A pulmonary vascular bed is low-pressure system at adult subjects. Pulmonary vessels react to hypoxia by two different processes. These are hypoxic pulmonary vasoconstriction (HPV) and hypoxic pulmonary hypertension (HPH). They differ in mechanism of origin, but there seems to be important role of reactive oxygen species and nitric oxide. It was assumed in the past that HPV is isolated reaction of small pulmonary arteries to acute hypoxia and HPH to chronic hypoxia. Recently we believe that HPH is developed on the basis HPV (Crossno, Garat et al. 2007) and remodelation of peripheral pulmonary vessels (Reid 1986).

Our main task was to learn, whether antioxidants given in the early phase of exposure to hypoxia influence pulmonary hypertension more than its late administration, in the period of already developed damage of pulmonary vessels. We have used N-acetyl-L-cysteine (NAC) as an antioxidant substance. We measured changes in resistance of pulmonary vascular bed, changes of reactivity of pulmonary vessels in dependence on concentration of oxygen in the inhalated air. Measurements were performed on the model of rat isolated perfused lungs. In addition we have observed influence of the early and late treatment of NAC on the pulmonary artery pressure at rats kept in hypoxic conditions.

Our results show that at pathogenesis of hypoxic pulmonary hypertension exerts increased production of ROS. The preventive treatment of antioxidants protects against hypoxia - induced damage of the wall of pulmonary vessels more than its treatment in the process of exposure to hypoxia. Oxidants in the early period of exposure to hypoxia determine later development of disease. The administration of antioxidants at already developed hypoxic pulmonary hypertension is without effect.