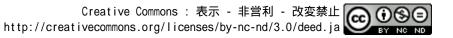
好酸球性気管支炎の咳需容体感受性亢進のメカニズ ム

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1994 Fiscal Year Final Research Report Summary

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

Research Project Project/Area Number 04807055 **Research Category** Grant-in-Aid for General Scientific Research (C) Allocation Type Single-year Grants **Research Field** Respiratory organ internal medicine **Research Institution** Kanazawa University **Principal Investigator** FUJIMURA Masaki Kanazawa University School of Medicine, The Third Department of Internal Medicine, Assistant Professor., 医学部付属病院, 講 師 (90190066) Co-Investigator(Kenkyū-buntansha) NISHI Kouichi Ishikawa Prefectual Hospital, Division of Pulmonary Medicine, Head., 呼吸器内科, 医長 **Project Period (FY)** 1992 - 1994 **Keywords** Chronic nonproductive cough / Pathophysiology / Cough receptor sensitivity / Bronchial responsiveness / Atopic cough / Cough variant asthma /

Research Abstract

Eosinophilic bronchitis

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 HI-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 polymycin 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 HI-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	I P	ublicatio	ons (2	20 re	esults)
[Publications] Fujimura M, et al.: "Cough keceptor sexsitivity and bronchial kesponsiveness in patients with only chromic no Journal of Asthma. 31. 463-472 (1994)	onpr	odu	ictive cou	ugh."		~
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[Publications] Fujimura M,et al.: "Effect of inhaled procaferol on cough rceptor sensitivity to capsaicin in patients with asth normal subjects." Thorax. 48. 615-618 (1993)	ma,	chro	onic bror	nchitis	and	~
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