

References

1. DEMERS PA, TESCHKE K, KENNEDY SM. What to do about softwood? A review of respiratory effects and recommendations regarding exposure limits. *Am J Ind Med* 1997;**31**:385–398.
2. HAUSEN BM. Wood injuries to human health; a manual. Berlin: Walther de Gruyter, 1981.
3. KANERVA L, VAHERI E. Occupational allergic rhinitis in Finland. *Arch Occup Health* 1993;**64**:565–568.
4. SCHLÜNSSEN V, SCHAUMBURG I, SKOVSTED TA, SKOV PS. IgE-mediated allergy and exposure to wood dust. WIP-study; results from a pilot study. In: NAM '97, 45. Nordic Occupational and Environmental Meeting, 1–3 Sept 1997. Institute of Occupational Health, Denmark: p. 93.
5. SKOV PS, NORN S, WEEKE B. A new method for detecting histamine release. *Agents Actions* 1984;**14**:213–218.

Rofecoxib should be tried in NSAID hypersensitivity¹

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Key words: COX-2 inhibitors; nimesulide; NSAIDs hypersensitivity; rofecoxib.

● NONSTEROIDAL anti-inflammatory drugs (NSAIDs) are frequently involved in nonallergic reactions, and constitute a major problem, as these drugs are commonly used in medical treatment (1).

Valid alternatives for NSAID-sensitive patients present a challenge to allergists. Nimesulide has recently been reported to be a useful alternative choice in these patients (2), being tolerated in 85.4% of those with hypersensitivity to NSAIDs (3). When nimesulide also is not tolerated, few drug alternatives are available. Two patients with hypersensitivity to NSAIDs and nimesulide

who showed good tolerance to rofecoxib are reported.

Case 1. A 32-year-old woman with a history of dust-mite rhinitis and allergy to fruits suffered several episodes of eyelid angioedema after taking acetylsalicylic acid (ASA), ibuprofen, and naproxyn. An oral challenge test with nimesulide was performed, and eyelid angioedema appeared a few minutes after a dose of 100 mg had been administered. Five months later, a single-blind oral challenge test to rofecoxib 25 mg b.i.d. for 3 days was performed with good tolerance.

Case 2. A 29-year-old man with a history of cholinergic urticaria suffered several episodes of generalized urticaria and lip angioedema after ingesting ASA and metimazol. He also reported a similar episode on the third day of a 100-mg b.i.d. course of nimesulide. An oral challenge test with nimesulide was performed and elicited the same response 12 h after the 100-mg dose. The same oral challenge test at the same doses as used in case 1 was performed with good tolerance.

Although the mechanisms implicated in hypersensitivity to NSAIDs remain unknown, most adverse responses are related to cyclooxygenase (COX) inhibition, particularly the COX-1 isoenzyme (1).

Currently available NSAIDs are more potent inhibitors of COX-1 than COX-2, and provoke or exacerbate bronchial asthma, rhinitis, and urticaria/angioedema in sensitive patients. Nevertheless, some NSAIDs, such as nimesulide, preferentially inhibit COX-2 (3), and others, such as rofecoxib and celecoxib, are truly selective COX-2 inhibitors (4, 5). Thus, as these drugs reduce inflammation with lower inhibition of the production of physiologically active eicosanoids, the risk of side-effects decreases (1).

To our knowledge, these are the first cases to be reported, and they suggest that rofecoxib may be an alternative in NSAID-

sensitive patients who also do not tolerate nimesulide. Further oral challenge tests to rofecoxib in nimesulide-sensitive patients are required.

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References

1. BAYBEK S, CELIK G, EDIGER D, MUNGAN D, DEMIREL YS, MISIRLIGIL Z. The use of nimesulide in patients with acetylsalicylic acid and nonsteroidal anti-inflammatory drug intolerance. *J Asthma* 1999;**36**:657–663.
2. SÁNCHEZ-BORGES M, CAPRILES-HULETT A. Atopy is a risk factor for nonsteroidal anti-inflammatory drug sensitivity. *Ann Allergy Asthma Immunol* 2000;**84**:101–106.
3. VALERO A, MARTÍ E, GONZÁLEZ MA, GETNIA (Study group of tolerance to nimesulide in NSAIDs intolerance patients). Tolerance to nimesulide in patients with intolerance to NSAIDs [Abstract]. International Symposium on Aspirin Intolerance and Related Syndromes: a Multidisciplinary Approach. Rome, November 1999.
4. PATTERSON R, BELLO AE, LEFKOWITH J. Immunologic tolerability profile of celecoxib. *Clin Ther* 1999;**21**:2065–2079.
5. SCOTT LJ, LAMB HM. Rofecoxib. *Drugs* 1999;**58**:499–505.

