

# 好酸球性気管支炎の咳需容体感受性亢進のメカニズム

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# 1994 Fiscal Year Final Research Report Summary

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Mechanisms of heightened airway cough receptor sensitivity in eosinophilic bronchitis (atopic cough : eosinophilic bronchitis without asthma).

Research Project

## Project/Area Number

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04807055

## Research Category

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Grant-in-Aid for General Scientific Research (C)

## Allocation Type

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Single-year Grants

## Research Field

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Respiratory organ internal medicine

## Research Institution

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Kanazawa University

## Principal Investigator

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## Project Period (FY)

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1992 - 1994

## Keywords

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Chronic nonproductive cough / Pathophysiology / Cough receptor sensitivity / Bronchial responsiveness / Atopic cough / Cough variant asthma / Eosinophilic bronchitis

## Research Abstract

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### 1. Clinical study on chronic nonproductive cough

- (1) A new clinical entity named as "ATOPIC COUGH" has been proposed, which is different from cough variant asthma.
- (2) Bronchodilator therapy is ineffective, bronchial responsiveness to methacholine is within normal range, and airway cough receptor sensitivity to inhaled capsaicin is heightened in atopic cough.
- (3) The histologic feature is considered to be large airway eosinophilic inflammation according to the following findings : no eosinophils in bronchoalveolar lavage fluid or bronchial lavage fluid, small number of eosinophils in biopsied bronchial specimens, and almost same number of eosinophils in hypertonic saline-induced sputum as bronchial asthma.
- (4) Histamine H1-antagonists and steroids are effective for the treatment of atopic cough.

### 2. Animal experiment on eosinophilic bronchitis

- (1) Eosinophilic bronchitis model of guinea pigs was successfully developed by intranasal administration of polymyxin B twice a week for 3 weeks.
- (2) Airway cough receptor sensitivity to inhaled capsaicin was heightened but bronchial responsiveness to inhaled histamine was not increased in this model.
- (3) A neuropeptide inhibitor (FK-224) and a thromboxane receptor antagonist (S-1452) but not a beta2-agonist (procaterol) significantly reduced the heightened airway cough receptor sensitivity.
- (4) A histamine H1-antagonist (azelastin) significantly suppressed the heightened airway cough receptor sensitivity.


### 3. Conclusion


From these results, it is suggested that there are at least 2 different mechanisms for the pathophysiology of persistent nonproductive cough (pathological cough) : heightened airway cough receptor sensitivity induced by eosinophilic inflammation of the large airway in atopic cough and bronchoconstriction based on bronchial hyperresponsiveness induced by eosinophilic inflammation of the central to peripheral airways in cough variant asthma.


## Research Products (20 results)


All Other

All Publications (20 results)


[Publications] Fujimura M, et al.: "Cough receptor sensitivity and bronchial responsiveness in patients with only chronic nonproductive cough." Journal of Asthma. 31. 463-472 (1994) 

[Publications] Ogawa H, Fujimura M, et al.: "The effect of the neurokinin antagonist FK-224 on the cough response to inhaled capsaicin in a new model of guinea-pig eosinophilic bronchitis induced by intranasal polymyxin B." Clinical Autonomic Research. 4. 19-27 (1994) 


[Publications] Fujimura M, et al.: "Effect of inhaled procaterol on cough receptor sensitivity to capsaicin in patients with asthma, chronic bronchitis and normal subjects." Thorax. 48. 615-618 (1993) 


[Publications] Fujimura M, et al.: "Effects of methacholine-induced bronchoconstriction and procaterol-induced bronchodilation on cough receptor sensitivity to inhaled capsaicin and tartaric acid." Thorax. 47. 441-445 (1992) 


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