Effects of intermittent hypobaric hypoxic exercise on cardiovascular adaptations

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Abstract  We examined the effects of acute and chronic exercise under hypobaric hypoxia on cardiovascular responses/adaptations to test the hypothesis that cardiovascular adaptations are more effective after exercise under hypobaric hypoxia than under normobaric normoxia. First, we found that a decrease in peripheral resistance and blood pressure (BP) concomitant with an increase in stroke volume (SV) and cardiac output (CO) could be more effectively induced by chronic exercise under hypobaric hypoxia, than under normoxia. Also, the decrease in peripheral resistance and BP might be attributable to a decrease in arterial stiffness and enhanced vasodilatory activity. Second, the effects of acute hypoxic exposure and exercise on arterial stiffness and vasodilatory activity were examined. Arterial stiffness decreased after acute exercise alone, but not after acute hypoxic exposure alone. However, vasodilatory activity was enhanced by hypoxic stimulus, and to greater extent after hypoxic exercise. These findings suggested that stimulation with concomitant exercise and hypoxia could bring about more beneficial vascular responses. Finally, the time course of cardiovascular adaptations to hypobaric hypoxic exercise were assessed, and we found that a significant decrease in BP, accompanied by an increase in SV and CO, occurs within one week of starting the exercise; and that there is a significant decrease in arterial stiffness by the end of the second week. These changes in cardiovascular responses persisted until the end of the training period. Our findings suggest that exercise under hypobaric hypoxia brings about more rapid and effective beneficial cardiovascular adaptations than that under normoxia.

Keywords: vascular function, arterial stiffness, peripheral resistance, cardiovascular disease

Introduction

Regular aerobic exercise reduces cardiovascular risks, for example, decreasing blood pressure and arterial stiffness, which would be associated with improved vascular function. A more essential interest is how exercise stimuli can be optimized to more effectively reduce these risks. Adding hypoxic stimuli to exercise might be a promising approach because hypoxia causes vasodilation either directly or indirectly through the production of metabolites such as nitric oxide (NO) and adenosine. This is also a process that is quite similar to the physiological responses evoked during aerobic exercise. Hence, we tested the hypothesis that exercise induces cardiovascular adaptations more effectively under hypobaric hypoxia than under normobaric normoxia.

Effects of chronic exercise under hypobaric hypoxia on cardiovascular adaptations

Twelve healthy males aged 23 to 37 (26 ± 6) years were matched for physical fitness into two groups and then randomized to groups that exercised under normobaric normoxia (N) or hypobaric hypoxia (H). Both groups performed aquatic exercise at an intensity of ~50% VO₂max under each condition for 30 min, four days per week for four weeks. The H group exercised under hypobaric hypoxic conditions corresponding to 2000 m above sea level for a total of 2.5 h per session. Before and after the experimental period, we determined VO₂max, heart rate (HR), stroke volume (SV), cardiac output (CO), and systolic (SBP), diastolic (DBP) and mean blood pressure (MBP) while cycling at an intensity of 50% VO₂max, which was determined before starting the experiment.

The VO₂max did not significantly change in both groups after four weeks of aquatic exercise. Cardiovascular responses during cycling at 50% VO₂max did not significantly change in the N group, whereas SV and CO significantly increased in the H group (P < 0.05). Among the responses of blood pressure during moderate cycling exercise, SBP did not significantly change, whereas DBP and MBP significantly decreased in the H group (P < 0.05; Fig. 1). Furthermore, total peripheral resistance (TPR) calculated as MBP divided by CO significantly decreased.
after four weeks of the training.

Regular physical activity, especially aerobic exercise, helps to improve cardiovascular structure and function. Our findings add further support to this notion and indicate that exercise under hypobaric hypoxia could induce even more beneficial cardiovascular adaptations, such as a reduction in total peripheral resistance and MBP and increase in SV and CO, than exercise under normoxia. Schobersberger et al.8) and Mair et al.9) similarly reported that three weeks of exposure at a mildly hypoxic altitude of 1700 m above sea level elicited short-term favorable effects on the cardiovascular functions of patients with metabolic syndrome. Bailey et al.10) also reported that normobaric hypoxic training is associated with significant improvements in selected metabolic risk markers and cardiovascular responses at rest and during exercise even in healthy men. In contrast, several studies have indicated that acclimatization to chronic moderate hypoxia (4000 – 5000 m above sea level) causes obvious and continuous activation of the sympathetic nervous system and elicits increases in systemic arterial pressure even in healthy humans11,12). These findings all suggested that hypoxia does not necessarily confer beneficial cardiovascular adaptations, and thus whether or not favorable effects would be elicited depends on the degree of hypoxia.

