

exposure to passive smoking is concerned, two issues have to be considered. The first, which has been developed in the preceding section, is that passive smoking could act to disclose an asthmatic state that was so far asymptomatic. The second issue lies in the natural history of asthma. Following Martinez *et al.*'s publications,<sup>20</sup> it has become obvious that transient wheezing could occur in non-atopic infants exposed to passive smoking. Because the relationships between passive smoking and asthma are mostly demonstrated in infants and young children, and have a tendency to weaken with increasing age, one cannot discard the hypothesis that passive smoking is responsible for transient wheezing, not for an asthmatic disease. In this field also, prospective long-term studies are clearly needed.

In conclusion, the relevance of such risk factors as allergen exposure, air pollution and passive smoking is far from being clear. Because the implication of such risk factors has enormous social consequences, such a clarification is strongly needed. More carefully designed epidemiological investigations should obviously be performed.

## References

- Charpin D, Raheison C, Dutau H, Taytard. Epidémiologie des maladies allergiques respiratoires. Données actuelles. *Rev Mal Resp* 2000; **17**: 139-158.
- Woolcock AJ, Peat JK. Evidence for the increase in asthma worldwide. In: *The Rising Trends in Asthma* (Ciba Foundation Symposium 206). Chichester: Wiley, 1997: 122-139.
- Custovic A, Smith A, Woodcock A. Indoor allergen are a primary cause of asthma. *Eur Respir Rev* 1998; **53**: 155-158.
- Green WF, Toelle B, Woolcock AJ. House dust mite increase in Wagga Wagga houses. *Aust NZ J Med* 1993; **93**: 409.
- Charpin D, Birbaum J, Haddi E, *et al.* Altitude and allergy to house-dust mites. A paradigm of the influence of environmental exposure on allergic sensitization. *Am Rev Respir Dis* 1991; **143**: 983-986.
- Peat JK, Tovey E, Toelle BG, Haby MM, Gray EJ, Mahmic A, Woolcock AJ. House dust mite allergens. A major risk factor for childhood asthma in Australia. *Am J Respir Crit Care Med* 1996; **153**: 141-146.
- Kuehr J, Frisher T, Meinert R, *et al.* Sensitization to mite allergens is a risk factor for early and late onset of asthma and for persistence of asthmatic signs in children. *J Allergy Clin Immunol* 1995; **95**: 655-662.
- Lau S, Illi S, Sommerfeld C, Niggeman B, Bergmann R, von Mutius E, Wahn U. Exposure to house-dust mite and cat allergens and development of childhood asthma. *The Lancet* 2000; **356**: 1392-1397.
- Vervloet D, de Adrade D, Pascal L, *et al.* The prevalence of reported asthma is independent of exposure in house-dust mite sensitized children. *Eur Respir J* 1999; **13**: 983-987.
- Charpin D. Pollution atmosphérique et atopie. *Rev Fr Allergol* 1996; **3**: 327-335.
- Hirsch T, Weiland SK, von Mutius E, *et al.* Inner city pollution and respiratory health and atopy in children. *Eur Respir J* 1999; **14**: 669-677.
- Braun-Fahrlander C, Wüthrich B, Gassner M, *et al.*, and the SCARPOL-Team. Prevalenz und Risikofaktoren einer allergischen Sensibilisierung bei schulkindern in der Schweiz. *Allergologie* 1999; **22**: S54-S64.
- Charpin D, Pascal L, Birnbaum J, Armendaud A, Sambuc R, Lauteaume A, Vervloet D. Gaseous air pollution and atopy. *Clin Exp Allergy* 1999; **29**: 1474-1480.
- Devereux G, Ayatllahi T, Ward R, Bromly C, Bourke SJ, Stenton SC, Hendrick DJ. Asthma, airway responsiveness and air pollution in two contrasting districts. *Thorax* 1996; **51**: 169-174.
- Anderson HR. Air pollution and trends in asthma. In: *The Rising Trends in Asthma* (Ciba Foundation Symposium 206). Chichester: Wiley, 1997: 190-207.
- Ramadour M, Dutau H, Burel C, Brisse F, Lanteaume A, Vervloet D, Charpin D. Prevalence of asthma and rhinitis in relation to long-term gaseous air pollution. *Allergy* 2000; **55**: 1163-1169.
- McDonnell WF, Abbey DE, Nishino N, Lebowitz MD. Long-term ambient ozone concentration and the incidence of asthma in non smoking adults: the Ahsmog Study. *Environ Res* 1999; **80**: 110-121.
- Halken S, Nilsson L, Taudorf E. Passive smoking as a risk factor for development of obstructive respiratory disease and allergic sensitization. *Allergy* 1995; **50**: 97-105.
- Trager IB. Smoking and childhood asthma. Were do we stand? *Am J Respir Crit Care Med* 1998; **158**: 349-351.
- Martinez FD, Morgan WJ, Wright AL, Holberg CJ, Taussig LM, Group Health Medical Associates Personnel. Diminished lung function as a predisposing factor for wheezing respiratory illness in infants. *N Engl J Med* 1995; **319**: 1112-1117.

