher workloads (Gallucci et al. 2020).

Pulmonary mechanisms should also be considered. In athletes and physically active individuals, smoking has been associated with less favorable spirometric variables and impaired ventilatory responses. Studies in professional and elite athletes indicate that training status does not eliminate the negative respiratory consequences of tobacco use. This is important because ventilatory limitation can indirectly affect heart rate and perceived exertion by increasing the physiological cost of exercise (Chaabane et al. 2016; Đorđević Šaranović et al. 2019; Di Gioia et al. 2026).

Smoking may also influence metabolic responses to exercise. Data from young adults and physically active smokers suggest that smoking can be associated with altered thresholds, lactate-related responses, and slower adjustment of gas exchange during exercise. Although the magnitude and visibility of these effects depend on the test protocol, the evidence indicates that smoking may reduce physiological reserve rather than simply change one isolated marker (Borrelli et al. 2025; Borrelli et al. 2026; Sumartiningsih et al. 2019).

3.3. Submaximal heart rate response and moderate exercise

Submaximal exercise is central to this review because most everyday physical activity, recreational training, and rehabilitation sessions occur below maximal intensity. In clinical and sport settings, interpretation of submaximal heart rate can help determine whether a given workload imposes an unusually high cardiovascular cost or whether the chronotropic response is blunted. Smoking-related effects in this range may be subtle and therefore require careful interpretation across different protocols (Borrelli et al. 2025; Sumartiningsih et al. 2019).

Sumartiningsih et al. conducted a study in young adult male cigarette smokers in which heart rate response and heart rate variability were assessed during exercise. The authors reported that after exposure to cigarette smoke, the maximal heart rate achieved during exercise was lower than in the control condition. This finding suggests a blunted exercise-induced heart rate response and altered autonomic modulation, indicating that unfavorable cardiovascular regulation may already be present in young, apparently healthy smokers, not only in older individuals or patients with diagnosed cardiovascular disease (Sumartiningsih et al. 2019).

An important nuance is provided by Borrelli et al. in 2025. In young physically active adults, the authors reported no clear between-group differences in the main cardiorespiratory and metabolic responses between smokers and non-smokers during submaximal exercise. This finding suggests that conventional cigarette smoking may not always produce obvious abnormalities during moderate workloads, especially in young active individuals. However, the same study showed that differences became more evident at thresholds, peak exercise, and recovery, supporting the interpretation that smoking-related impairment may emerge when physiological reserve is challenged (Borrelli et al. 2025).

The 2026 study by Borrelli et al. complements this interpretation by focusing on moderate exercise and the dynamic adjustment of cardiorespiratory and gas-exchange variables. The authors reported that cigarette smoking slowed the on- and off-responses during moderate exercise in young physically active adults. Thus, even when a single steady-state heart rate value is not markedly different, the speed of physiological adjustment during the transition into and out of exercise may be less favorable in smokers (Borrelli et al. 2026).

Population-based evidence also supports a relationship between cigarette smoking and lower physical fitness. Ho et al. analyzed male Taiwanese adults and reported that current smoking was associated with less favorable health-related physical fitness, whereas former smoking was associated with more favorable results in perceived health, cardiorespiratory fitness, and muscular fitness. These results are relevant to submaximal exercise because step tests and field fitness tests often reflect the efficiency of integrated cardiovascular and muscular responses rather than peak exercise capacity alone (Ho et al. 2022).

The CHIEF study in Taiwanese military males also linked tobacco smoking with poorer physical fitness. Although military populations may differ from recreationally active adults and athletes, this study provides useful evidence because physical fitness tests in military settings often involve repeated functional efforts and tasks of submaximal to high intensity. The findings support the interpretation that smoking status should be considered when evaluating physical readiness and exercise tolerance in young adult men (Su et al. 2020).

Taken together, the evidence indicates that submaximal responses in smokers are not uniformly abnormal. Some protocols show a blunted heart rate response or slower adjustment, whereas others do not show clear differences in basic cardiorespiratory variables at moderate workloads. Therefore, submaximal findings should be interpreted alongside thresholds, recovery markers, smoking history, and fitness status rather than as isolated indicators (Borrelli et al. 2025; Borrelli et al. 2026; Ho et al. 2022; Sumartiningsih et al. 2019).

