

Does Nasal Hair (Vibrissae) Density Affect the Risk of Developing Asthma in Patients with Seasonal Rhinitis?

A.B. Ozturk E. Damadoglu G. Karakaya A.F. Kalyoncu

Adult Allergy Unit, Department of Chest Diseases, Hacettepe University School of Medicine, Ankara, Turkey

Key Words

Asthma · Nasal hair · Seasonal rhinitis

Abstract

Background: Large particles entering the nose are collected by nasal hair present in the anterior nares. Increased hair density provides an improvement in the filtering efficiency of the nose, while reduced amounts of nasal hair cause a decrease in its efficiency. The amount of nasal hair can vary between individuals, which can make a difference in the filtering efficiency of the nose. Reduced filter function of the nose leads to increased exposure of the airways to allergens. The aim of this study was to determine the effect of nasal hair density on the risk of developing asthma in seasonal rhinitis (SR) patients. **Methods:** A standard questionnaire was filled in, and physical examination and allergy tests were performed in 233 patients. Patients were divided into 3 groups according to the amount of nasal hair [few (few or none), moderate and many]. The association between asthma and nasal hair density was assessed. **Results:** Asthma was detected in 75 patients (32.2%), and of these, 45 (60%) also had pollen asthma. The rate of asthma was 44.7, 26.2 and 16.7% in the few, moderate and many groups, respectively ($p = 0.002$). Few nasal hairs significantly increased the risk of developing asthma [odds ratio (95% confidence interval): few, reference; moderate, 0.41 (0.21–0.78); many, 0.19 (0.06–0.55); $p = 0.002$]. **Conclusion:** Our findings suggest that the amount of nasal

hair providing a nose filtration function has a protective effect on the risk of developing asthma in SR patients. To the best of our knowledge, this is the first report on this subject in the literature.

Copyright © 2011 S. Karger AG, Basel

Introduction

The nose is strategically placed at the entrance to the airways and is an important protective organ that captures harmful particles and allergens and prevents them from entering the respiratory tract. Particles enter the nose by the nares, and those particles larger than $3\ \mu\text{m}$ accumulate in the anterior part of the nose; particles between 0.5 and $3\ \mu\text{m}$ are filtered by the nasal mucosa and transported by cilia to the nasopharynx, and the remaining particles smaller than $0.5\ \mu\text{m}$ pass easily into the lower respiratory tract [1].

Nasal hair is present in the anterior nares, and particles larger than $5\ \mu\text{m}$ passing these passageways are deposited on these hairs [2, 3]. Nostril hairs are larger than those in other parts of the body and can easily block the entrance of big particles [4]. A previous study showed that particle deposition is greater on nasal hair than in the nasal passages, and increased hair length provides a larger surface area for the particles to be deposited on, leading to an increase in filtering efficiency [2].



Fig. 1. Examples of nasal hair density in 3 different patients.

The amount and length of nasal hair can vary between individuals, causing individual differences in the filtering efficiency of the nose. Decreased filtering efficiency of the nose causes reduced clearance of allergens from the nose and respiratory tract, which can affect the risk of developing asthma in allergic rhinitis patients. However, there are no data supporting this hypothesis in the literature. Our aim was to determine the effect of nasal hair density on the risk of developing asthma in seasonal rhinitis (SR) patients using a standard questionnaire, physical examination and allergy tests.

Patients and Methods

Subjects, Study Design and Testing

We identified adult patients (14–72 years of age) with a diagnosis of SR, perennial rhinitis with seasonal exacerbation or both SR and asthma admitted to our allergy clinic between 25 May and 31 July 2009. During this period, 1,065 patients were admitted to our clinic, and 233 SR patients were enrolled in the study. The university ethics committee approved the study, and informed consent was obtained from all participants. A standard questionnaire was filled in, and physical examination and allergy tests were performed on all patients. The questionnaire was administered by two junior and two senior allergists to obtain data on demographic details (age, sex and employment), clinical features (predominant symptoms, age of onset, duration of disease), accompanying disorders (perennial rhinitis, asthma and others), smoking status, familial atopy and ear-nose-throat operation history. Main nasal, ocular and respiratory symptoms were scored by the patients. A simple scoring system was used to classify disease severity, with scores ranging from 1 to 10 (1 = very good, 10 = poor), and symptom scores were included in the questionnaire.