## Fungal allergies

Nicole Nolard

Section Mycology, Scientific Institute of Public Health, Rue J. Wytsman 14, 1050 Brussels, Belgium

Tel: +32 2 642 55 18

Fax: +32 2 642 55 19

E-mail: n.nolard@iph.fgov.be

## Introduction

Inhalation of fungal spores may induce a wide range of allergic diseases: rhinitis, dermatitis, allergic bronchitis, asthma, allergic broncho-pulmonary aspergillosis, and even hypersensitivity pneumonitis.

Asexual fungal spores are among the most numerous and diversified airborne microorganisms that we breathe. Filamentous fungi are disseminating their spores in the air by thousands and, in temperate zones, hundreds of species are continuously invading our environment.

Few surveys are carried out to define the airborne fungal flora in indoor spaces. Results are often incomplete and should require further investigation. As a matter of fact, a lot of mould species grow only on specific media and, depending on the temperature of incubation, the results will be completely biased by the selection. Moreover, sedimentation sampling methods are still currently used too often (contact plates left open); they only give a restricted overview of the mycoflora since numerous species do not grow in these conditions. Furthermore, many researchers do not identify moulds up to species level. And as far as allergy is concerned, specificity is the key element when making a diagnosis.

How many allergenic species are there?

## Indoor moulds and their adverse health effects

Indoor moulds grow abundantly in dark, moist, warm and ill-ventilated places. These ideal conditions are found not only under warm and moist climates, but nowadays also in temperate zones. Indeed, excessive insulation after the oil crisis has markedly favoured condensation areas (cold bridges) from cellars to attics, which rapidly become the centre of intensive mycelial growth. A parallel change in lifestyle has led

to increasing production and accumulation of moisture in homes (frequent showers, new cooking methods, inadequate airing of bedrooms, etc.).

From 1981 to 2000, the Scientific Institute of Public Health sampled more than 500 home environments of allergic and/or asthmatic patients in urban or rural areas throughout Belgium. Results showed that more than 150 fungal species, among them *Cladosporium*, *Penicillium* and *Aspergillus*, were collected in 90–98% of the sampled environments. *Cladosporium sphaerospermum* was found in 60% of the dwellings, with the highest levels of contamination especially in bedrooms and bathrooms (hundreds of spores/m<sup>3</sup>). *C. sphaerospermum* is often associated with *Aureobasidium pullulans*, *Phoma* sp., *Acremonium strictum* and some yeast on window frames, whereas *Cladosporium herbarum*, which is an outdoor mould, does not grow in dwellings. However, its spores invade home environments through open doors and windows, mainly during the summer months.

*Aspergillus versicolor*, *Penicillium chrysogenum*, *Penicillium aurantiogriseum*, *Penicillium spinulosum*, *Penicillium brevicompactum*, *Chaetomium globosum*, *Stachybotrys chartarum*, *A. strictum* and *Alternaria alternata* are sometimes found in huge quantities on walls in bedrooms, living rooms and kitchens, most frequently inducing allergic asthma.

