Endothelin-1 Levels in Young Healthy Smokers and Non-Smokers Before and After Acute Physical Stress

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Introduction: Endothelin-1 (ET-1) is a potent vasoconstrictor produced by vascular endothelial cells in response to many stimuli, including tissue injury, and its overexpression is a marker of endothelial dysfunction. However, the effect of smoking, both chronic and acute, and nicotine gum on the ET-1 response to acute physical stress in young healthy smokers has not been investigated.

Methods: Healthy smokers (n=30) and non-smokers (n=30) underwent an exercise test to exhaustion (maximal oxygen consumption). Smokers were assessed a) after 12h smoking abstinence (chronic), b) immediately after smoking one cigarette (acute), and c) immediately after chewing nicotine gum. Blood was drawn immediately pre-exercise, and at 3 minutes post-exercise. Plasma ET-1 levels were quantified using enzyme-linked immunoassay.

Results: Mean age was 29.3±7.4 years and body mass index was 24.2±3.5 kg/m². Post-exercise ET-1 levels were significantly lower than pre-exercise levels in non-smokers (p=0.0001) and smokers under all 3 conditions (p=0.009, p<0.001, p=0.05, respectively). There were no differences in pre-exercise ET-1 levels between non-smokers and smokers after abstinence; however, post-exercise ET-1 levels were higher in smokers compared with non-smokers (p=0.008). Furthermore, both absolute (p=0.04) and relative decreases in ET-1 levels (p=0.02) were significantly smaller in smokers on their abstinence day compared with non-smokers. Moreover, after acute smoking smokers had significantly higher ET-1 at rest (p=0.05), as well as a greater absolute decrease in ET-1 levels post-exercise (p=0.02) compared with their abstinence day.

Discussion: This ongoing study suggests for the first time that acute exercise decreased ET-1 levels in non-smokers and to an even greater extent in smokers. Importantly, we showed that smoking altered the ET-1 response to exercise. Furthermore, acute smoking caused a significant increase in ET-1 levels at rest. Together, these results suggest that smoking may lead to endothelial dysfunction and impaired exercise response even in young, healthy adults. Future work is needed to confirm the implicated mechanisms.

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No potential conflict of interest. This study is supported by a grant from the Canadian Institutes of Health Research.