# Ozone-induced nasal hyperresponsiveness to tachykinins in guinea pigs

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## Abstract

OBJECTIVE: To assess role of hydroxyl radials in the ozone-induced upper airway hyper-responsiveness to tachykinins. METHODS: A prospective, controlled, animal model (n = 96) was performed. Half of them exposed to air (A-group, placebo) and the other half exposed to 3 ppm ozone (O-group) for 2 h. Two hours post air/ozone exposure, animals were anesthetized and equally randomized to be pretreated with one of the three treatments, including saline vehicle, dimethylthiourea (DMTU; 500 mg/kg m, a hydroxyl radical scavenger), or phosphoramidon (Phos; 2 mg/kg, an inhibitor for neutral endopeptidase). Ten minutes after pretreatment, half of the animals in each group were i.v. injected with capsaicin (2 mug/kg), and the other half were i.v. injected with substance P (10 mug/kg) to produce Evans blue dye extravasation. RESULTS: Nasal exudative response to capsaicin or substance P in O-group was found to be significantly greater than that in A-group. This ozone-induced nasal airway hyperresponsiveness was largely prevented by DMTU. Phosphoramidon produced a similar nasal airway hyperresponsiveness in the A-group, but failed to alter ozone-induced nasal airway hyperresponsiveness in O-group. In sharp contrast, only substance P, but not capsaicin, produced a laryngeal exudative response in the A-group, which was similar to that in the O-group. The laryngeal exudative response to substance P was not significantly affected by DMTU or Phos. Conculsion: In the guinea-pig model, hydroxyl radicals play a vital role in the development of ozone-induced nasal airway hyperresponsiveness to tachykinins. It is possibly mediated through the suppressive action of ozone on the tachykinin degradation.