Anterior rhinoscopy and physical examination of the respiratory system were performed, and the presence of septum deviation was noted; nasal polyps and the amount of nasal hair were assessed by an allergist who was unaware of the patients' charac-

teristics. Patients were divided into 3 groups according to the amount of nasal hair [few (few or none), moderate and many]. Few was defined as 'few or no hairs in the nostrils, hair length very short and nasal cavity can be seen clearly', many as 'many and not counted exactly, hair length long and nasal cavity cannot be seen clearly' and moderate as 'between few and many'. Examples of few, moderate and many nasal hairs can be seen in figure 1.

Atopy was assessed by skin prick test (SPT) reactions to 13 common aeroallergens prepared by the firm Alyostal (France; *Dermatophagoides pteronyssinus*, *Phleum pratense*, *Olea europea*, *Artemisia vulgaris*, *Parietaria officinalis*, *Corylus avellana*, *Betula verrucosa*, cat, dog, *Alternaria alternata*, *Cladosporium herbarum*, *Aspergillus* mixture, *Blatella germanica*). Tests were carried out as described by Österballe and Weeke [5]. Histamine and saline were used as positive and negative controls, respectively. Resulting wheals were measured after 15 min. A positive reaction was defined as a wheal with a geometric mean diameter of at least 3 mm. SPT was not performed in cases of pregnancy, dermographism or use of antihistamines.

Sera were obtained from 28 patients to measure specific IgE levels against mixed grass pollens, for which SPTs were not performed. Specific IgE levels to mixed grass pollens were measured using the Pharmacia CAP system (Pharmacia Diagnostic AB, Uppsala, Sweden).

Asthma was diagnosed according to the Global Initiative for Asthma guidelines [6]. Pollen asthma was defined as having asthma symptoms and bronchoconstriction only during pollen season. Pulmonary function testing was performed according to the American Thoracic Society/European Respiratory Society guidelines with a spirometer (Spirolab III, MIR, Italy) [7]. A ratio of predicted forced expiratory volume in 1 s (FEV₁) to predicted forced vital capacity of less than 80% was considered to indicate airway obstruction. In this way, 75 patients were diagnosed with asthma, and among these, 45 had pollen asthma.

Statistical Analysis

The amount of nasal hair, grouped as defined above (few, moderate, many), was the main risk factor. The association between asthma and potential risk factors was investigated. χ^2 testing and Student's *t* test were used for categorical and continuous variables, respectively. An independent association between asthma and na-

sal hair was assessed using a multiple logistic regression model. Factors which were significantly associated with asthma in the univariate analysis were selected as variables for the logistic regression model. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated to indicate the model findings. Statistical significance was defined for p values less than 0.05. SPSS MS Windows Release 10.0 was used for statistical analysis.

Results

The demographic features of the patients are shown in table 1. The mean age was 31.2 ± 10.5 years, and 56.7% were women. The mean age at onset of SR was 22.7 ± 10.8 years, and the mean duration of disease was 8.7 ± 7.3 years. The initial complaints were nasal in 96.1% of the patients, ocular in 92.6% and respiratory in 47.2%. The major symptom was due to rhinitis in 55.8%, rhinoconjunctivitis in 25.3%, conjunctivitis in 10.3% and asthma in 8.6%. Patients were symptomatic for a mean period of 3.0 ± 1.7 months in a year. The mean nasal, ocular and respiratory symptom scores were 8, 6 and 5, respectively. Regarding symptom scores, there was no difference between the nasal hair density groups ($p > 0.05$).

SPTs were positive in 90.2% of the patients, and 58% of them were sensitized to a single pollen. Specific IgE levels to mixed grass pollens were measured in 28 patients, and all were positive at the level classes 2–3. All SPT-positive patients except 3 were sensitized to *Phleum pratense*. Mite, cat and dog allergens were the most common allergens to which patients were sensitized following pollen. There was no significant relationship between mono-pollen and poly-pollen sensitization and the risk of developing asthma ($p = 0.331$ and $p = 0.936$, respectively). Nor were mite, cat and dog sensitization associated with the risk of developing asthma ($p > 0.05$).

A total of 128 patients (54.9%) had at least one accompanying allergic disease. Asthma was detected in 75 patients (32.2%), and of these, 45 (60%) had pollen asthma. The FEV₁ value was between 330 and 5,220 ml, and FEV₁% was between 53 and 30% in the asthmatics. In addition, the peak expiratory flow rate was found to be between 3,270 and 11,400 ml, and percent peak expiratory flow was between 61 and 117%.

