

Letters to the Editor

Exhaled nitric oxide and atopy in children

To the Editor:

Asthma is an inflammatory disease characterized by inflammatory cells and their cytokines in airway mucosa.¹ The concentration of nitric oxide in exhaled air (eNO) is a reliable marker of airway inflammation and is thus a tool in the assessment of asthma control.² Exhaled nitric oxide concentrations are increased in asthmatics,^{3,4} but it is not known whether atopy influences bronchial inflammation or the level of eNO.

We investigated whether eNO levels in atopic and nonatopic children differed depending on asthmatic status. Eighty children, aged 7 to 12 years, were recruited for the study: 20 nonatopic nonasthmatic children (group 1), 13 nonatopic asthmatic children (group 2), 27 atopic asthmatic children (group 3), and 20 atopic nonasthmatic children (group 4). Atopy was identified from specific serum IgE as measured by RAST through use of a standard battery of 8 allergens. Exhaled NO was measured online with a chemiluminescence analyzer. Lung function variables, forced vital capacity (FVC), and FEV₁ were measured with a dry spirometer after eNO measurement.

FEV₁ (mean $\pm$ SD) values were as follows: 102.7 $\pm$ 9 in group 1, 96.1 $\pm$ 2.6 in group 2, 93.7 $\pm$ 1.8 in group 3, and 99.4 $\pm$ 13.7 in group 4 (Table I). FEV₁ values did not differ significantly between the groups. Levels of eNO were higher in atopic children (groups 3 and 4, 55.0 and 36.5 ppb, respectively) than in nonatopic children (groups 1 and 2, 8.5 and 16.1 ppb, respectively; $P < .001$). Of the atopic children (groups 3 and 4), those who were asthmatic had a mean eNO of 55.0 ppb; this compared with 36.5 ppb for those who were not asthmatic ($P < .001$). Of the nonatopic children (groups 1 and 2), those who were asthmatic had a mean eNO of 16.1 ppb; this compared with 8.5 ppb for those who were not asthmatic ($P < .001$).

Forty-three atopic children had positive RAST results; of these children, 24 were positive to a single allergen and the remaining 19 were poly-primed. Of the 27 atopic asthmatic children, 16 were mono-primed and 11 were poly-primed. eNO concentrations were similar in the 16 mono-primed children (mean, 55.9 ppb) and the 11 poly-primed children (mean, 58 ppb). Of the 20 children with atopic dermatitis (group 4), 12 were poly-primed and 8 were mono-primed. eNO values were 39 ppb in the 8 mono-primed children and 34.8 ppb in the 12 poly-primed children; this difference was not significant.

This study shows that eNO levels are higher in atopic than in nonatopic children. In fact, the atopic status was associated with a 4-fold increase in eNO. The asthmatic status increased the eNO concentration only if we consider separately the atopic children (group 3 vs group 4) and the nonatopic children (group 2 vs group 1).

The source of elevated NO concentrations in atopic children is not known. Exposure to inhaled allergens in the atopic asthmatic group might have contributed to the elevated eNO concentration, but little is known about the effects of allergen exposure on sensitized children without clinical signs of asthma.

We did not detect any differences between mono-primed and poly-primed children. However, the type of allergen does not seem to affect eNO concentrations. This finding should be investigated in a larger population.

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TABLE I. Number, sex, mean age, mean eNO, and FEV₁ values in nonatopic nonasthmatic children (group 1), nonatopic asthmatic children (group 2), atopic asthmatic children (group 3), and atopic nonasthmatic children (group 4)

	Group			
	1	2	3	4
No. of children	20	13	27	20
Female	8	5	13	9
Mean age (y)	9	8.5	8	9
Mean eNO (ppb)	8.5	16.1	55.0	36.5
SD	2.2	14.9	23.6	10
FEV ₁	102.7	96.1	93.7	99.4
Asthma	—	+	+	—
Atopy	—	—	+	+

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Anaphylaxis to rice by inhalation

To the Editor:

The ingestion of rice can trigger eczema, bronchial asthma, rhinitis, or gastrointestinal symptoms, but anaphylaxis from the inhalation of rice has not hitherto been described.¹ The case we present is unusual in that an 8-year-old child was unconscious with cardiopulmonary collapse and generalized urticaria, the proximate cause of which was the inhalation of steam while rice was being cooked in his presence.

An 11-year old boy had a history of atopic dermatitis (AD) from his first year and developed severe asthma in his third year. His asthma had been brought under control from severe to mild/moderate with budesonide 100 μ g/day. At the age of 5 years, an immediate reaction, later confirmed allergic by double-blind, placebo-controlled food challenge (DBPCFC), had developed soon after he had eaten a few peanuts during a school party. He did not then have active AD but was sensitized to rice (3 mm wheal on skin prick testing) and peanut (4 mm), among other food and environmental allergens. His parents were counseled that he should avoid any sort of