



Asthma and Domestic Environment

- What is asthma (and allergy)
- Domestic environment
 - Allergies
 - Damp and mould
 - Nitrogen oxides (gas)

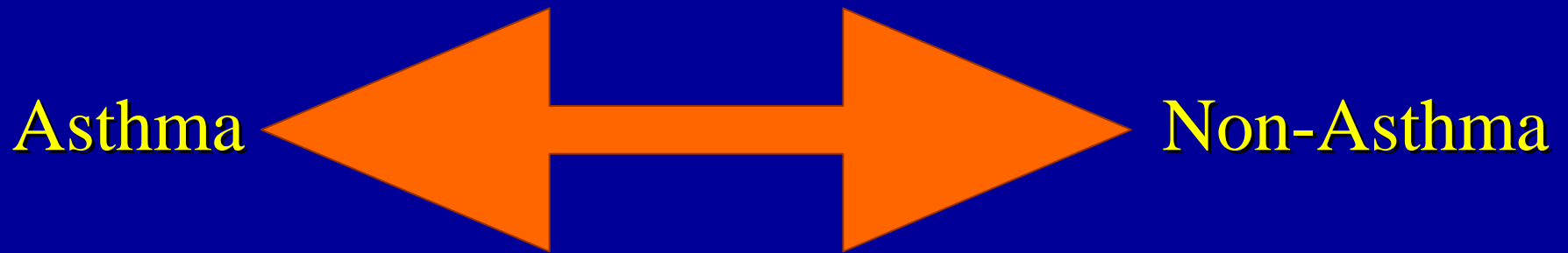
What is Asthma ??

....widespread narrowing of the bronchial airways, which changes its severity over short periods of time either spontaneously or under treatment, and is not due to cardiovascular disease.

