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EXPERIMENTAL MODEL OF AIRWAYS HYPERREACTIVITY INDUCED BY ALLERGEN

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Allergic asthma is a chronic inflammatory disease of the airways. Characteristic features are allergen-induced bronchial obstructive reactions, airway inflammation, structural changes, decline in lung function, and airway hyperresponsiveness. The aim of our experimental study was to prepare experimental model of allergic asthma simulated the changes of airways defence reflexes during experimentally induced asthmatic inflammation.
The changes in chough reflex, bronchoconstriction and degree of inflammation were study in ovalbumin (OVA) sensitized guinea pigs after 0, 7, 14, 21 days of allergen exposition. The cough reflex was induces by citric acid inhalation in conscious animals in double chamber bodyplethysmograph. The tracheal smooth muscle reactivity was examined by in vitro method to bronchoconstricting mediators: histamine (Hi) and acetylcholine (Ach) (10-8-10-3 mol/l) and the changes in airways resistance by in vivo method to nebulized Hi (10-6 mol/l). The BALF levels of IL-4, IL-5 and levels of exhaled nitric oxide (NO) were used as parameters of airway inflammation.
We observed after 7 days of OVA sensitization: the increase in tracheal smooth muscle contraction (in vitro) to cumulative concentration of Hi and Ach and the increase of cough reflex parameters only. After 14 days of OVA sensitization: the significant increase of tracheal smooth muscle contraction amplitude to Hi, Ach; partial increase in airways resistance; mild decline of cough parameters were observed. After 21 day of OVA sensitization: the cough parameters were significantly reduced; the airways resistance after histamine inhalation was increased; the significant degree of inflammation characterized by IL-4, IL-5, eosinophilia and by increase of exhaled NO levels were noticed.
In conclusion we can summarized that progress in asthmatic inflammation after 21 days OVA sensitization demonstrated by gradual increase in inflammatory mediators (cytokines IL-4, IL-5 and exhaled NO) caused the decline of cough reflex and strengthen of bronchoconstriction. This experimental model of allergic asthma can be used for pharmacological modulation of defence reflexes and inflammation.
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