3.4. Maximal exercise, peak heart rate and cardiorespiratory fitness

Maximal or peak exercise testing reveals the upper capacity of the cardiovascular, respiratory, and muscular systems to respond to increasing demand. In smokers, limitations may become more visible under these conditions because the ability to increase ventilation, deliver oxygen, and maintain cardiac output is challenged more strongly than during moderate exercise. Studies in young adults and athletes indicate that peak exercise variables are often more sensitive to smoking-related impairment than resting or simple submaximal variables (Borrelli et al. 2025; Di Gioia et al. 2026).

In the 2025 study by Borrelli et al., young physically active smokers showed less favorable peak exercise and recovery responses than non-smokers. This study is important for the present review because it shows that smoking-related impairment may be present even in young and physically active people. At the same time, the authors' observation that submaximal differences were not prominent prevents an overly simplistic conclusion and suggests that peak exercise and recovery testing may be necessary to detect early impairment (Borrelli et al. 2025).

Jang et al. evaluated habitual smoking in taekwondo athletes and reported less favorable cardiopulmonary function in smokers. This finding is relevant because combat sport athletes often have high training loads and substantial anaerobic and aerobic demands. The presence of smoking-related differences in this group supports the interpretation that athletic participation does not fully remove the physiological burden of tobacco use (Jang et al. 2017).

Di Gioia et al. provided particularly important evidence in elite athletes assessed using spirometry and cardiopulmonary exercise testing. The authors concluded that chronic smoking was associated with early spirometric and ventilatory impairments and lower aerobic capacity despite high fitness levels. This directly supports the practical conclusion that sport participation is not sufficient to neutralize smoking-related impairment and that smoking may negatively affect performance-relevant variables while the athlete continues to smoke (Di Gioia et al. 2026).

Additional athlete-focused studies support the importance of pulmonary assessment. Chaabane et al. reported tobacco use and related health effects among professional athletes in Qatar, while Đorđević Šaranović et al. analyzed the influence of tobacco use on pulmonary function in elite athletes. These studies emphasize that the respiratory consequences of smoking are relevant even among athletes and that spirometry or respiratory monitoring may be justified when smoking is present (Chaabane et al. 2016; Đorđević Šaranović et al. 2019).

For the interpretation of maximal exercise tests, smoking should therefore be considered not only as a cardiovascular risk factor but also as a potential modifier of respiratory and metabolic performance. Lower aerobic fitness, less favorable ventilatory parameters, and altered thresholds may influence exercise prescription, athlete monitoring, and rehabilitation planning. These implications are supported by studies in physically active adults, athletes, and population-based male cohorts (Borrelli et al. 2025; Di Gioia et al. 2026; Ho et al. 2022; Su et al. 2020).

3.5. Post-exercise heart rate recovery and restoration of autonomic balance

Post-exercise heart rate recovery is one of the most clinically meaningful elements of the evidence base on smoking and exercise response. Recovery reflects the transition from sympathetic exercise drive toward parasympathetic reactivation. A delayed reduction in heart rate after exercise suggests less favorable autonomic restoration and has prognostic relevance in broader cardiovascular research (Qiu et al. 2017).

Erat et al. evaluated the heart rate recovery index in heavy smokers after treadmill exercise testing. The authors reported impaired heart rate recovery in heavy smokers and interpreted this finding as evidence of autonomic dysfunction. This study is directly relevant to the review because it links heavy smoking with a measurable post-exercise marker rather than only with resting cardiovascular risk (Erat et al. 2016).

Pepera and Panagiota compared smoker and non-smoker athletes after a step test. They reported less favorable heart rate response and recovery among athletes who smoked, suggesting that regular sport participation does not fully protect against smoking-related alterations in post-exercise autonomic behavior. This finding is relevant for field testing, where step tests are simple, inexpensive, and frequently used in sport and health assessment (Pepera and Panagiota 2021).

Cosgun and Oren extended this field by examining smokers without known cardiovascular disease during exercise testing. Their study showed abnormal recovery of heart rate and ventricular repolarization parameters after exercise. The clinical relevance of this finding lies in the possibility that impaired autonomic regulation and abnormal repolarization may contribute to arrhythmia susceptibility and sudden cardiac death risk. The broader relationship between smoking and coronary or ischemic cardiovascular risk is supported by evidence on endothelial dysfunction, oxidative stress, inflammation, and atherothrombotic mechanisms (Cosgun and Oren 2026; Gallucci et al. 2020).

Hartmann et al. reported that smoking history may influence autonomic and cerebral oxygenation responses during an acute exercise bout in smokers. Although this work does not focus exclusively on simple heart rate recovery, it reinforces the view that the effects of smoking extend beyond the peripheral cardiovascular system and may involve integrated autonomic and cerebral oxygenation responses to physical stress (Hartmann et al. 2020).