+ bronchial hyperresponsiveness *physiology*

+ airway inflammation *pathology*

Descriptions of Asthma



wheezy bronchitis

occupational asthma

cough variant asthma

post viral wheeze

Allergic – Eosinophil

Non allergic – Neutrophil

Asthma Epidemiology

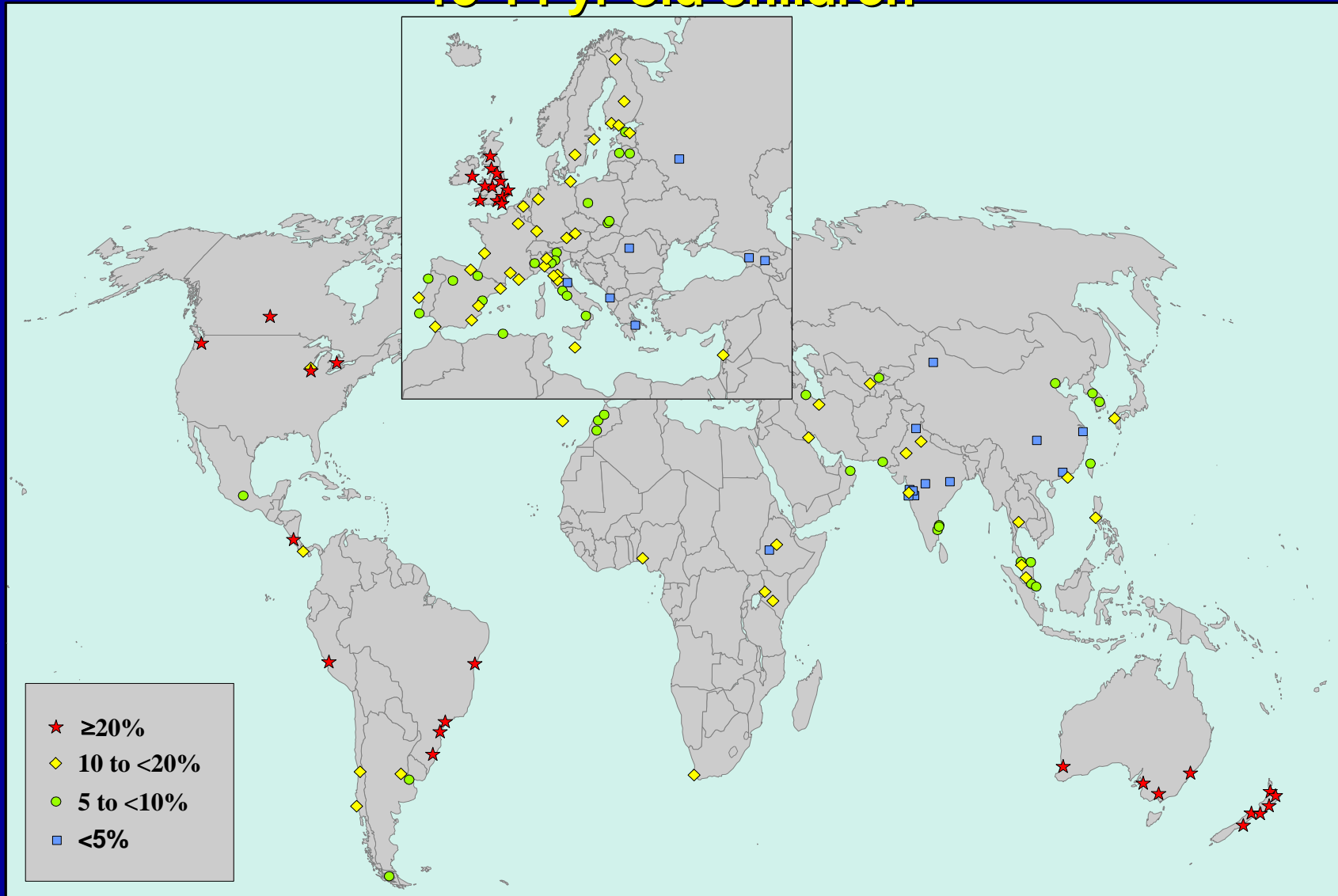
Wheeze = Asthma

Melbourne children prevalence Wheeze = 54%

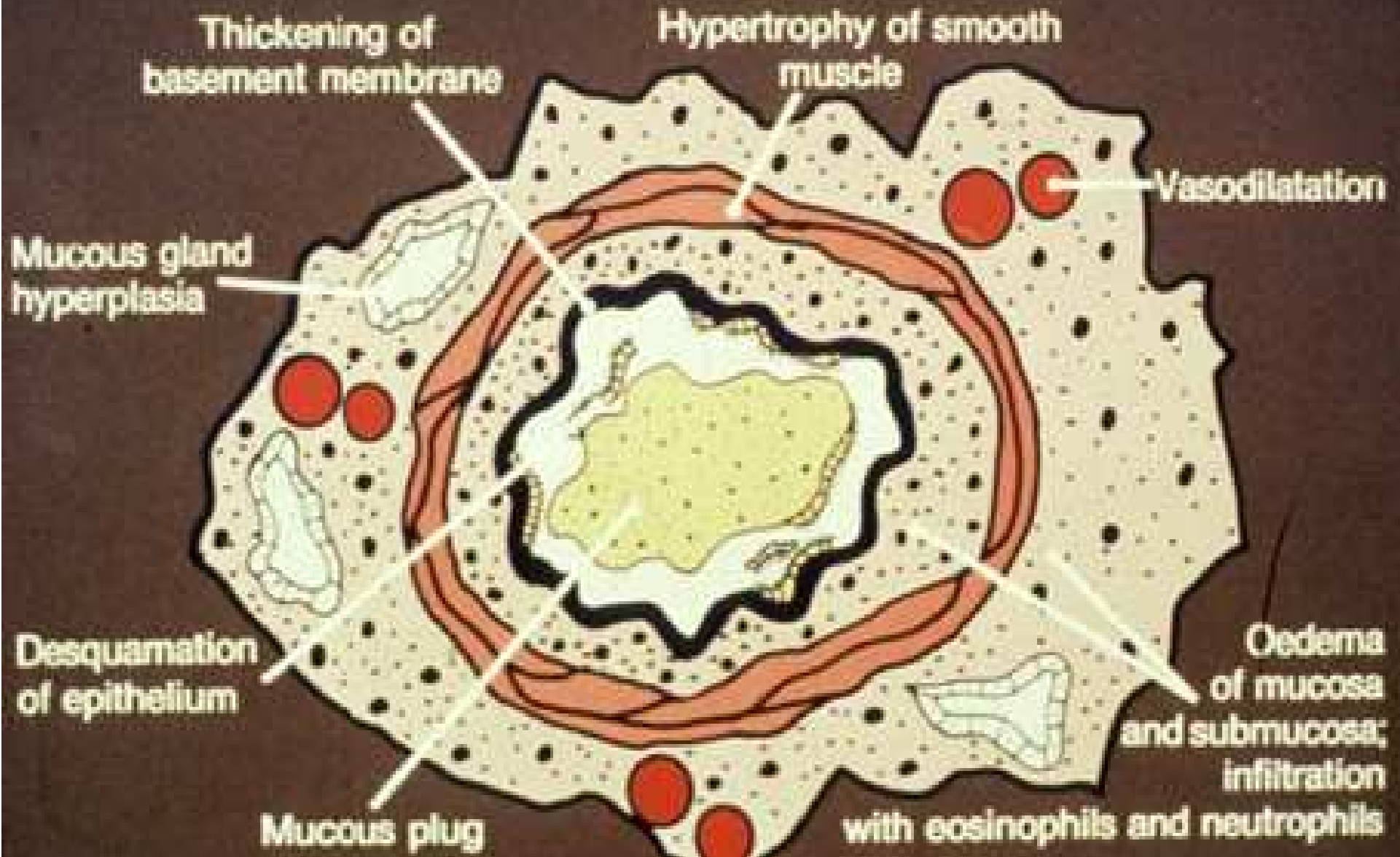
Dunedin Cohort age 26 years – 70% 1 report wheeze
50% 2 or more

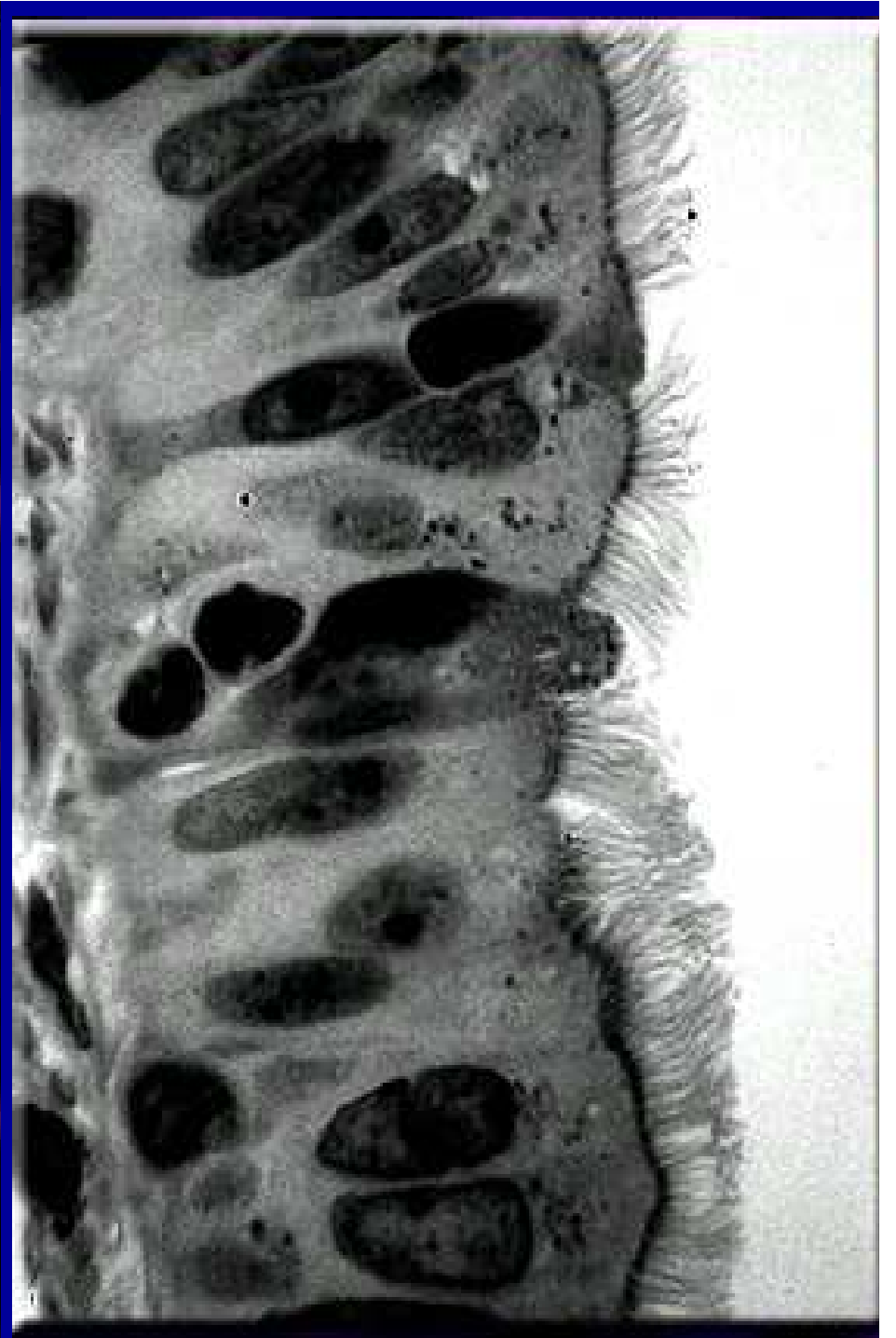
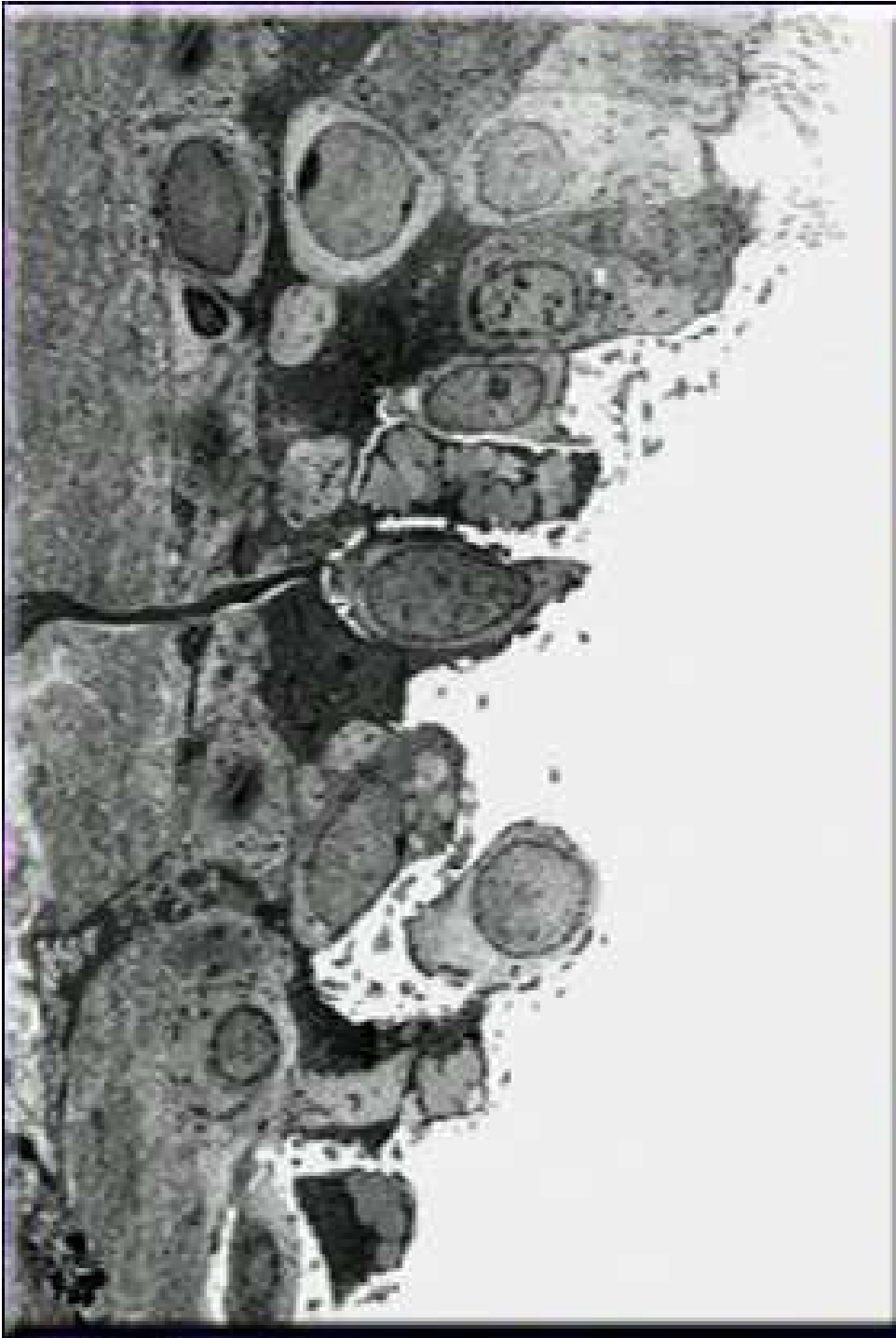
Perfect 'data' wheeze = 100% by 20 years age

