ALLERGIC RHINITIS-INDUCED NASAL SYMPTOMS AND THEIR RELATIONSHIP TO FOS-LIKE IMMUNOREACTIVITY WITHIN THE GUINEA PIG BRAINSTEM

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Diseases of upper airways are relatively common cause of chronic cough. Neuronal plasticity seems to play an important role in its pathogenesis. In naive guinea pigs we have shown that nociceptive stimuli within the nose could induce considerable fos-like immunoreactivity (FLI) in the nucleus tractus solitarii and the region of ventral respiratory group. These neuronal populations could participate in up-regulation of cough, thus contributing to the pathogenesis of upper airway cough syndrome. Recent study addressed the question, which additional neuronal groups could reveal FLI after intranasal TRPV1 agonist capsaicin challenge in the model of trigeminal sensorineural hyperresponsiveness in guinea pigs with allergic rhinitis. Guinea pigs (n=24) sensitized with intraperitoneal ovalbumin were once weekly challenged with intranasal ovalbumin as follows: during 2 wks, 4 wk, and 6 wk (each group n=8), to develop the neural hyperresponsiveness. The nasal symptom score was evaluated. After that the animals were anaesthetized and challenged with i.n. capsaicin (15 mcl, 50 mcM) into the both nostrils. Following the survival time (1.5 to 2 hours), animals were deeply anaesthetized with sodium pentobarbitone, exsanguinated, and transcardially perfused with heparinized saline (200 ml) warmed up to body temperature and then with paraformaldehyde fixative solution (200 ml). The brainstems were removed, postfixed, and paraffin - embedded brainstem slices were processed immunohistochemically (c-fos kitt, Calbiochem, Merck, SR). The levels of FLI (counts of Fos positive neurons) in defined groups were compared. In all groups the FLI was detected bilaterally in the trigeminal complex, solitary tract nucleus, lateral reticular nucleus, and retroambigual nucleus. There were no differences between the designed groups of animals. Count of fos-positive neurons within the trigeminal complex does not correlate with the magnitude of clinical symptoms, which are known to gradually increase from challenge to challenge in this model of hyperresponsiveness. Whereas trigeminal hyperresponsiveness contributes to the up-regulation of cough in animal models, it does not induce any additional neuronal FLI at the middle medulla than that observed in naive animals. Possibly the peripheral and central neuronal plasticity employ different mechanisms or cellular mechanisms (higher level of neuronal excitation) are involved.

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