The convergence of findings across heavy smokers, athletes, and apparently healthy smokers supports the interpretation that post-exercise recovery may be a sensitive marker of smoking-related physiological burden. This is important because recovery abnormalities may be detected even when resting variables or basic submaximal responses appear acceptable (Cosgun and Oren 2026; Erat et al. 2016; Pepera and Panagiota 2021; Qiu et al. 2017).

3.6. Physical fitness, training, athletes and smoking cessation

Smoking status has practical implications for physical fitness assessment. Ho et al. studied male Taiwanese adults and reported that current smoking was associated with poorer health-related physical fitness, whereas former smoking was associated with more favorable results in several domains. This suggests that smoking cessation may have measurable benefits for physical performance and perceived health, although the magnitude of improvement depends on the population and outcome measure (Ho et al. 2022).

Bao et al. conducted a systematic review and meta-analysis on nicotine withdrawal, exercise-related physical ability, and sport performance. A key conclusion was that mid- to long-term nicotine withdrawal, especially withdrawal lasting at least three months, was associated with significant improvement in exercise-related physical ability and sports performance. This point is important for the present review because it indicates that cessation should not be viewed only as disease prevention, but also as a strategy that may improve functional and sport-related outcomes over time (Bao et al. 2024).

The benefits of exercise training among smokers should be interpreted carefully. Kim et al. reported that aerobic training improved autonomic balance in habitual smokers despite continued smoking, with favorable changes in time-domain heart rate variability indices. However, these results do not mean that training neutralizes the harmful effects of smoking. Rather, they suggest that exercise may partially improve autonomic regulation while smoking remains an independent adverse exposure (Kim et al. 2017; Makhoul et al. 2020; Murgia et al. 2019).

Athlete-focused studies are particularly useful because they challenge the assumption that high fitness fully compensates for smoking. Findings in taekwondo athletes, professional athletes from Qatar, and elite athletes assessed with cardiopulmonary testing show that smoking can still be associated with less favorable pulmonary or performance-related variables. Therefore, preventive counseling and smoking cessation support should be incorporated into athlete health programs rather than reserved only for sedentary populations (Chaabane et al. 2016; Di Gioia et al. 2026; Jang et al. 2017; Đorđević Šaranović et al. 2019).

From a practical perspective, the evidence supports a combined strategy: identify smoking status, assess exercise tolerance and recovery, provide smoking cessation counseling, and use exercise training as supportive rather than substitutive therapy. This approach is consistent with evidence on sustained nicotine withdrawal and exercise-related outcomes, broader benefits of smoking cessation, training-related autonomic improvement, and the persistent adverse effects of smoking in active populations (Bao et al. 2024; Gallucci et al. 2020; Ho et al. 2022; Kim et al. 2017; Di Gioia et al. 2026).

Table 1. Selected publications included in the narrative synthesis.

Study	Population / design	Main focus	Relevance to this review
Sumartiningsih et al. 2019	Young adult male smokers; experimental exposure	Exercise-induced heart rate response and HRV	Supports a blunted exercise-induced heart rate response after cigarette smoke exposure.
Borrelli et al. 2025	Young physically active adults	Peak exercise and recovery	Shows that impairment may be more evident at thresholds, peak exercise, and recovery than during submaximal work.
Borrelli et al. 2026	Young physically active adults	Moderate exercise transitions	Shows slower cardiorespiratory and gas-exchange adjustment during moderate exercise.
Erat et al. 2016	Heavy smokers	Heart rate recovery index	Links heavy smoking with impaired post-exercise autonomic recovery.
Pepera and Panagiota 2021	Athletes who smoked and athletes who did not smoke	Step test response and recovery	Shows less favorable heart rate response and recovery in athletes who smoked.
Cosgun and Oren 2026	Smokers without known cardiovascular disease	Recovery and repolarization	Highlights arrhythmia- and repolarization-related implications of abnormal recovery.
Di Gioia et al. 2026	Elite athletes	Spirometry and CPET	Shows that high fitness does not fully protect against smoking-related impairment.
Ho et al. 2022	Male Taiwanese adults	Health-related physical fitness	Supports the negative association between current smoking and physical fitness and the potential benefit of cessation.
Kim et al. 2017	Habitual smokers undergoing training	Autonomic regulation	Shows partial autonomic improvement with aerobic training despite continued smoking.
Bao et al. 2024	Systematic review and meta-analysis	Nicotine withdrawal and performance	Supports beneficial effects of mid- to long-term nicotine withdrawal.