U-EPX and severity of asthma

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Key words: asthma; child; peak expiratory flow rate; quality of life; severity of illness index; urinary eosinophil protein X.

● THE relationship between asthma severity and urinary concentration of eosinophil

protein X (U-EPX) in children is not clear-cut (1). As part of a study of the medical, psychological, and social determinants of quality of life (QoL) in childhood asthma, we wished to examine this relationship.

All 55 consecutive 7–9-year-old children with asthma and their families attending a hospital asthma/allergy clinic and all 16 attending a local pediatric clinic were approached. A total of 61 children (86%) participated, 36 boys and 25 girls.

U-EPX was analyzed from a frozen first morning urinary sample by the Pharmacia radioimmunoassay method. Asthma-specific QoL was measured with the Swedish version of the Paediatric Asthma Quality of Life Questionnaire (2). Peak expiratory flow rate (PEFR) was measured with a MIR Spirobank[®] portable spirometer, as the best of three values, without termination of any medication, and expressed as percentage of expected rate with adjustment for sex and height (3). Parental scoring from 0 to 3 of daytime coughing/wheezing, exercise-induced symptoms, nighttime awakening, and absence from school due to asthma was summed to a 1-week symptom score. According to the child's medical records (clinical grading), 11 children had mild asthma, 40 moderate, and 10 severe disease by the criteria given by Barnes & Godfrey (4). Nonparametric rank correlation was used for statistical analysis.

Relation to PEFR, but not general severity, in children.

Median U-EPX was 88.6 µg/mmol creatinine (95% confidence interval 67.5–135.7 µg/mmol), with no difference between boys and girls. In healthy children of the same age group, Lugosi et al. found median U-EPX 35.3 µg/mmol (95% CI 25.9–50.2 µg/mmol) with the same method of analysis as ours (5).

As seen in Table 1, U-EPX is related to PEFR, as shown earlier (5), but not to other severity measures.

The severity measures based on the child's own understanding (asthma-specific QoL), parental perception (symptom score), and the physician's interpretation (clinical grading) interrelate, indicating the validity of the measures.

In conclusion, U-EPX and PEFR reflect an aspect in common of the asthmatic response, but our findings give no further support for applying U-EPX as a general measure of disease severity in childhood asthma.

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Table 1. Urinary eosinophil protein X (U-EPX) and asthma severity measures: asthma-specific quality of life (QoL), peak expiratory flow rate (PEFR), parental symptom score, and physician's clinical grading. Rank correlation (Spearman's rho), one-tailed *P*, *n*=61

	U-EPX	<i>P</i>	QoL	<i>P</i>	PEFR	<i>P</i>	Symptom score	<i>P</i>
QoL	–0.044	0.367						
PEFR	–0.280	0.015	0.300	0.009				
Symptom score	–0.086	0.254	–0.395	0.001	–0.110	0.199		
Clinical grading	–0.025	0.425	–0.218	0.045	–0.016	0.451	0.396	0.001

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References

1. HOEKSTRA MO. Can eosinophil-derived proteins be used to diagnose or to monitor childhood asthma? *Clin Exp Allergy* 1999;29:873–874.
2. JUNIPER EF, GUYATT GH, FEENY DH, FERRIE PJ, GRIFFITH LE, TOWNSEND M. Measuring quality of life in children with asthma. *Qual Life Res* 1996;5:35–46.
3. SOLYMAR L, ARONSSON PH, BAKE B, BJURE J. Nitrogen single breath test, flow-volume curves and spirometry in healthy children, 7–18 years of age. *Eur J Respir Dis* 1980;61:275–286.
4. BARNES PJ, GODFREY S. Asthma and wheezing in children. London: Martin Dunitz, 1997.
5. LUGOSI E, HALMERBAUER G, FRISCHER F, KOLLER DY. Urinary eosinophil protein X in relation to disease activity in childhood asthma. *Allergy* 1997;52:584–588.

A new concept for treatment of sinonasal polyposis

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Key words: antileukotrienes; sinonasal polyposis; therapy.

● NASAL polyposis is considered to be a non-IgE-mediated inflammatory condition of the nose and sinuses, often associated with chronic nonallergic rhinitis, aspirin intolerance, and nonallergic asthma (1). Despite recent improvements in endonasal surgery

Oral corticosteroids in combination with antileukotrienes might be a promising approach.

(e.g., ethmoidectomy and functional endoscopic sinus surgery), diffuse nasal polyposis remains a challenge. Surgical procedures alone are insufficient to treat the underlying inflammation of the nasal