# Abstract

The aim of present project was to uncover the effect of pharmacological increase in acute and chronic nitric oxide (NO) production on cardioprotective effect of chronic hypoxia. We studied the effect of NO donor molsidomine on hemodynamic conditions and ischemia - induced myocardium injury. Male Wistar rats were exposed to continual hypoxia in a normobaric chamber (10 % $O_2$, 4 weeks). Rats received molsidomine either chronically (15 mg/kg/day) in drinking water or acutely (10 mg/kg) in saline infused 30 min before ischemia. Control rats were kept under normoxia and treated in a corresponding manner. Adaptation to chronic hypoxia resulted in development of pulmonary hypertension. Chronic treatment with molsidomine slightly reduced these consequences of chronic hypoxia but it had no effect on increased cardiac ischemic tolerance in chronically hypoxic rats. On the other hand acute treatment with molsidomine significantly reduced infarct size and increased the number of arrhythmias in both normoxic and chronically hypoxic animals. In conclusion, our data suggests that acute increase in availability of NO is cardioprotective in both normoxic and chronically hypoxic rats contrary to its chronic increase which seems to have no protective contribution.