12 month period prevalence of asthma symptoms in 13-14 yr old children



PATHOLOGICAL CHANGES IN ASTHMA

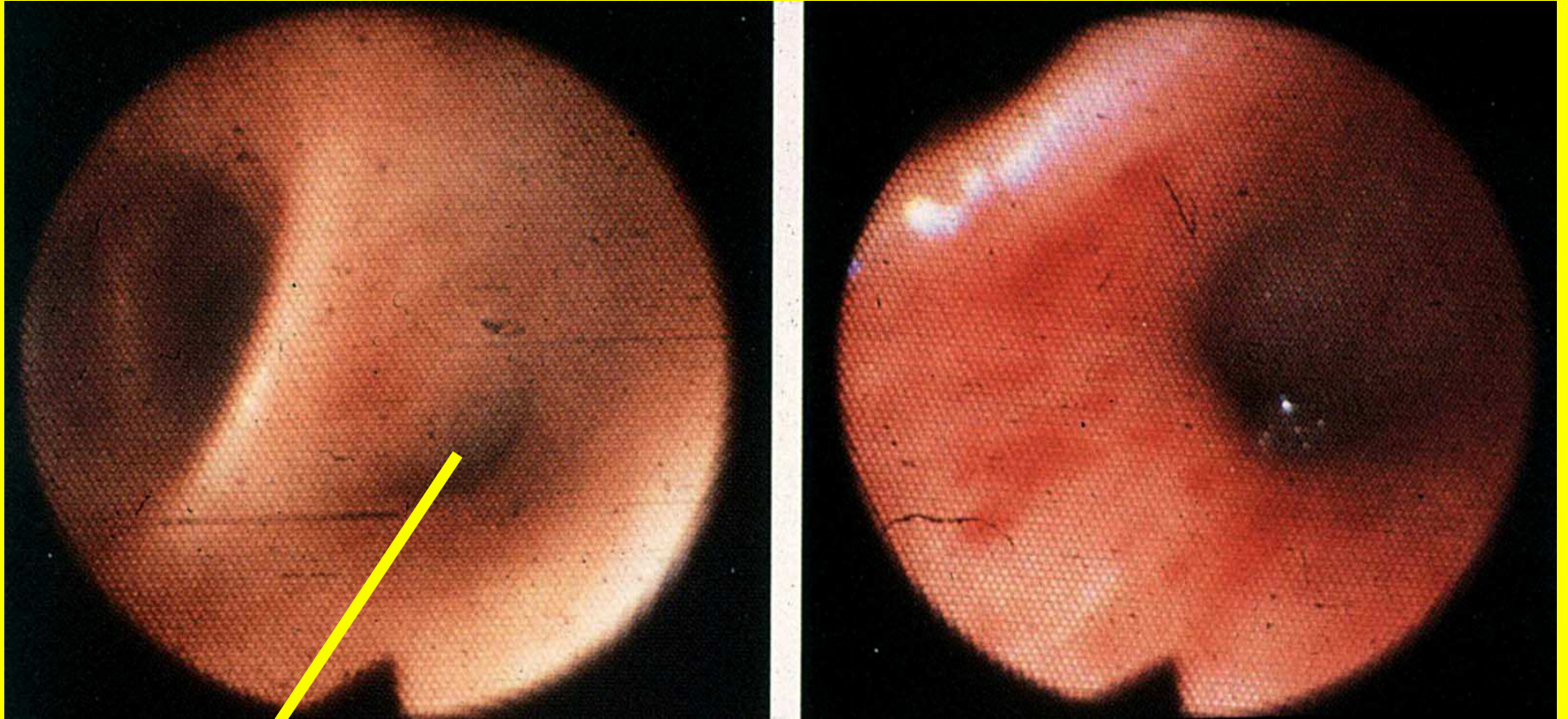




ALLERGY



Allergen in the airway



Constriction

Inflammation

Asthma

VOLUME 1

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Lippincott-Raven

Asthma

VOLUME 2

EDITORS

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Lippincott-Raven

Overview

Ann J. Woolcock and Peter J. Barnes

What is Asthma

What Do We Know About Asthma?

What are the Controversial Issues?

What is the Role of Infections?

How Important are Genetic Factors?

How Should We Classify Asthma?

What is the Role of IgE?

Why Does Asthma Differ in Severity Between Patients?

How Should Asthma Best be Managed?

Why is the Disease So Poorly Understood?

How Could More Effective Progress Be Made?