Mattresses are also often badly looked after, and the concentrations of dust are often quite important: 10<sup>3</sup>–10<sup>7</sup> spores/g of dust. In temperate regions, moulds, like mites, are thriving in environments that are excessively moist because of a lack of ventilation and new living conditions. In tropical zones, the conditions that stimulate the growth of moulds are found naturally. Among the most frequent species, *C. sphaerospermum*, *A. alternata*, *Epicoccum purpurascens*, *A. pullulans*, *Aspergillus restrictus* and *A. versicolor*, various species of *Mucorales* and *Trichoderma* should be pointed out. Mould strips due to *C. sphaerospermum* can even sometimes be seen at contact point of mattresses and fixed bedslats.

It should also be noted that some Basidiomycetes grow mainly in enclosed spaces. *Serpula lacrymans* or *Merulius lacrymans* is the dreadful dry rot responsible for considerable damage in dwellings. This fungus attacks damp wood and can very rapidly grow across timber and even walls. It only develops in enclosed spaces. For about 20 years now, lack of ventilation in present dwellings has made this fungus active again, and cases of sensitization have been diagnosed.

Mycotoxins associated with moulds are secondary metabolites with low molecular weight compared with allergens. It is well known that ingestion of mycotoxins (ex. aflatoxins) can cause illness and even prove fatal for Man. According to various studies, it has been established now that inhalation of the same amounts of mycotoxins is even more toxic. Trichothecenes produced by *S. chartarum* and various *Fusa-*

*rium* spp., patulin, and penicillic acid produced by various *Penicillium* have shown acute toxicity. Large areas contaminated by *S. chartarum* and *A. versicolor* can sometimes be seen on damp walls in dwellings. Great care should thus be exercised with patients living in 'musty' dwellings and complaining of irritation symptoms and nausea when at home.

B1–3 glucans, components of the walls of moulds, act as potent inflammatory agents. Their role as asthma exacerbator in musty homes should not be neglected.

In summary, moulds are linked at different stages to allergic reactions and more especially asthma:

- Some moulds (*Alternaria*, for example), like pollens, are potent allergens that can cause severe asthma, calling for emergency treatment.
- A lot of moulds produce secondary metabolites that accumulate in airborne spores. Inflammatory reactions, which play an important role in asthma, are consequently exacerbated by the inhalation of these spores.
- Some moulds produce toxins that directly work on and sensitise the bronchus and lungs.

In fact, people should not be allowed to live in environments contaminated by moulds and fungi.

## References

1. Beguin H. Mould biodiversity in homes. II. Analysis of mattress dust. *Aerobiologia* 1995; **11**: 3–10.
2. Beguin H, Nolard N. Mould biodiversity in homes. I. Air and surface analysis of 130 dwellings. *Aerobiologia* 1994; **10**: 157–166.
3. Beguin H, Nolard N. Prevalence of fungi in carpeted floor environment: analysis of dust samples from living-rooms, bedrooms, offices and school classrooms. *Aerobiologia* 1996; **12**: 113–120.
4. Beguin H, Nolard N. Relationship between mycobiota in wall-to-wall carpet dust and age of carpet. *Aerobiologia* 1999; **15**: 299–306.
5. Johanning E, ed. *Bioaerosols, Fungi and Mycotoxins: Health Effects, Assessment and Control*. Albany, NY: Eastern New York. Occupational and Environmental Health Center, 1999.
6. Macher J, ed. *Bioaerosols: Assessment and Control*. Cincinnati, OH: ACGIH, 1999.

## Environmental allergen exposure and asthma: prospects for primary prevention

Adnan Custovic<sup>CA</sup> and Ashley Woodcock  
North West Lung Centre, Wythenshawe  
Hospital, Southmoor Road, Manchester M23  
9LT, UK

<sup>CA</sup> Corresponding author

Tel: +44 161 291 2494

Fax: +44 161 291 5057

E-mail: acustovic@fs1.with.man.ac.uk

## Why are asthma and atopy increasing – relevance for prevention

The observed increase in asthma prevalence cannot be genetic in origin.<sup>1</sup> Many aspects of modern life