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CHRONIC INTERMITTENT HYPOXIA INDUCES HIGH BLOOD PRESSURE AND OXIDATIVE STRESS IN RATS

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Objectives Chronic intermittent hypoxia (CIH) is a typical feature of obstructive sleep apnoea (OSA) and is associated with oxidative stress and systemic endothelial dysfunction, which leads to systemic hypertension and other cardiovascular diseases. The objective of the present study was to examine whether CIH may induce oxidative stress and high blood pressure in Wistar rats.

Methods Forty male Wistar rats, 8 weeks of age, were subjected either to CIH (nadir $\text{FIO}_2=8\pm0.5\%$, 180 s/cycle, 20 cycles/h, 8 h per day diurnally) or intermittent air (control conditions) for 28 consecutive days. Arteria caudalis blood pressure of rats were recorded at day 0, 7, 14, 21 and 28 d by Softron sphygmomanometer; Blood and tissues was collected before and after exposure.

Results There was no differences in blood pressure between groups at baseline. Compared with the baseline, CIH significantly increases the blood pressure ($p<0.01$). SBP of CIH rats were higher than that of Control rats after 3 weeks of hypoxia exposure (124.5 ± 5.2 mm Hg vs 113.6 ± 10.2 mm Hg, $p<0.01$); and after 4 weeks, SBP (127.6 ± 5.3 mm Hg vs 118.0 ± 3.3 mm Hg, $p<0.05$) and DBP (82.9 ± 4.5 mm Hg vs 73.3 ± 10.3 mm Hg, $p<0.05$) of CIH rats were both increased significantly. The tissue of myocardium and vessels in CIH did not show obvious hypoxic change. But the expression of SOD, MDA in heart tissues in CIH was obviously increased compared with that in Control group ($p<0.05$); The levels of plasm ET-1 concentration in CIH rats was increased following the time, and all higher than that in Control rats ($p<0.05$).

Conclusions From these results we conclude that CIH do increase blood pressure in Wistar rats. And increased blood pressure maybe associated with oxidative stress and increased level of ET-1.