It appeared that the risk of asthma increased with age, and age >40 years carried a significantly greater risk ($p = 0.002$). Gender was not a significant risk factor for developing asthma ($p = 0.669$). Age at onset of SR did not affect the risk of developing asthma ($p = 0.257$), whereas a long duration of the disease significantly increased the risk ($p = 0.018$). Only 64 patients were smokers (current or ex-

Table 1. Demographic features and initial symptoms of SR patients (n = 233)

	Patients, n
Females	132 (56.7)
Age groups	
<20 years	33 (14.2)
21–29 years	85 (36.5)
30–39 years	66 (28.3)
>40 years	49 (21)
Smoking status	
Nonsmoker	169 (72.5)
Ex-smoker	23 (9.9)
Current smoker	41 (17.6)
Familial atopy	163 (70)
Septal deviation	46 (19.7)
Polyps	3 (1.3)
Nasal hair	
Few	94 (40.3)
Moderate	103 (44.2)
Many	36 (15.5)

Figures in parentheses are percentages.

smokers), and smoking history was not associated with the risk of developing asthma ($p = 0.466$). Atopic status was not a significant risk factor for developing asthma ($p = 0.879$), but the incidence of asthma was higher in patients with a family history of atopy ($p = 0.03$). Septal deviation and ear-nose-throat operation history had no significant relationship with the risk of developing asthma ($p > 0.05$).

With regard to nasal hair amounts, 94 patients (40.3%) were in the few group, and only one female patient in this group had had a laser procedure for nasal hair reduction. The rate of asthma was 44.7, 26.2 and 16.7% in the few, moderate and many groups, respectively ($p = 0.002$). The association between asthma, pollen asthma and nasal hair density adjusted for age, gender, SR duration, and familial atopy was examined in a multiple logistic regression model (tables 2, 3). Few nasal hairs were associated with the risk of developing asthma in SR patients [OR (95% CI): few, reference; moderate, 0.41 (0.21–0.78); many, 0.19 (0.06–0.55); $p = 0.002$]. Many nasal hairs were significantly correlated with a very low risk of developing either pollen asthma (OR 0.29, 95% CI 0.08–0.96) or asthma (OR 0.19, 95% CI 0.06–0.55) compared to few nasal hairs.

The association between nasal hair and asthma adjusted for other risk factors in different subgroups was

Table 2. Association between asthma and nasal hair density in the multiple logistic regression model

Variable	OR (95% CI)	p	OR (95% CI)	p
Age >40 years	1.03 (1.00–1.06)	0.01	1.04 (1.01–1.07)	0.02
Duration of SR	1.02 (0.98–1.06)	0.24	1.02 (0.98–1.07)	0.27
Female gender	0.66 (0.33–1.29)	0.22	0.74 (0.39–1.43)	0.37
Familial atopy	2.27 (1.09–4.73)	0.02	2.25 (1.09–4.66)	0.03
Nasal hair				
Few		p for trend: 0.002	1	
Moderate	0.40 (0.21–0.78)	0.007	–	
Many	0.18 (0.06–0.55)	0.002	–	
Mix (moderate and many)			0.35 (0.19–0.67)	<0.001

Table 3. Association between pollen asthma and nasal hair density in the multiple logistic regression model

Variable	OR (95% CI)	p	OR (95% CI)	p
Age >40 years	1.03 (1.00–1.07)	0.03	1.04 (1.00–1.08)	0.039
Duration of SR	1.02 (0.97–1.07)	0.36	1.02 (0.97–1.07)	0.374
Female gender	0.56 (0.25–1.26)	0.16	0.62 (0.28–1.35)	0.229
Familial atopy	1.99 (0.81–4.87)	0.12	1.97 (0.81–4.77)	0.135
Nasal hair				
Few	1	p for trend: 0.08	1	
Moderate	0.49 (0.22–1.09)	0.08	–	
Many	0.28	0.28 (0.08–0.96)	–	
Mix (moderate and many)			0.44 (0.20–0.96)	0.038

examined, and adjustments were made for age, gender, familial atopy and duration of SR in the multiple logistic regression analysis (table 4). Nasal hair density was associated with age and male gender. Subgroup analysis suggested similar associations in males and females for asthma risk. Smoking history changed the association between asthma and nasal hair.

Discussion

Since nasal hairs are often unwanted, many people are in the habit of cutting them. Removal or absence of nasal hair could be a risk for the development of asthma. This study shows that asthma is more common in patients with few nasal hairs. Our findings suggest that the presence of many nasal hairs seems to be more protective against the risk of developing asthma in SR patients, and to the best of our knowledge, this is the first report on this subject in the literature.

