ORAL *N*-ACETYLCYSTEINE REVERSES HYPEROXIA-INDUCED OXIDATIVE STRESS-RELATED COUGH SUPPRESSION IN GUINEA PIGS

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Hyperoxia-induced lung injury is well known in animal and human studies. We have previously shown that hyperoxic exposure of guinea pigs is associated with the suppression of cough reflex. The goal of this study was to determine the effects of oral *N*-acetylcysteine (NAC) on hyperoxia-induced oxidative stress in lung tissue directed on cough reflex. The experimental group (n=8) was pretreated with NAC (200mg/kg body weight) daily for 7 days and subsequently exposed to 100% O_2 for 60 h. Hyperoxic group (positive control, n=8) received vehicle and then inhaled 100% O_2. Control group (n=8) was exposed to normoxia. Cough was induced by inhalation of citric acid aerosol at time before and after exposure to 100% O_2. Cough was also induced by mechanical stimulation of laryngopharyngeal (LPh) and tracheobronchial (TBr) region in anaesthetized animals just after the end of oxygen exposition. Our results showed a significant decrease [1(0-1.5) *vs*. 5(4-6.5); P=0.002] in citric acid-induced cough in hyperoxic animals and no significant changes (P=0.07) in animals with antioxidant therapy after hyperoxia. In addition, there was a significant interaction between antioxidant therapy and hyperoxia (P=0.005). The significant inhibition was obtained from mechanically-induced TBr and LPh cough comparing with normoxic animals in both tested group with less significance in the group pretreated with NAC. In conclusion, our results indicate that NAC attenuated hyperoxia-induced down-regulation on chemically and mechanically-induced cough.

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