INTRODUCTION: Conventionally, as an exercise mode which leads to reduction of arterial stiffness, it is well used that moderate-intensity continuous exercise training (MICT). High-intensity interval training (HIIT), such as HIIT program with a total exercise time of 15–30 min is highly time-effective as an exercise mode for which reduces risks of cardiovascular disease. Recent study showed that even shorter HIIT leads to an effective reduction in arterial stiffness. Therefore, HIIT is an effectively exercise therapy which can use in patients with cardiovascular diseases. Although MICT induces vasodilation by acceleration of nitric oxide (NO) production via upregulating arterial endothelial NO synthase (eNOS)/protein kinase B (Akt) signalling pathway, an underlying mechanism of HIIT effects remains unclear.

PURPOSE: This study aimed to clarify the effects of short HIIT on arterial stiffness and arterial NO production in rats.

METHODS: Forty 10-week-old male Sprague-Dawley rats were randomly divided into four groups; CON: 8-week sedentary control, MICT: treadmill running for 60 min at 30m/min, 5days/wk for 8weeks, HIIT: fourteen 20 sec swimming sessions with a weight equivalent to 14-16% of each body weight and 10 sec pause was allowed between exercise sessions, 4days/wk for 6 weeks from 12-week-old, and RT: ladder climbing, 8-10sets/day, 3days/wk for 8 weeks groups (n=10 each group). After training session, we measured aortic pulse wave velocity (aortic PWV) as an index of arterial stiffness and plasma nitrite/nitrate (NOx) concentrations and phosphorylation of eNOS and Akt in the aorta.

RESULTS: Aortic PWV was significantly reduced in both MICT and HIIT groups as compared to CON and RT groups (P<0.05), whereas there was no difference between RT and CON groups. Additionally, arterial phosphorylations of eNOS and Akt and plasma NOx levels were significantly elevated in both MICT and HIIT groups as compared to CON and RT groups (P<0.05), whereas there was no difference between RT and CON groups. Moreover, HIIT-induced reduction of aortic PWV and increase in eNOS and Akt phosphorylations and plasma NOx levels were equal to these MICT effects. Arterial eNOS phosphorylation was negatively correlated with aortic PWV in all groups (r=-0.38, P<0.05). Further study was conducted whether a single-bout high-intensity intermittent exercise accelerates NO production. After acute high-intensity intermittent exercise, plasma NOx levels were significantly elevated (P<0.05).

CONCLUSION: These results suggest that HIIT increases NO production via the upregulation of arterial Akt/eNOS signalling pathway, resulting in the reduction of arterial stiffness, despite a reduction in total exercise volume as compared with MICT.