Source: own elaboration based on the cited publications.

3.7. Practical implications for exercise testing, sport and rehabilitation

The reviewed evidence has practical implications for exercise testing. First, smoking status should be recorded before interpreting heart rate response, cardiopulmonary exercise variables, spirometry, and recovery indices. This is particularly important when comparing athletes, physically active adults, or rehabilitation patients, because smoking may shift thresholds, reduce aerobic capacity, or delay recovery even when resting assessment appears normal (Borrelli et al. 2025; Di Gioia et al. 2026; Erat et al. 2016).

Second, practitioners should avoid assuming that a normal submaximal response excludes smoking-related impairment. The finding that smokers and non-smokers may not differ clearly during moderate steady-state work does not contradict evidence of impairment at peak exercise or during recovery. Rather, it indicates that test selection matters: recovery heart rate, ventilatory thresholds, spirometry, and cardiopulmonary exercise testing may provide a more complete profile than submaximal heart rate alone (Borrelli et al. 2025; Borrelli et al. 2026; Cosgun and Oren 2026).

Third, athlete health programs should integrate smoking prevention and cessation counseling with routine performance monitoring. Evidence from athlete populations shows that high training status does not fully prevent smoking-related pulmonary and cardiopulmonary limitations. Therefore, coaches, sport physicians, and physiotherapists should treat smoking as a performance-relevant exposure rather than only as a long-term public health risk (Chaabane et al. 2016; Di Gioia et al. 2026; Jang et al. 2017; Đorđević Šaranović et al. 2019).

Fourth, exercise prescription for smokers should consider recovery as well as workload. If post-exercise heart rate recovery is delayed, this may justify closer monitoring of training tolerance, recovery time, and progression, especially in clinical or rehabilitation settings. Such monitoring should be combined with smoking cessation support rather than interpreted as a reason to avoid exercise (Bao et al. 2024; Kim et al. 2017; Pepera and Panagiota 2021; Qiu et al. 2017).

4. Discussion

The reviewed literature indicates that the effect of conventional cigarette smoking on heart rate response to exercise is real but not uniform across all phases of exertion. During moderate submaximal work, differences between smokers and non-smokers may be absent or subtle, especially in young physically active adults. However, more demanding outcomes, such as ventilatory thresholds, peak exercise capacity, dynamic adjustment, and post-exercise recovery, reveal a less favorable profile among smokers (Borrelli et al. 2025; Borrelli et al. 2026; Sumartiningsih et al. 2019).

This pattern has important methodological implications. Studies that rely only on resting heart rate or a single submaximal measurement may underestimate the physiological burden of smoking. Conversely, protocols that include peak exercise testing, heart rate recovery, heart rate variability, spirometry, or cardiopulmonary exercise testing are more likely to capture the integrated effects of smoking on cardiovascular, respiratory, and autonomic function (Cosgun and Oren 2026; Di Gioia et al. 2026; Erat et al. 2016; Makhoul et al. 2020).

The mechanistic evidence supports a multidimensional interpretation. Smoking may impair exercise response through sympathetic activation, reduced parasympathetic modulation, oxidative stress, carbon monoxide-related limitation of oxygen transport, endothelial dysfunction, and pulmonary impairment. These mechanisms do not act separately during exercise; rather, they converge when the body must increase oxygen delivery and then restore autonomic balance after exercise (Dimitriadis et al. 2022; Gallucci et al. 2020; Murgia et al. 2019; Đorđević Šaranović et al. 2019).

The athlete literature is especially relevant to prevention. Several studies show that smoking-related impairment can be observed even in physically active or athletic populations. Di Gioia et al. demonstrated that elite athletes who smoke may show early spirometric and ventilatory impairments and lower aerobic capacity despite high fitness. This supports the conclusion that physical training is beneficial but cannot be treated as a biological shield against tobacco exposure (Chaabane et al. 2016; Di Gioia et al. 2026; Jang et al. 2017).

The evidence on training, cessation, and nicotine withdrawal should be communicated carefully. Kim et al. suggest that aerobic training can improve selected autonomic markers in habitual smokers, whereas Bao et al. indicate that mid- to long-term nicotine withdrawal may improve exercise-related physical ability and sports performance. Ho et al. support the relevance of smoking cessation for selected physical fitness outcomes, while Gallucci et al. summarize broader cardiovascular benefits of cessation. These findings are complementary: exercise training may support cardiovascular regulation, but cessation or sustained withdrawal addresses the underlying harmful exposure more directly (Bao et al. 2024; Gallucci et al. 2020; Ho et al. 2022; Kim et al. 2017; Makhoul et al. 2020).

