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Letter to the Editor

Smoking-Suppressed Heart Rate Recovery in Young Male College Students Who Regularly Exercised

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Dear Editor-in-Chief

Smoking is a major risk factor of several types of cancer and cardiovascular disease (1). According to WHO report, every year, approximately 6 million people worldwide die of heart diseases, lung cancer, or other diseases associated with tobacco smoking. If this trend continues, 8 million people are estimated to die every year by 2030 (2). From this perspective, smoking is obviously a serious health-related issue. Regular exercise is recommended for the prevention of coronary heart diseases, as it counterbalances the prevalence of these diseases (3). Heart rate response and coronary heart disease are highly correlated (4). Delayed heart rate recovery (HR_R) after exercise is considered as a predictive indicator of coronary heart disease (5). Moreover, HR_R after exercise is faster among healthy people who exercise regularly than among people who do not exercise (6). In particular, HR_R in smokers is often delayed (7). As such, regular exercise and smoking are considered to affect the HR_R after exercise, but the effect of smoking on the time to HR_R in individuals who exercise regularly has not been clarified yet.

This study aimed to investigate the HR_R rate after exercise according to smoking status in healthy college male students who regularly exercised.

Participants were categorized into either a nonsmoker group (n = 24) or a smoker group (n = 19). Those who had been smoking 10 cigarettes per day for more than 5 years were categorized as smokers. The mean ± standard deviation age of the patients in the nonsmoker and smoker groups were 20.5 ± 2.7 and 21.5 ± 2.3 years, respectively, without any significant difference ($t = -0.874$, $P = 0.393$). The participants in this study were healthy people who regularly exercised at least 3 times a week and had no history of disease or cardiac malfunction (dysfunction) on resting electrocardiography. In a treadmill exercise test (graded exercise test) using the Bruce protocol (8), VO_2max (mL·kg·min) and heart rate (bpm) were determined. HR_R was assessed at 20 (HRR_{20}), 40 (HRR_{40}), 60 (HRR_{60}), and 80 seconds (HRR_{80}) after exercise. Decreasing heart rate (%) was calculated by dividing it by the maximum heart rate (HRmax). Statistical verification was performed by performing an independent *t* test by using SPSS 19.0 (IBM, Armonk, NY, USA). Statistical significance was set at $P < 0.05$.

The resting heart rate in the nonsmoker and smoker groups were 72.4 ± 5 and 80.1 ± 5.8 bpm, indicating a significant difference ($t = -5.205$, $P < 0.001$). However, no significant difference in maximum heart rate was observed. In the nonsmoker and smoker groups, the VO_2max values according to smoking status were 57.3 ± 6.4 and 56.8 ± 4.2

mL·kg·min, respectively, without significant difference between the groups. The heart rate reduction rate at HRR_{20} was 4.46 ± 2.2 % in the non-smoker group and 2.4 ± 2.1 % in the smoker group, with significant difference between the groups (t = 2.827, P = 0.007). At HRR_{40}, the rates were 10 ± 4.4 % and 6.72 ± 1.8 %, respectively, with a significant difference between the groups (t = 3.335, P = 0.002). Similarly, at HRR_{60}, the rates were 15.2 ± 6.4 % and 10.9 % ± 2.7%, respectively, confirming a significant reduction rate (t = 3.027, P = 0.004). Finally, at HRR*80*, the heart rate reduction rates were 18.6 ± 6.9 % and 14 ± 2.2 %, respectively, showing a significant difference (t = 3.216, P = 0.003).

Our results show that even if the maximum exercise capacity of the young healthy persons who regularly exercised was not affected by smoking, their HR_R after exercise was still delayed. Moreover, we strongly suggest that even with regular exercise, the prevalence of coronary artery disease is still higher among smokers than among non-smokers. Based on our present results, we are currently conducting research on the relationship of heart rate recovery to exercise, smoking frequency, and smoking duration.

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