Forming a Global Strategy

Defining the Important Questions

In this book we have attempted to bring together the current understanding of asthma: its causes, pathology, clinical features, treatment, and management. All the chapters are written by people who are experts in their field and each chapter is extensively referenced to provide source material for those interested in the various aspects of the disease.

Knowledge about asthma can be divided into a number of subjects as illustrated in Figure 1. Genetic and environmental factors are covered in the section on Epidemiology. Airway inflammation is covered by the sections on Structure, Biology, Pathology, Inflammatory Cells, Inflammatory Mediators, Target Cells, and Neural Mechanisms. Airway narrowing is covered by the sections on Inducers and

poor understanding of the causes, natural history, and airway behavior in this disease have led us to use the word "asthma" in a number of different ways—to describe symptoms, to describe exacerbations, and to describe the underlying abnormalities in the airways. This loose use of the word asthma causes confusion, and many patients and clinicians fail to appreciate that, frequently, it is a chronic, life-long disease. Asthma is a clinical syndrome that describes a set of symptoms. The cause(s) of asthma remains unknown (1).

Since we do not know what asthma is, we do not know if it is best thought of as a single disease of the airways which varies with severity or if it is better thought of as abnormal behavior of the airways in which they narrow

Asthma - Overview

- What is asthma?

“Despite the immense amount of material presented here (2200 pages) we do not know the answer to this question...”

- How should we classify asthma?

“The classification of asthma will remain a problem until more is known about the disease”

Asthma - Overview

- Why does asthma differ in severity between patients?

“We have little understanding of why the disease differs in severity between patients...”

- How should asthma be managed?

“The whole area of management remains controversial and has led to a mania in the production of management guidelines...”

Asthma at the 'coalface'

History
Wheeze

+

**reversible
airflow obstruction**

+

Response to Rx

Family history

Allergies

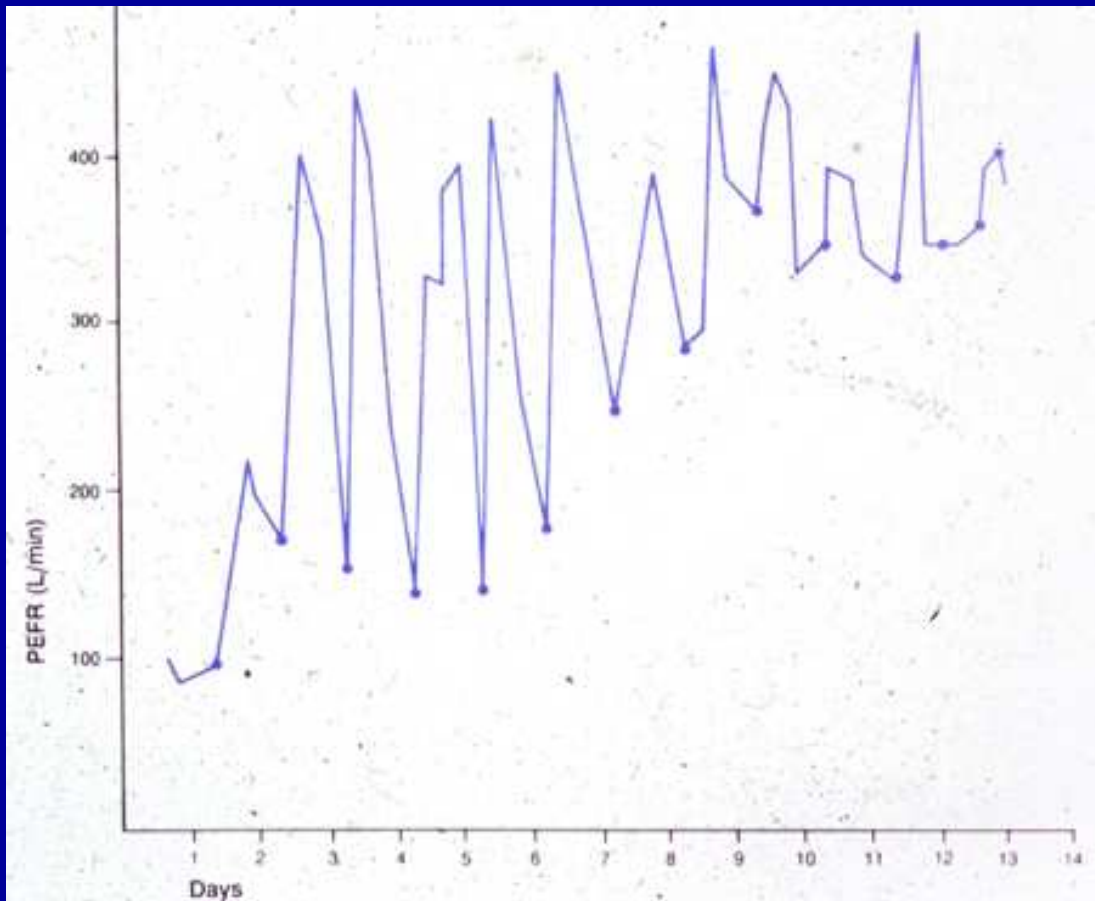
IgE

Hay fever

Eczema

TRIGGERS

OCCUPATION



- PEFR variability
- Response to Rx
reduced variability
increasing PEFR

PEFR = maximum “peak” airflow in litres/min



Wheezing
at rest



Coughing
at night



Wheezing
at night



Exercise
induced

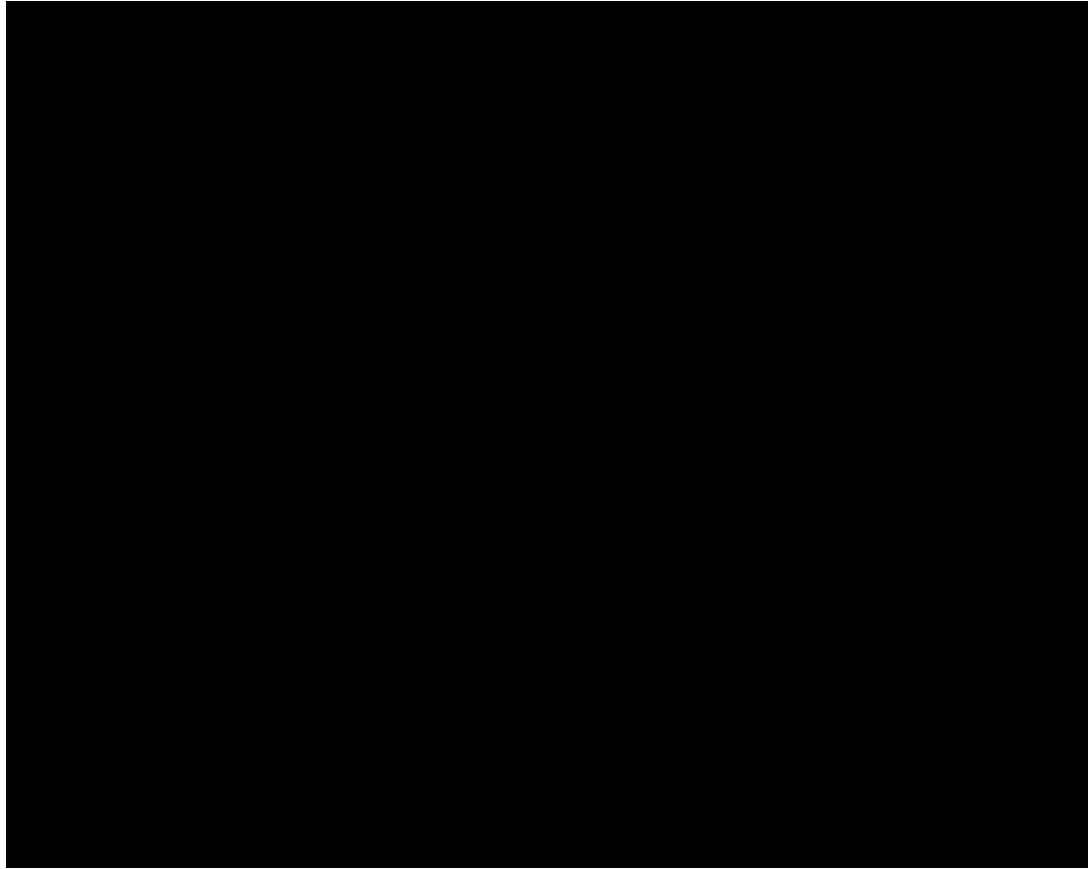


Severe
wheeze
(SOB)

