HYPEROXIA-INDUCED GUINEA PIG COUGH CHALLENGE MODEL

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Hyperoxia-induced lung injury is well-known in animal and human studies. It can be considered as a bimodal process resulting from the direct action of increased reactive oxygen species and from the accumulation of inflammatory mediators within the lungs.

Because the lungs are directly exposed to high levels of oxygen there is no doubt that respiratory epithelium including sensory nerve endings is a major target for the oxidative injury that manifested in lung function changes including coughing. On the basis of available information, we supposed that hyperoxia alone or in combination with primary lung tissue injury should have a damaging effect on lung tissue including neuronal pathways regulating cough.

This review summarizes the effect of hyperoxia on the cough reflex in the guinea pig model using different concentrations of oxygen and different time of exposure. Here we carried out additional analysis of the experiments from previous studies and data obtained in model of hyperoxia were pooled and analysed for the cough reflex sensitivity.

To summarize, on contrary to low oxygen exposure high oxygen concentration evokes down-regulation of the cough reflex and suppresses the cough reflex sensitivity. Inhibition of cough was also accompanied by a decreased respiratory rate. Other findings have shown that antioxidants have the potential in reversal of hyperoxia-related cough suppression in animal model. Despite these results, many questions concerning the oxygen pulmonary toxicity and effect of hyperoxia on the cough reflex remain to be elucidated.

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