Nasal hair only prevents bigger allergen particles from entering the nose, while very small ones can pass into the airways. Therefore, what could be the mechanism of asthma in patients with few nasal hairs? McCusker et al. [8] developed a model of allergic rhinitis by local sensitization of mouse nares. In this model, mice are sensitized with low- and high-dose allergens which are primarily deposited in the upper airways, leading to allergic inflammation in both the upper and lower airways despite little or no allergen deposition in the lungs. Nasal provocation in nonasthmatic individuals with allergic rhinitis leads to increased expression of adhesion molecules and results in generalized eosinophilic allergic lower airway inflammation [9]. These findings support the hypothesis that local allergen exposure results in inflammation in both the upper and lower airways. The degree of inflammation varies depending on the concentration of allergens, and sensitization with high doses of allergens produces the greatest eosinophilic inflammatory changes [8]. Although the severity of disease in patients with different nasal hair densities seems to be similar with regard

Table 4. Association between nasal hair and asthma adjusted for other risk factors in different subgroups

	Nasal hair			Subgroup model findings ¹	
	few (few or none)	moderate/many	p value	OR	95% CI
Gender			<0.001		
Male	23 (24.5)	78 (54.1)		0.52	0.19–1.42
Female	71 (75.5)	61 (43.9)		0.28	0.12–0.65
Familial atopy			0.81		
No	27 (28.7)	38 (27.3)		0.36	0.08–1.55
Yes	67 (71.3)	101 (72.7)		0.37	0.18–0.75
Age			0.04		
<40 years	68 (72.3)	116 (83.5)		0.52	0.25–1.70
≥40 years	26 (27.7)	23 (16.5)		0.14	0.03–0.59
Smoking status			0.58		
Nonsmoker	70 (74.5)	99 (71.2)		0.26	0.12–0.55
Smoker	24 (25.5)	40 (28.8)		1.39	0.31–6.36
Atopy			0.23		
No	6 (7.0)	13 (12.1)		0.14	0.01–1.86
Yes	80 (93.0)	94 (87.9)		0.40	0.19–0.85

Figures in parentheses are percentages.

¹ Adjustments were made for age, gender, familial atopy and duration of SR in the multiple logistic regression analysis.

to symptom scores, our results suggest that people with few nasal hairs may have more allergen load in their nares, and that sensitization with high doses may cause these people to be more susceptible to asthma due to more severe upper and lower airway inflammation.

Air is conditioned by the nose while passing to the lower airways. When it is not fully conditioned by the nose, it has to be conditioned further by the lower airways, and the transferal of this function induces inflammation in the lower airways [10, 11]. A previous study showed that patients with asthma have a reduced ability to condition cold, dry air [12]. Hair has a thermoregulatory function in mammals, and evolutionary hairs have remained in some areas of the body to contribute to warming [13]. Although there are no data supporting the thermoregulatory function of nasal hair, nasal hair may also help to heat air in the nostrils. People with few nasal hairs may have less ability to heat cold air, which may contribute to the risk of developing asthma.

We investigated nasal hair density with regard to the amount and length of nasal hair and divided our patients into 3 groups of few (few or none), moderate and many nasal hairs. The method for determination of nasal hair density is totally qualitative. Although the few- and many-nasal hair groups were markedly different from each other, as shown in figure 1, there could be misclas-

sification between the moderate and many groups. Similar significant results were obtained when we combined the moderate and many groups due to this possibility and the low number of individuals with many nasal hairs. We examined the association between nasal hair and asthma adjusted for other risk factors to exclude other possible epidemiological overlap between a lot of nasal hair and lower rates of asthma. Only smoking changed the relationship between asthma and nasal hair. Male gender (with greater density of nasal hair and likelihood of chronic obstructive pulmonary disease) and the strong effect of smoking on airway hyperresponsiveness [14] could be potential explanations. This finding requires further investigation.

Another important factor is that patients with a blocked nose breathe through their mouth and consequently lose the filtration function. Septal deviation reduces the mucociliary clearance of the nose and depresses its filtration function [15]. Septoplasty may also have an impact on decreasing nasal filtration capacity by influencing the structure of the nasal valve. It is known that patients with nasal polyps are more likely to develop asthma [16], but there is no such direct relationship between the risk of developing asthma and septal deviation and septoplasty. When we investigated the contribution of nasal blockage factors such as polyps, septal deviation

and septoplasty to the risk of developing asthma, we showed that there was no significant relationship between these factors and asthma. However, these nonsignificant results may be due to the low number of individuals with nasal polyps and septal deviation.

The upper airway is the first site of allergic inflammation, and then it affects the respiratory tree. The Copenhagen Allergy Study followed a random population of 734 individuals for 8 years. Forty-eight cases of asthma were reported within 8 years, and rhinitis was present in all of these cases before the development of asthma [17]. Nasal symptoms occur before bronchial symptoms in many patients, as shown in the above study, suggesting that the nose might be preventing the development of asthma for a while. The study of McCusker et al. [8] demonstrated that local intense sensitization leads to a systemic im-

mune response more slowly. The filtering function of nasal hair might help to slow down this response for a while by decreasing allergen load and nasal inflammation. The relationship between the upper and lower airways is rather complex, and further studies are needed to clarify the interactions between the nose and bronchi.