No language on screen

Research staff read in local language(s):

- “Has your breathing been like this at any time”
- “last year”
- “last month”



Asthma – domestic causes and global trends

answer to asthma would be found in bedroom

- *Allergen exposure bedding*
- *+/- genes which are mixed in bedroom*

Last 20 years – associations more complex and varied

Lots more data BUT less understanding

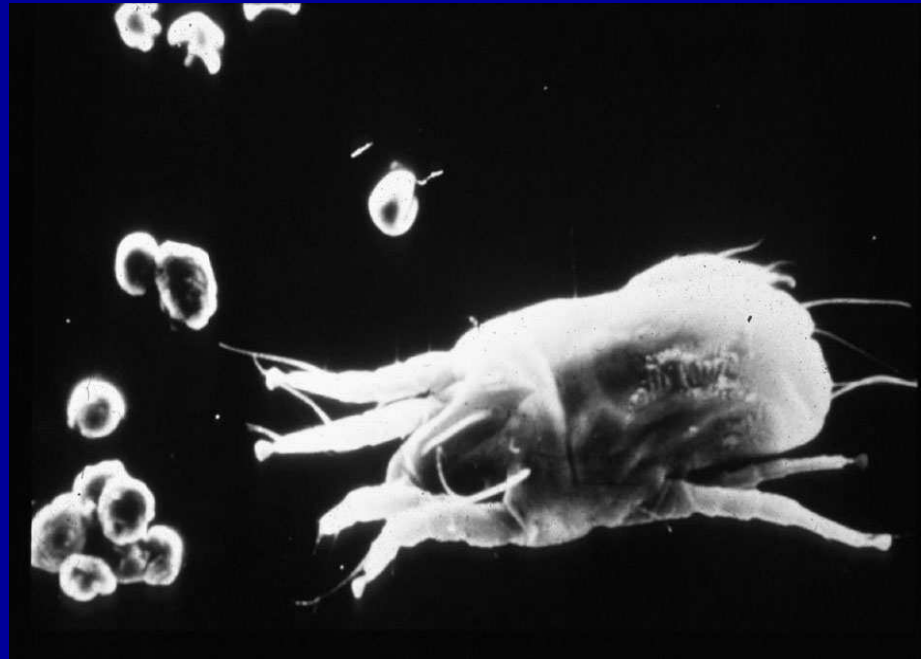
Asthma and domestic environment

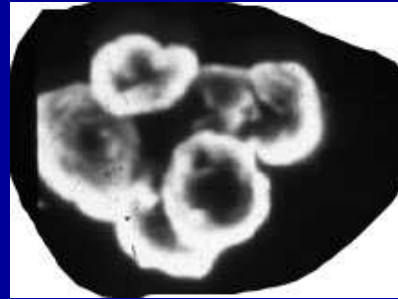
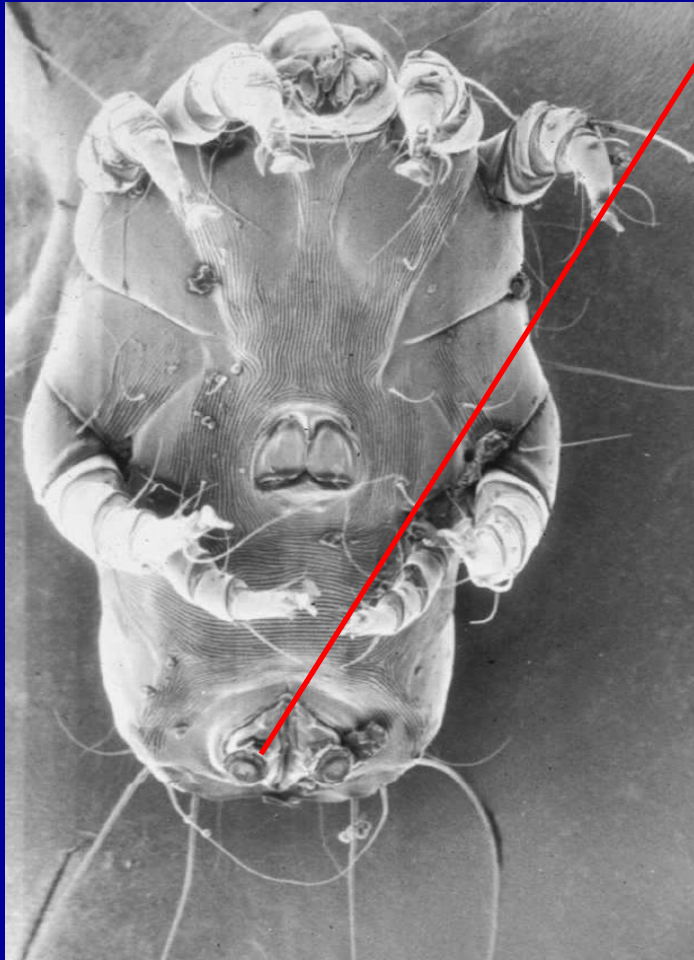
- Domestic 'causes' primary and secondary
 - allergy –
 - House Dust Mite allergen
 - Pet allergens
- Main reservoir
 - Bedding and Carpets
- Treatment
 - Remove pets, kill mites, denature allergen
 - Cover bedding, remove carpets
 - Ventilate houses – RH% < 50%

Too difficult implement, hard to show benefit

Non allergic asthma – endotoxin, moulds – beta glucans

Domestic Environmental Allergens



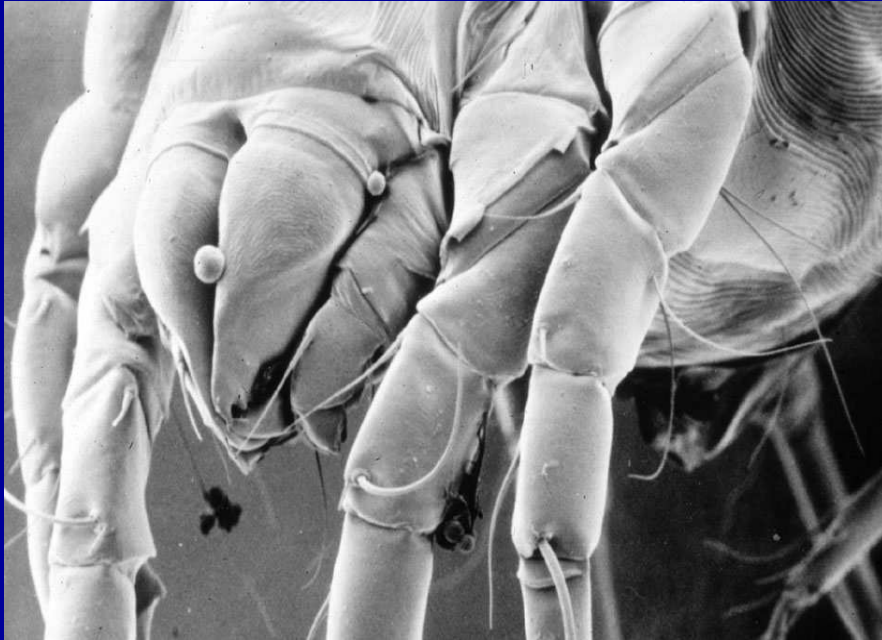


Measure the chemical
causing allergy in dust

>2mcg/g - sensitisation

>10mcg/g - asthma attacks

Dermatophagoides Pteronyssinus “feather loving”