To explain the dissimilar cardiovascular adaptations induced by performing the same exercise under normoxia and hypobaric hypoxia, we evaluated the effects of chronic exercise (i.e. four weeks of aquatic exercise) under hypobaric hypoxia on arterial stiffness and vasodilatory activity by determining brachial-ankle pulse wave velocity (baPWV) and flow mediated vasodilation (FMD) 13). The measured values did not significantly differ after four weeks of training in the N group, whereas baPWV and FMD (%FMD) significantly decreased and increased, respectively (both P < 0.01), in the H group. These changes were associated with a simultaneous reduction in blood pressure during exercise. These results indicate that arterial stiffness was reduced and that vasodilatory activity was improved only after the chronic exercise under hypobaric hypoxia, which might be attributed to improved vascular endothelial function. We then confirmed these findings not only in healthy adults but also in postmenopausal women with several risk markers, who participated in an identical experimental protocol14). Overall, these favorable vascular adaptations determined in our studies could be explained partly by a decrease in peripheral resistance and blood pressure, which would be elicited by a reduction in arterial stiffness and an increase in vasodilatory activity. Furthermore, decreased peripheral resistance and blood pressure might contribute to a decrease in cardiac afterload and, consequently, an increase in SV and CO.

Vascular response to acute hypoxic exposure and hypoxic exercise

Arterial stiffness significantly decreases after acute aerobic exercise, and it occurs only in active limbs15,16). The augmentation index indicates arterial stiffness is significantly reduced in healthy men while inspiring hypoxic gas17,18). These findings indicate that a decrease in arterial stiffness would probably be more effectively induced by exercise under hypoxic conditions. The extent to which exercise or hypoxic stimuli per se, and both together, decrease arterial stiffness was compared by Nishiwaki et al.19), who assessed changes in the cardio-ankle vascular index (CAVI) as an index of arterial stiffness and FMD in the active and inactive legs after unilateral exercise under normoxic or hypoxic conditions.

Seven healthy men rested under normoxia (RN) or hypoxia (RH), and randomly performed one-legged exercise under normoxic (EN) or hypoxia (EH) on different days. The men sat on a cycling ergometer for 40 min while inspiring normoxic (20.9% O2) or hypoxic (15.5% O2) gas.
and cycled using one leg only for the last 20 min of the 40 min experimental period at an intensity corresponding to a heart rate of 115 beats·min⁻¹ for the EN and EH experiments.

The CAVI did not significantly change in either leg after 40 min under RN and RH. These data suggest that acute mild hypoxia (15.5% O₂) per se did not necessarily reduce arterial stiffness in these men. The CAVI of the active leg significantly decreased in EN and EH (P < 0.01), and to a greater extent in EH (P < 0.05) (Fig. 2), but did not significantly change in the inactive leg after either EN or EH. On the other hand, FMD significantly increased in both legs after 40 min in HR and HE (P < 0.01), but did not significantly change after NR and NE. Katayama et al.⁰² have also recently reported similar FMD findings after acute hypoxic exercise. These results indicate that arterial stiffness decreases only in active limbs, which implies the importance of exercise in improving and preventing vascular disease. Furthermore, a greater decrease in arterial stiffness can be elicited after exercise in hypoxia than in normoxia; and FMD increases only under hypoxic conditions. These findings suggest that exercise combined with hypoxia confers more beneficial vascular responses/adaptations.

Time course of cardiovascular adaptations to hypobaric hypoxic exercise

Although acute aerobic exercise can reduce arterial stiffness more effectively under hypoxic conditions than under normoxic conditions, the acute effect rapidly disappears, and the CAVI returns to the baseline value within one hour⁹⁹. Until today, the duration of exposure to hypoxia required to elicit exercise-induced chronic cardiovascular adaptations has not yet been clarified. Therefore, we evaluated the time course of cardiovascular adaptations to exercise under hypobaric hypoxia. The exercise and all other procedures were as previously described¹³. Parameters were measured before, and after one, two and four weeks of exercise.

The VO₂max did not significantly change in either group. Cardiovascular responses during moderate exercise, as well as CAVI and %FMD did not significantly change in the N group throughout the exercise period, as previously reported¹³. On the other hand, SV and CO significantly increased, and SBP and MBP significantly decreased during moderate cycling exercise after one week of exercise in the H group (P < 0.05, Fig. 3), and these effects persisted throughout the study period. Furthermore, CAVI tended to decrease after one week and significantly decreased after two weeks of exercise (P < 0.05). The decreased CAVI persisted until the end of the study period. The %FMD tended to become enhanced after one week, but the difference did not reach statistical significance throughout the study period.

These findings suggest that four 30-min exercise sessions per week, under hypobaric hypoxia equivalent to 2000 m above sea level, are sufficient to induce beneficial cardiovascular adaptations within one to two weeks, which is relatively sooner than those generally expected.

Conclusions

Our findings suggest that exercise under hypobaric hypoxia induces beneficial cardiovascular adaptations, such as a reduction in peripheral resistance and blood pressure with a decrease in arterial stiffness and increase in vasodilatory activity, an increase in SV and CO, etc., more effectively and rapidly compared to exercise under normoxia. We believe that exercise under hypobaric hypoxia...
(2000 – 2500 m above sea level) would help to prevent cardiovascular diseases by improving cardiovascular risk markers.

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References


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**Fig. 3** Changes in systolic, diastolic and mean blood pressure during moderate cycling exercise for four weeks. DBP: diastolic blood pressure; Hypo: hypobaric hypoxic exercise; MBP: mean blood pressure; Norm: normoxic exercise.

