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Editorial Focus

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유산소 운동 후 흡연이 혈관기능에 미치는 악영향

네브라스카 주립대학교 오마하 캠퍼스 건강 신체 운동학과

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Cigarette Smoking after Aerobic Exercise: The Unfavorable Impacts on Vascular Function

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Background and Study Overview

It is well-accepted that vascular dysfunction plays a key role in the pathophysiology of cardiovascular diseases. Although vascular dysfunction is multi-faceted, exercise is a commonly recommended prophylactic strategy to preserve vascular function. We and others have shown that exercise training can elicit beneficial effects on vascular function (e.g., blood pressure and conduit artery function) in healthy and clinical populations[1-4]. In fact, indices of vascular function are enhanced shortly after acute exercise[5,6], suggesting that the postexercise recovery period may be a crucial component for facilitating long-term vascular adaptations[7]. Poor habits may be detrimental to this recovery window, such as cigarette smoking. Cigarette smoking is considered a common modifiable risk factor for cardiovascular diseases and is associated with arterial stiffness and endothelial dysfunction[8,9]. Since exercise is often recommended to individuals with cardiovascular risk factors to prevent disease, it is imperative to understand how smoking can impact acute exercise recovery. Previous studies have investigated cigarette smoking prior to acute exercise and revealed that this can impair normal vascular and exercise pressor responses, thus inducing greater cardiac and arterial strain[10-12]. However, until recently, the impacts of cigarette smoking on hemodynamics and conduit artery function during recovery after aerobic exercise have not been explored. In this issue of *The Korean Journal of Sports Medicine*, Cho et al.[13] investigated the effects of cigarette smoking on blood pressure and conduit artery function during recovery after an acute bout of moderate-intensity aerobic exercise.

Physically inactive male habitual smokers (n=13, age 22.3±3.4 years) participated in two study visits, which included acute moderate-intensity aerobic exercise that was immediately followed

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by either (1) cigarette smoking or (2) sham smoking in a randomized crossover design. Measurements of resting heart rate, peripheral and central blood pressures, carotid-to-femoral pulse-wave velocity, and brachial artery flow-mediated dilation were taken before and after 30 minutes of moderate-intensity aerobic exercise. This study had several noteworthy findings. First, Cho et al.[13] noted that elevations in heart rate, rate pressure product (indicator of myocardial oxygen demand), and central and peripheral blood pressures were sustained throughout the exercise recovery period after cigarette smoking compared to sham. Second, conduit artery function recovery after exercise, assessed by pulse-wave velocity and brachial artery flow-mediated dilation, was impeded in the cigarette smoking condition versus sham. Collectively, their results suggest that cigarette smoking may undermine the autonomic and vascular recovery after aerobic exercise, which may have adverse impacts on long-term physiological adaptations and vascular protection[7].

Experimental Considerations and Future Direction

Cho et al.[13] boast a comprehensive investigation of arterial function, including flow-mediated dilation and carotid-to-femoral pulse-wave velocity, which are viewed as noninvasive gold standard assessments in our field[14,15]. Despite providing evidence that cigarette smoking hinders acute aerobic exercise recovery, the authors did not directly investigate potential mechanisms. They speculated that the arterial recovery deficits were mediated by increases in inflammation and reactive oxygen species, which would attenuate nitric oxide bioavailability[16,17]. Although we tend to agree with this conjecture, these mechanisms should be characterized in future work. Investigation of plasma nitric oxide bioavailability and/or plasma oxidant status and the relationship(s) to arterial function may supply further insight into these potential mechanisms. In this case, the study must be meticulously designed to differentiate the impacts of cigarette smoking versus exercise on these markers, as exercise-induced increases in metabolism are well-understood to acutely upregulate reactive oxygen species[17].

Furthermore, even though the arterial system was thoroughly investigated, the authors did not investigate the skeletal muscle microcirculation. This may also be a critical component of vascular recovery, as the microcirculation is a key regulator of blood flow, perfusion, peripheral vascular resistance, and blood pressure regulation at rest and during exercise[18]. Of note, the authors stated that cigarette smoking attenuated peripheral blood pressure recovery, which may have been due to increased plasma catecholamines and total peripheral resistance[19,20]. Future investigation of the microcirculation, by noninvasive methods such as near-infrared spectroscopy and/or laser Doppler flowmetry, may provide further evidence to support these mechanisms.

It is important to note that the study population consisted of otherwise healthy young males who reported that they were regularly smoking for nearly 7 years. Despite being otherwise healthy, the baseline brachial flow-mediated dilation was ~6%, placing them just below the 50th percentile for their age[21]. Therefore, these individuals potentially had compromised endothelial function, which may have attenuated the exercise pressor and recovery responses regardless of postexercise cigarette use. It may be reasonable to include nonsmoking control groups in future work. Last, the trends of smoking have been changing. E-cigarette use, or vaping, has been on the rise in younger individuals[22]. Other forthcoming studies should also consider investigating the impacts of E-cigarettes on acute aerobic exercise recovery, as this area has not been well-examined.

Conclusion

This work by Cho et al.[13] is critical contribution to our field, as their findings provide the groundwork necessary to understand the impacts of cigarette smoking on vascular reactivity during aerobic exercise recovery. Future work regarding the potential mechanism(s) and long-term effects of postexercise cigarette smoking on vascular adaptation may subsequently provide a more coherent understanding of the impending dangers, thus giving better insight into the protection against vascular dysfunction and cardiovascular diseases.

Conflict of Interest

No potential conflict of interest relevant to this article was reported.

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