Dog Ownership: A Risk Factor for Corticosteroid Resistant Asthma?

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Poster presentation by Pia Hauk, MD, February 26, 2007, at the annual meeting of the American Academy of Allergy, Asthma & Immunology

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RATIONALE: The cause of CR asthma is poorly understood. To identify new molecules that may be involved in steroid resistance, gene microarrays in airway macrophages from CR and corticosteroid-sensitive (CS) asthmatics were evaluated.

METHODS: Gene microarray analysis was performed in bronchoalveolar lavage (BAL) cells of CR and CS asthmatics. LPS levels in BAL fluid were measured by the limulus amebocyte lysate test. Dog ownership was evaluated by questionnaire; dog allergy by skin prick test. Steroid response was assessed by LPS-induced TNF-alpha production by murine BAL macrophages (24h).

RESULTS: Microarray analyses of BAL cells from 3 CR asthmatics showed significantly higher levels ($p<0.05$) of TNF-alpha, TNF-alpha-induced protein 3, early growth response 1, dual specificity phosphatase 2, and molecule possessing ankyrin repeats (8.9-, 4.9-, 12.5-, 10.5-, 7.2-fold increase, respectively) as compared to 3 CS asthmatics, identifying activation of LPS responsive genes. Ten CR asthmatics had significantly higher amounts ($p<0.01$) of LPS in BAL fluid than 8 CS asthmatics (1444±457 pg versus 271±216 pg per mg/ml of total protein). Since dogs are a significant source of LPS, we assessed dog ownership and found that 67% (12/18) of CR asthmatics lived with a dog versus 37.5% (6/16) of CS asthmatics ($p<0.05$). Dog allergy was present in 72.2% of CR and 31.3% of CS asthmatics ($p<0.01$). 24h stimulation of murine BAL macrophages with LPS resulted in loss of the ability of dexamethasone to suppress TNF-alpha production.

CONCLUSIONS: High LPS exposure by living with a dog may contribute to activation of LPS responsive genes, resulting in CR asthma.

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