Indoor pollution
The role of allergy

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Indoor allergens

There is now strong evidence that sensitisation and exposure to indoor allergens is a primary cause for asthma.

Over the last 4 decades population of the developed world retreated indoors. Homes have become tighter and more energy efficient ideal for house dust mite growth.

Pet ownership increased

Substandard housing in inner cities associated with cockroach infestation.
House dust mites
Where air pollutants may be found in the home

1. Tobacco smoke
2. Animal dusts
3. Air conditioners
4. Mould, mildew and bacteria
5. Formaldehyde gas
6. Cooking and heating appliances
7. House dust mites
8. Toxic chemicals
9. Radon gas
10. Asbestos
Criteria for causality

In 1965, Hill postulated criteria to be fulfilled in order to demonstrate that an association represents a causal relationship.

In 1992 Sporik applied the criteria of Hill to the association of dust mites and asthma.
Hill postulated criteria for a causal relationship

Strength: the strength of the association is large
Consistency: repeated observations in different populations have consistent findings
Specificity: a cause leads to a specific effect
Temporality: a cause precedes an effect
Biological gradient: there is a dose-response gradient
Experiment: there is experimental evidence
Analogy: there are analogous explanations
Plausibility and coherence: the mechanism is biologically plausible
The strength of the association is large

Up to 85% of patients with asthma are skin prick sensitive to house dust mites, as compared to 5-30% of the general population.

Importance of sensitisation to allergens of domestic pets - data from Los Alamos, New Mexico, USA (altitude - 2,195m) and Scandinavia.
Consistency

Studies from many different countries have demonstrated an increased prevalence of immediate hypersensitivity to indoor allergens in patients with asthma compared to controls.
Specificity

Indoor allergens do not appear to be implicated in the aetiology of any other disorder apart from asthma

A cause precedes an effect

• Until the early 1970s asthma was almost nonexistent in Papua New Guinea with a prevalence of 0.1
• After the arrival of missionaries with blankets infested with mites prevalence of asthma rose to 7.3.
The dose-response gradient

Exposure and sensitization – already proven for mites

The higher the level of exposure the higher the risk of sensitization.

Exposure and the severity of asthma

More difficult to prove...
The experimental evidence

Reducing the exposure of patients improve their asthma control

*Studies in which patients were removed from their homes:*

To the mountains with reductions in BHR to histamine — Kerrebijn et al. 1970.


*Studies in which allergen avoidance measures were applied to patients homes*

The analogous explanations

In occupational asthma exposure to allergenic proteins present in the workplace induces asthma in the same way allergenic proteins present in the home do.

The treatment of choice is removal from the workplace resembling allergen avoidance in asthmatics sensitized to indoor allergens.
The mechanism is biologically plausible

Asthma occurs as a result of a complex interaction between genetic predisposition and the environment.

If predisposed, individuals who are continuously exposed to the allergens they are sensitized, may be at risk of developing airway inflammation and BHR which are the key features of asthma.
Facteurs d'environnement

GÉNOTYPE

Temps d'exposition à un ou plusieurs allergènes

[Diagram showing various environmental factors leading to genotype with time exposure to allergens]
200 homes of children allergic to House dust mites (SPT + symptoms)

- Without infestation: 1.50%
- With infestation: 98.50%
  - Light infestation: 4.50%
  - Moderate infestation: 8.50%
  - Strong infestation: 4%
  - Mixed infestation: 81.50%

Bedroom and living room with different degrees of infestation

78% of cases BR > LR
Pets
<table>
<thead>
<tr>
<th><strong>Main sources</strong></th>
<th>Anal sacs</th>
<th>Sebaceous glands</th>
<th>Saliva</th>
<th><strong>Dander</strong></th>
<th>Saliva</th>
<th>Skin</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Production</strong></td>
<td>Hormonal control</td>
<td>Male &gt; concentration</td>
<td>High / low producers</td>
<td>Diff. breeds</td>
<td>Same breed</td>
<td></td>
</tr>
<tr>
<td><strong>Exposition %</strong></td>
<td>21.8 - 30.8 %</td>
<td>31 - 50.2 %</td>
<td></td>
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<tr>
<td><strong>Airborne (microns)</strong></td>
<td>25 % &lt; 2.5</td>
<td>~ 20 % &lt; 5</td>
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THRESHOLD VALUES (Fel d 1)

• 8 or > mcgr/gm of dust in dust should be considered a TLV for inducing both: SENSITIZATION and ASTHMA.

• Levels < 1 mcgr/gm are not clinically significant.


• Values of 1 mcgr/gm of dust can create a RISK OF SENSITIZATION.

(3rd International Workshop of Indoor Allergens and Asthma. Cuenca, Spain -July 1995)
Lessons from the epidemiology of asthma

The unification of Germany and the impact of environment on the development of allergies in two ethnically similar populations.

Prevalence of asthma in West and East Germany

The prevalence of doctor diagnosed asthma, hay fever and atopy was higher in West Germany. Respiratory infections were higher in East Germany. Day care establishments were attended by 69-71% of East German children aged 1-3 years whereas only 6.9-8.2% of all children in this age group had access to day care in West Germany.

The prevalence of atopic sensitisation decreased significantly with increasing numbers of siblings. High rates of road traffic impaired lung function and increased respiratory symptoms in children living in West Germany, but didn’t increase the prevalence of rhinitis and asthma.

Several other authors reported similar results. An East-West European gradient is likely to exist.
Possible explanations

These findings suggest that outdoor air pollution is not a strong determinant for the development of asthma and atopy.

It seems unlikely that differences in allergen exposure may explain the difference in atopic sensitisation between East and West Germany. Factors such as early childhood exposure to viral infections could play a role in the difference of the prevalence of atopic sensitisation between East and West Germany.