In conclusion, this study provides new epidemiological data on the association between nasal hair density and asthma. We showed that lack of nasal hair is a significant risk factor for developing asthma in adult SR patients. This association was independent of other known risk factors for asthma, including age, sex, SR duration and familial atopy. These findings suggest that lack of hair in the nose may increase inflammation in the airways by increasing allergen load in the nose and thus make people more susceptible to asthma.

References

- Schwab JA, Zenkel M: Filtration of particulates in the human nose. *Laryngoscope* 1998; 108:120–124.
- Scott WR, Taulbee DB, Yu CP: Theoretical study of nasal deposition. *Bull Math Biol* 1978;40:581–604.
- Swift DL, Kesavanathan J: The anterior nasal passage as a fibrous filter for particles. *Chem Eng Commun* 1996;151:65–78.
- Hess WM, Seegmiller RE, Gardner JS, Allen JV, Barendregt S: Human hair morphology: a scanning electron microscopy study on a male Caucasoid and a computerized classification of regional differences. *Scanning Microsc* 1990;4:375–386.
- Österballe O, Weeke B: A new lancet for skin prick testing. *Allergy* 1979;34:187–194.
- Bateman ED, Hurd SS, Barnes PJ, Bousquet J, Drazen JM, FitzGerald M, Gibson P, Ohta K, O'Byrne P, Pedersen SE, Pizzichini E, Sullivan SD, Wenzel SE, Zar HJ: Global strategy for asthma management and prevention: GINA executive summary. *Eur Respir J* 2008;31:143–178.
- Miller MR, Crapo R, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, Enright P, van der Grinten CP, Gustafsson P, Jensen R, Johnson DC, MacIntyre N, McKay R, Navares D, Pedersen OF, Pellegrino R, Viegi G, Wanger J: ATS/ERS Task Force: General considerations for lung function testing. *Eur Respir J* 2005;26:153–161.
- McCusker C, Chicoine M, Hamid Q, Mazer B: Murine model of allergic rhinitis: role of the upper airway in lower airways disease. *J Allergy Clin Immunol* 2002;110:891–898.
- Braunstaal GJ, Overbeek SE, Kleinjan A, Prins JB, Hoogsteden HC, Fokkens WJ: Nasal allergen provocation induces adhesion molecule expression and tissue eosinophilia in upper and lower airways. *J Allergy Clin Immunol* 2001;107:469–476.
- McFadden ER Jr, Pichurko BM, Bowman HF, Ingenito E, Burns S, Dowling N, Solway J: Thermal mapping of the airways in humans. *J Appl Physiol* 1985;58:564–570.
- Karjalainen EM, Laitinen A, Sue-Chu M, Altraja A, Bjerrmer L, Laitinen LA: Evidence of airway inflammation and remodeling in ski athletes with and without bronchial responsiveness to methacholine. *Am J Respir Crit Care Med* 2000;161:2086–2091.
- Assanasen P, Baroody FM, Naureckas E, Solway J, Naclerio RM: The nasal passage of subjects with asthma has a decreased ability to warm and humidify inspired air. *Am J Respir Crit Care Med* 2001;164:1640–1646.
- Schneider MR, Schmidt-Ullrich R, Paus R: The hair follicle as a dynamic miniorgan. *Curr Biol* 2009;19:R132–R142.
- Finkelstein R, Ma HD, Ghezzi H, Whittaker K, Fraser RS, Cosio MG: Morphometry of small airways in smokers and its relationship to emphysema type and hyperresponsiveness. *Am J Respir Crit Care Med* 1995;152:267–276.
- Uslu H, Uslu C, Varoglu E, Demirci M, Seven B: Effects of septoplasty and septal deviation on nasal mucociliary clearance. *Int J Clin Pract* 2004;58:1108–1111.
- Romanet-Manent S, Charpin D, Magnan A, Lanteaume A, Vervloet D; EGEA Cooperative Group: Allergic vs nonallergic asthma: what makes the difference? *Allergy* 2002;57:607–613.
- Linneberg A, Henrik Nielsen N, Frølund L, Madsen F, Dirksen A, Jørgensen T; Copenhagen Allergy Study: The link between allergic rhinitis and allergic asthma: a prospective population-based study. *The Copenhagen Allergy Study. Allergy* 2002;57:1048–1052.