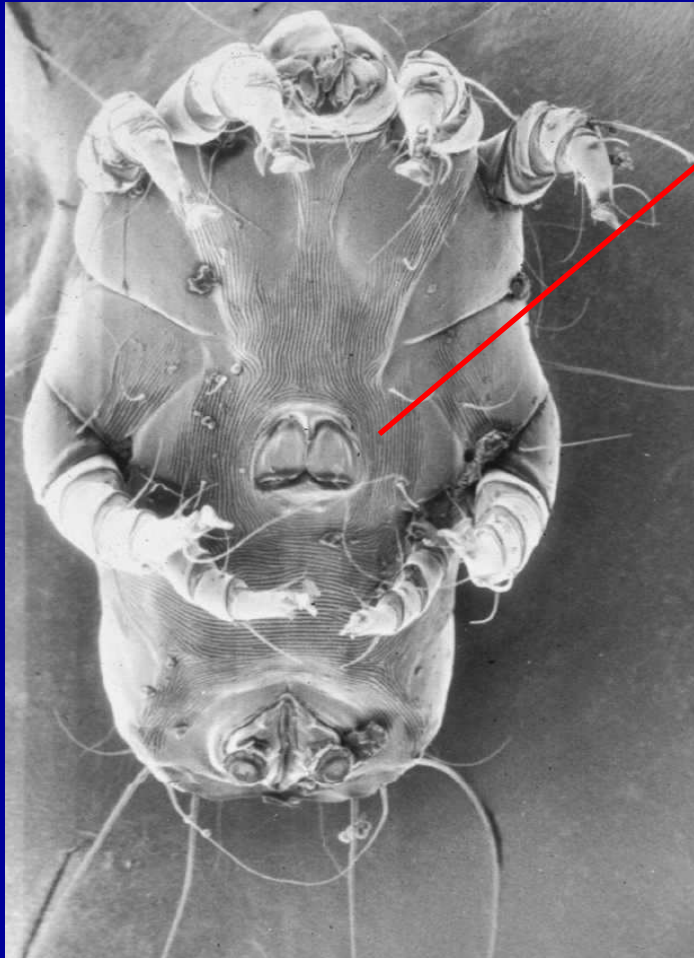
First seen 1850's
Rediscovered 1960s
Most species live with birds
2 species important and
live only with humans

blind

8 legs

close relative of spiders

feeds on skin scales and any organic material



‘valve’ to control the mite’s water supply from the surrounding air

Cannot survive in dry climates

Requires RH >50%

Desert

Mountains

- prevention strategy

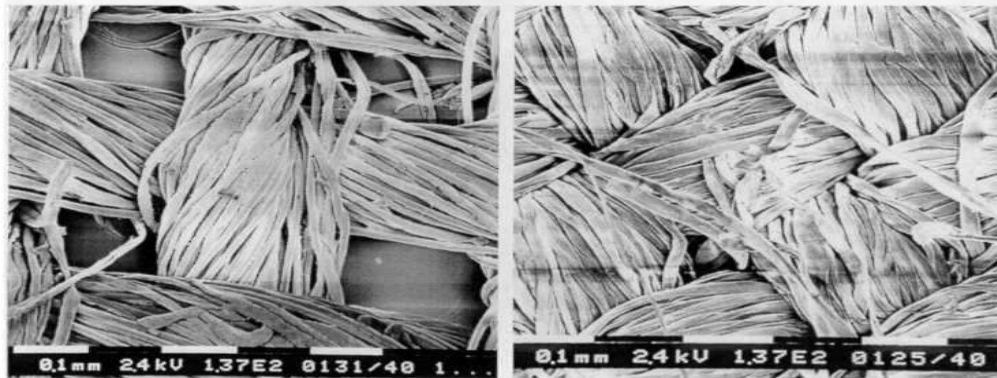




Managing allergies

- Bedding covers – pillows, duvets, mattresses
- Miticides, chemicals to denature allergen
- Humidity control
 - Problem – mites can survive 23/24 hrs 0RH%
- Low allergen pets, washing ??

Electron Microscopy of Synthetic (left) and
Feather (right) Pillow Encasement



Asthma and domestic environment

- Damp and cold
 - Moulds - allergic and non-allergic
 - Non specific effect of fungal wall material
 - ? Primary and secondary
 - Increased risk URTI - ?? Viral
 - Other?
- VOC's – carpets, synthetic bedding, flooring
- NO_x – unflued gas
- ETS – only modifiable asthma risk factor

Meta-analyses of health effects of dampness

Table 2 Key results of the meta-analyses

Outcome	Subjects	No. studies	Odds ratio central estimate (CI)	Estimated % increase in damp homes
Upper respiratory tract symptoms	All	13	1.70 (1.44–2.00)	52
Cough	All	18	1.67 (1.49–1.86)	50
	Adults	6	1.52 (1.18–1.96)	–
	Children	12	1.75 (1.56–1.96)	–
Wheeze	All	22	1.50 (1.38–1.64)	44
	Adults	5	1.39 (1.04–1.85)	–
	Children	17	1.53 (1.39–1.68)	–
Current asthma	All	10	1.56 (1.30–1.86)	50
Ever-diagnosed asthma	All	8	1.37 (1.23–1.53)	33
Asthma development	All	4	1.34 (0.86–2.10)	30

Fisk, W et al. Indoor Air 2007; 17: 284

Moulds and Asthma Severity

- Asthmatics sensitised to moulds have more severe asthma
 - Studies asthma clinic and specialist referrals
 - Hospital and ICU admissions
 - Life threatening asthma and deaths
 - A/E, hospital admissions - spore counts

Short communication

Sensitivity to fungal allergens is a risk factor for life-threatening asthma

Background: Previous studies have suggested that sensitivity to *Alternaria* and *Cladosporium* may be risk factors for life-threatening asthma. We have investigated this by studying the relationship between skin tests for fungal spores and admission to an intensive care unit (ICU) for asthma.

Methods: Skin prick tests for fungal spores (*Alternaria tenuis*, *Cladosporium cladosporoides*, *Helminthosporium maydis*, and *Epicoccum nigrum*), cat dander, house-dust mite (*Dermatophagoides pteronyssinus*), and a seven-grass mix were performed in three groups of patients: patients admitted to an ICU with an attack of asthma; those who had received emergency treatment for asthma but had not been admitted to an ICU, and those who had never required emergency treatment for their asthma.

