Nitric Oxide Synthase in the Nasal Mucosa

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ABSTRACT

Objective

The amount of nitric oxide (NO) in the nasal cavity is significantly higher in allergic rhinitis patients than in healthy individuals. The purpose of this study was to identify NO synthase (NOS) isoforms (NOS-1, NOS-2, and NOS-3) in nasal mucosal tissues and examine their localization, gene expression, and protein expression in allergic rhinitis.

Methods

Male BALB/c mice were intraperitoneally administered Cryptomeria japonica 1 (Cryj1) and adjuvant (primary immunization) three times; thereafter, Cryj1 was administered daily in the sensitization group. The nasal mucosa was removed from the sensitization group and the control group (n=8). NOS-1, NOS-2, and NOS-3 in the nasal mucosa were investigated using real-time polymerase chain reaction, and western blot analysis.

INTRODUCTION

It was reported that the exhalation nitric oxide (NO) increased in bronchial asthma, and it decreased by treatment. The productive cells in asthma were regarded as inflammatory cells such as trachea epithelium cells and a macrophage, neutrophils. The most of NO in the human nasal cavity is produced in the nasal mucosa. It was reported that there is more NO in the nasal cavity of the patient with allergic rhinitis, and the amount of NO increased in the nasal cavity of the patient with allergic asthma. Furthermore, it is suggested that the NO production in the nasal cavity in the allergic patient may be mediated by NOS-2 and NOS-3. However, it is not clear which cells of the nasal mucosa produce NO. Therefore, we estimated about identification of NO synthase (NOS) isoforms (NOS-1, NOS-2, NOS-3) and examine their localization in the nasal mucosa using allergic rhinitis model and control mice.

RESULTS

The expression of NOS-1 mRNA was significantly decreased, and NOS-2 and NOS-3 mRNA levels were significantly increased in the allergic group compared to the control group. Protein expression of NOS-1 was significantly decreased and that of NOS-2 was significantly increased compared to the control group. Gene expression in the control group was compared with the allergy group (P<0.05).

CONCLUSIONS

In conclusion, this study using animal model has provided valuable insight into the specific molecular mechanisms of allergic rhinitis. The most novel finding of the present study is detailed localization of NOS by using immunohistochemistry and the quantification of NOS mRNA. It was suggested that NOS-1 and NOS-3 are involved in allergic rhinitis, and NOS-3 is believed to be the factor involved in the pathogenesis of allergic rhinitis.

REFERENCES