The review also highlights the clinical value of post-exercise heart rate recovery. Impaired recovery in heavy smokers, athletes who smoke, and smokers without known cardiovascular disease suggests that recovery may be a sensitive marker of early autonomic burden. Cosgun and Oren additionally link abnormal recovery with ventricular repolarization parameters, which increases the clinical relevance of recovery assessment for arrhythmia susceptibility and sudden cardiac death risk. The wider association between smoking and ischemic cardiovascular disease is better supported by mechanisms summarized in cardiovascular risk literature (Cosgun and Oren 2026; Erat et al. 2016; Gallucci et al. 2020; Pepera and Panagiota 2021; Qiu et al. 2017).

There are limitations in the available evidence. The reviewed studies differ in age, sex distribution, training status, smoking intensity, exercise protocols, and outcomes. Some studies focus on athletes, others on general or military populations, and some assess acute exposure while others analyze chronic smoking status. These differences limit direct comparison and explain why not all studies report the same pattern of submaximal response (Borrelli et al. 2025; Ho et al. 2022; Jang et al. 2017; Su et al. 2020).

Another limitation is that many studies do not fully separate conventional cigarette smoking from other nicotine products or from lifestyle factors such as diet, sleep, alcohol use, and habitual physical activity. Although this review focused on conventional cigarette smoking, some mechanistic data involving mixed nicotine exposure were considered only when directly relevant to autonomic or cardiovascular regulation. Future studies should distinguish conventional cigarettes from electronic nicotine delivery systems and heated tobacco products more explicitly (Dimitriadis et al. 2022; Sumartiningsih et al. 2019).

Despite these limitations, the practical message is consistent. Smoking status should be recorded when interpreting exercise tests, fitness assessments, and athlete health evaluations. Heart rate recovery, spirometry, and cardiorespiratory fitness measures may add meaningful information beyond resting examination. Smoking cessation should be promoted not only for long-term disease prevention but also for preserving exercise tolerance, recovery capacity, and performance-related physiological reserve (Bao et al. 2024; Di Gioia et al. 2026; Gallucci et al. 2020; Ho et al. 2022).

5. Conclusions

Conventional cigarette smoking is associated with less favorable cardiovascular, respiratory, and autonomic responses to physical exercise. The available evidence suggests that impairment may be modest or difficult to detect during simple submaximal work, but becomes more evident during physiological transitions, threshold intensities, peak exercise, and post-exercise recovery (Borrelli et al. 2025; Borrelli et al. 2026; Sumartiningsih et al. 2019).

Post-exercise heart rate recovery appears to be a particularly important marker in smokers. Impaired recovery has been reported in heavy smokers, athletes who smoke, and smokers without known cardiovascular disease, supporting its use as a practical indicator of autonomic burden and potential cardiovascular risk (Cosgun and Oren 2026; Erat et al. 2016; Pepera and Panagiota 2021; Qiu et al. 2017).

Sport participation and high physical fitness do not fully protect against the adverse effects of smoking. Evidence from athlete populations indicates that smoking may still be associated with impaired pulmonary function, lower aerobic capacity, and less favorable cardiopulmonary exercise parameters. Therefore, smoking prevention and cessation should remain part of athlete health and performance programs (Chaabane et al. 2016; Di Gioia et al. 2026; Jang et al. 2017; Đorđević Šaranović et al. 2019).

Sustained nicotine withdrawal may improve exercise-related physical ability and sports performance, while smoking cessation is associated with broader cardiovascular and selected physical fitness benefits. Aerobic training may partially improve autonomic regulation in habitual smokers, but exercise should be considered supportive rather than a substitute for cessation because continued smoking remains an adverse cardiovascular and respiratory exposure (Bao et al. 2024; Gallucci et al. 2020; Ho et al. 2022; Kim et al. 2017).

Disclosure

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All authors have read and agreed with the published version of the manuscript.

Financing statement

This research received no external funding.

Institutional Review Board Statement

Not applicable.

Informed Consent Statement

Not applicable.

Data Availability Statement

Not applicable.

Conflict of interest

The authors deny any conflict of interest.

Declaration of the use of generative AI and AI-assisted technologies in the writing process

In preparing this work, the author(s) used ChatGPT for language improvement and grammatical correction. After using this tool/service, the author(s) have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

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