Results: Twenty of 37 patients (54%) admitted to the ICU had a positive skin test for one or more fungal allergens compared with 15/50 patients (30%) in each of the other groups ($P=0.005$). The ICU patients were no more likely to have positive skin tests for the grass mix, cat dander, or house-dust mite than the other patients.

Conclusions: A positive skin test for fungal allergens is a risk factor for admission to an ICU with an acute attack of asthma.

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Key words: asthma; fungal allergens.

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Table 1. Baseline characteristics of subjects

	No. of subjects	Mean age (years) ³	Male:female
ICU	37	36.0 ± 9.8	13/24
Hospital ¹	50	32.5 ± 8.5	10/40
Community ²	50	35.9 ± 9.8	20/30

Table 2. Percentage of positive skin tests for different allergens

	Hospital	Community	ICU
<i>Alternaria</i>	26.0%	24.0%	40.5%
<i>Cladosporium</i>	14.0%	14.0%	24.3%
<i>Epicoccum</i>	20.0%	2.0%	21.6%
<i>Helminthosporium</i>	14.0%	8.0%	21.6%
All fungi	30.0%	30.0%	54.1%
Grass mix	76.0%	72.0%	59.5%
Cat dander	62.0%	62.0%	51.4%
<i>D. pteronyssinus</i>	90.0%	84.0%	89.2%

Sensitisation to airborne moulds and severity of asthma: cross sectional study from European Community respiratory health survey

Mahmoud Zureik, Catherine Neukirch, Bénédicte Leynaert, Renata Liard, Jean Bousquet, Françoise Neukirch, on behalf of the European Community Respiratory Health Survey

Abstract

Objective To assess whether the severity of asthma is associated with sensitisation to airborne moulds rather than to other seasonal or perennial allergens.

Design Multicentre epidemiological survey in 30 centres.

Setting European Community respiratory health survey.

Participants 11 32 adults aged 20-44 years with current asthma and with skin prick test results.

Main outcome measure Severity of asthma according to score based on forced expiratory volume in one second, number of asthma attacks, hospital admissions for breathing problems, and use of corticosteroids in past 12 months.

Results The frequency of sensitisation to moulds (*Alternaria alternata* or *Cladosporium herbarum*, or both) increased significantly with increasing asthma severity (odds ratio 2.34 (95% confidence interval 1.56 to 3.52) for either for severe v mild asthma). This association existed in all of the study areas (gathered into regions), although there were differences in the frequency of sensitisation. There was no association

ity but the identification of such factors is necessary for management and prevention.

Sensitisation to airborne allergens might be involved in the underlying mechanisms of severity. The associations between exposure, sensitisation, and asthma have suggested that house dust mite,^{1,2} animal dander,^{3,4} cockroaches,⁵ pollens,⁶ and mould spores⁷ have a causal role in development. However, the associations between sensitisation to different allergens and the severity of asthma have been poorly explored.

Sensitisation to moulds has been suggested as a risk factor for life threatening asthma. In a study of 11 patients with episodes of respiratory arrest, 10 had positive results on skin prick testing for *Alternaria alternata* compared with only 31 of the 99 matched controls with asthma and no history of respiratory arrest.⁸ It was recently reported that 20 of 37 (54%) patients admitted to an intensive care unit for asthma had a positive result on skin testing for one or more fungal allergens (*Alternaria tenuis*, *Cladosporium cladosporioides*, *Helminthosporium maydis*, or *Epicoecium nigrum*) compared with 30% in patients not admitted to intensive care units. The patients admitted to inten-

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Table 3 Associations between sensitisation to moulds and severity of asthma (% of sensitised participants by severity and odds ratios (95% confidence interval) for moderate versus mild asthma and severe versus mild asthma)

	Current asthma			P value for trend
	Mild (n=564)	Moderate (n=333)	Severe (n=235)	
Alternaria alternata				
% sensitised	8.9	13.8	16.6	<0.001
Unadjusted odds ratio	1	1.64 (1.08 to 2.52)	2.05 (1.31 to 3.21)	<0.001
Multivariate adjusted odds ratio*	1	1.61 (1.04 to 2.50)	2.03 (1.26 to 3.27)	<0.001
Cladosporium herbarum				
% sensitised	3.9	5.4	11.1	<0.001
Unadjusted odds ratio	1	1.41 (0.74 to 2.66)	3.07 (1.70 to 5.50)	<0.001
Multivariate adjusted odds ratio*	1	1.21 (0.62 to 2.36)	3.20 (1.72 to 5.94)	<0.001
Either mould				
% sensitised	10.8	15.9	22.1	<0.001
Unadjusted odds ratio	1	1.56 (1.05 to 2.32)	2.34 (1.56 to 3.52)	<0.001
Multivariate adjusted odds ratio*	1	1.48 (0.98 to 2.24)	2.34 (1.52 to 3.60)	<0.001

*Adjusted for age, sex, smoking, passive smoking, parental history of asthma, and region.

Severity classified by score
 Predicted FEV1
 asthma attacks last 12/12
 Hospital admissions last 12/12
 Oral or ICS last 12/12



Moisture damage and childhood asthma: a population-based incident case–control study

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T. Putus^{*} and A. Nevalainen^{*}

ABSTRACT: Most previous studies on the association between moisture damage and asthma have been cross-sectional and relied on self-reported exposure and health. The present authors studied the association by carrying out careful home inspections among new, clinically determined cases of asthma and controls.

New cases of asthma aged 12–84 months (n=121) were recruited prospectively and matched for year of birth, sex and living area with two randomly selected population controls (n=241). Trained engineers visited all homes. Both cases and controls had lived $\geq 75\%$ of their lifetime or the past 2 yrs in their current home.

Risk of asthma increased with severity of moisture damage and presence of visible mould in the main living quarters but not in other areas of the house. Cases more often had damage in their bedroom. Associations were comparable for atopic and nonatopic asthma and for children aged >30 months or ≤ 30 months.

The present results, using standardised assessment of exposure and asthma, suggest that moisture damage and mould growth in the main living quarters are associated with the development of asthma in early childhood.

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Can damp home 'cause' asthma??

- Case control study
 - New cases asthma age 12-84 months (121)
 - Age, gender, municipality matched controls (2 x 241)
 - Engineer assessment moisture damage
 - Atopic and non-atopic asthma

ASTHMA ASSOCIATED WITH

- Living areas
 - Visible mould and water damage
- Damage dose response effect
- Atopic asthma > non atopic

	Nonatopic cases versus their controls		Atopic cases versus their controls	
	n	OR (95% CI)	n	OR (95% CI)
Main living area				
Severity of damage				
No	77	1	137	1
Minor or major	43	2.11 (0.68–6.53)	49	2.84 (1.15–7.05)
Visible mould				
No	60	1	95	1
Yes	57	1.08 (0.32–3.64)	91	4.74 (0.94–24.01)
Damage in child's bedroom*				
No	91	1	24	1
Yes	26	1.89 (0.57–6.25)	161	2.10 (0.73–6.01)

Mould and asthma severity

- Outdoor mould exposure ✓
 - ? Thunderstorm and seasonal associations
- Evidence from NZ ✓
- Indoor exposure ✓ needs more work
 - Dampness water damage - development asthma ?
- Need for objective measures ✓
 - Building inspection vs self report
 - Evidence for reporting bias
 - **Reports + objective markers**

Effects of improved home heating on asthma in community dwelling children: randomised controlled trial

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ABSTRACT

Objective To assess whether non-polluting, more effective home heating (heat pump, wood pellet burner, flued gas) has a positive effect on the health of children with asthma.

