

CENTRAL IMIDAZOLINE AND ANGIOTENSIN II RECEPTORS IN CARDIOVASCULAR RESPONSES TO CHRONIC COLD EXPOSURE IN RATS

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Clinical observation and epidemiological survey have proved that people living and working in cold areas have a high incidence of hypertension and related cardiovascular diseases. Cold winter weather makes hypertension severer and induces myocardial infarction and stroke in hypertensive patients. Thus, it is important to understand the mechanism underlying cardiovascular responses to chronic cold exposure. Our previous studies have shown that chronic exposure of rats to mild cold (5°C) induces hypertension and cardiac hypertrophy. It has been reported that central imidazoline (I) receptors play an important role in cardiovascular responses to environmental stimuli. The objective of this experiment is to determine whether activation of central I₁ receptors affects the development of cold-induced hypertension (CIH). Four groups (7/each) of Harlan Sprague-Dawley rats were used. Blood pressures (BP) of all groups were similar during the control period. Two groups were exposed to cold (41°F; 5°C), while the remaining groups kept at 25°C. One cold-exposed (CE) and 1 warm-adapted (WA) group were treated chronically, via osmotic minipumps, with an I₁-receptor agonist, rilmenidine (R; in artificial CSF, 30 µg/hr, icv) and a specific α₁-receptor blocker, SK&F-86466 (5 µg/hr, icv). The remaining groups received CSF only and served as controls. The implantation of minipumps were carried out under anesthesia (sodium pentobarbital 35 mg/kg, ip). The treatment lasted for 4 weeks. BP of the CE control group increased significantly during the first week of exposure to cold and rose to 162±5 mmHg by the 4th week, while BP of the CE, R-treated group did not increase and remained at the WA control level (118±3 mmHg). Withdrawal of R resulted in an increase in BP to the level of the CE controls at the 8th week of exposure to cold. Plasma renin activity and urine norepinephrine output were significantly decreased in the CE, R-treated group during the 4th week, suggesting inhibition of sympathetic nervous system and renin-angiotensin system. Pressor response to icv angiotensin II (AII) was significantly increased in the CE control group. However, AII-induced pressor response was absent in the CE, R-treated rats at the 4th week, indicating that AII-induced pressor effect was inhibited by activation of I₁ receptors. Inhibition of pressor response to AII disappeared at the 8th week upon withdrawal of R at the end of the 4th week. Thus, it is concluded that activation of central I₁ receptors could prevent the development of CIH and this may be related to inhibition of pressor effect of central AII. This finding also reveals possible relationship between central AII and I₁ receptors in cardiovascular responses to chronic cold exposure.

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