Design Randomised controlled trial.

Setting Households in five communities in New Zealand.

Participants 409 children aged 6-12 years with doctor diagnosed asthma.

Interventions Installation of a non-polluting, more effective home heater before winter. The control group received a replacement heater at the end of the trial.

Main outcome measures The primary outcome was change in lung function (peak expiratory flow rate and forced expiratory volume in one second, FEV₁). Secondary outcomes were child reported respiratory tract symptoms and daily use of preventer and reliever drugs. At the end of winter 2005 (baseline) and winter 2006 (follow-up) parents reported their child's general health, use of health services, overall respiratory health, and housing conditions. Nitrogen dioxide levels were measured monthly for four months and temperatures in the living room and child's bedroom were recorded hourly.

Results Improvements in lung function were not significant (difference in mean FEV₁ 130.7 ml, 95% confidence interval -20.3 to 281.7). Compared with children in the control group, however, children in the intervention group had 1.80 fewer days off school (95% confidence interval 0.11 to 3.13), 0.40 fewer visits to a doctor for asthma (0.11 to 0.62), and 0.25 fewer visits to a pharmacist for asthma (0.09 to 0.32). Children in the intervention group also had fewer reports of poor health (adjusted odds ratio 0.48, 95% confidence interval 0.31 to 0.74), less sleep disturbed by wheezing (0.55, 0.35 to 0.85), less dry cough at night (0.52, 0.32 to 0.83), and reduced scores for lower respiratory tract symptoms (0.77,

C to 1.64°C) and in the child's bedroom of 0.57°C (0.05°C to 1.08°C). Lower levels of nitrogen dioxide were measured in the living rooms of the intervention households than in those of the control households (geometric mean 8.5 µg/m³ v 15.7 µg/m³, P<0.001). A similar effect was found in the children's bedrooms (7.3 µg/m³ v 10.9 µg/m³, P<0.001).

Conclusion Installing non-polluting, more effective heating in the homes of children with asthma did not significantly improve lung function but did significantly reduce symptoms of asthma, days off school, healthcare utilisation, and visits to a pharmacist.

Trial registration Clinical Trials NCT00489762.

INTRODUCTION

Asthma is one of the most prevalent chronic diseases in childhood. In New Zealand about 25% of children report symptoms of asthma, and asthma is the second most common reason for children being admitted to hospital.¹ As well as the stress associated with having a chronic disease, asthma can lead to higher utilisation of health services and drug costs.² Children with asthma are likely to have more days off school, with adverse effects on academic performance,³ and their caregivers may lose significant time from work.⁴

Evidence is growing that symptoms of asthma can be aggravated or triggered by adverse aspects of the indoor environment.^{5,6} Evidence from studies of excess morbidity and mortality during winter in temperate climates shows that temperatures in many homes are below the levels recommended by the World Health Organization for maintaining health in vulnerable populations.⁷ This is the case in New Zealand, where home heating seems not to be treated as a necessity like it is in the cooler parts of continental Europe.^{7,8}

Table 3 | Effect of heating intervention on parent reported health outcomes in children

Health outcome	No of children (n=349)	% with outcome in control group	Unadjusted		Adjusted*	
			Odds ratio (95% CI)	P value	Odds ratio (95% CI)	P value
Poor or fair health†	346	60	0.46 (0.30 to 0.71)	<0.001	0.48 (0.31 to 0.74)	<0.001
Attacks of wheezing‡	345	43	0.68 (0.44 to 1.05)	0.08	0.71 (0.45 to 1.11)	0.13
Sleep disturbed by wheeze	344	60	0.54 (0.35 to 0.83)	0.005	0.55 (0.35 to 0.85)	<0.001
Speech limited by wheeze	344	19	0.74 (0.43 to 1.27)	0.27	0.69 (0.40 to 1.18)	0.18
Wheeze during exercise	344	66	0.73 (0.46 to 1.14)	0.16	0.67 (0.42 to 1.06)	0.09
Dry cough at night	345	66	0.50 (0.32 to 0.79)	0.003	0.52 (0.32 to 0.83)	0.01
Diarrhoea	343	34	0.81 (0.51 to 1.26)	0.34	0.72 (0.45 to 1.16)	0.18
Vomiting	344	31	1.01 (0.64 to 1.59)	0.98	0.88 (0.55 to 1.40)	0.58
Ear infections	344	24	1.40 (0.85 to 2.31)	0.19	1.16 (0.68 to 1.99)	0.58
Twisted ankles	346	12	1.86 (1.03 to 3.35)	0.04	—§	—§

*Controlled for baseline measure.

†Compared with good, very good, and excellent health.

‡More than four attacks per week compared with fewer than three attacks.

§Question not asked in 2005.

Table 4 | Effect of heating intervention on daily differences of asthma symptoms and drug use as reported in daily diaries

Variable	No of person days	No of children	Unadjusted		Adjusted*	
			Mean ratio† (95% CI)	P value	Mean ratio† (95% CI)	P value
Lower respiratory tract symptoms	23 475	345	0.83 (0.66 to 1.05)	0.12	0.77 (0.73 to 0.81)	0.01
Cough at night	26 532	352	0.80 (0.63 to 1.00)	0.05	0.72 (0.59 to 0.89)	0.002
Wheeze at night	26 407	351	0.78 (0.54 to 1.12)	0.18	0.67 (0.49 to 0.93)	0.02
Cough on waking	26 514	352	0.74 (0.58 to 0.94)	0.02	0.67 (0.53 to 0.84)	<0.001
Wheeze on waking	26 417	351	0.68 (0.49 to 0.94)	0.02	0.60 (0.45 to 0.81)	0.001
Cough during day	27 348	365	0.90 (0.75 to 1.10)	0.31	0.84 (0.70 to 1.01)	0.06
Wheeze during day	27 117	363	0.85 (0.61 to 1.17)	0.32	0.78 (0.59 to 1.04)	0.09
Cough symptoms	23 713	349	0.82 (0.67 to 1.02)	0.08	0.75 (0.62 to 0.92)	0.005
Overall wheeze symptoms	23 532	345	0.76 (0.54 to 1.07)	0.11	0.67 (0.50 to 0.91)	0.01
No of reliever puffs	27 261	364	0.73 (0.46 to 1.14)	0.17	0.68 (0.44 to 1.05)	0.08
Reliever use at night (yes or no)‡	26 725	352	0.52 (0.24 to 1.13)	0.10	0.55 (0.28 to 1.08)	0.08
No of preventer puffs	27 567	363	1.05 (0.61 to 1.8)	0.87	1.08 (0.67 to 1.74)	0.74
Upper respiratory tract symptoms	26 844	360	0.95 (0.76 to 1.19)	0.65	0.92 (0.74 to 1.14)	0.43

*Adjusted for baseline outcome.

†Average score for intervention group divided by average score for control group.

‡Binary model used and results presented as odds ratio.

Asthma and homes

- Allergens – bedding, carpets – dust
- Damp and Moulds
 - ?? Cause asthma
 - Specific allergy – rare
 - Non specific effects
- Chemicals
- NO_x
- Open and closed fires?

Thank you

