

Occupational and environmental lung diseases: an update

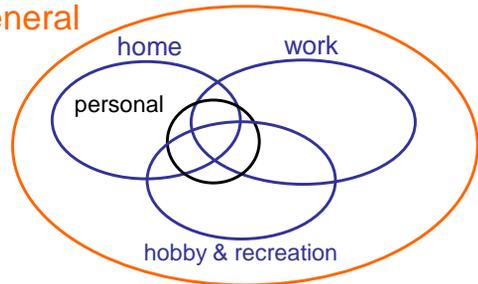
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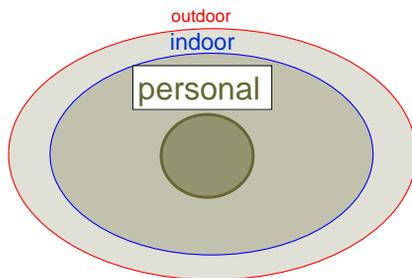


Environment

general



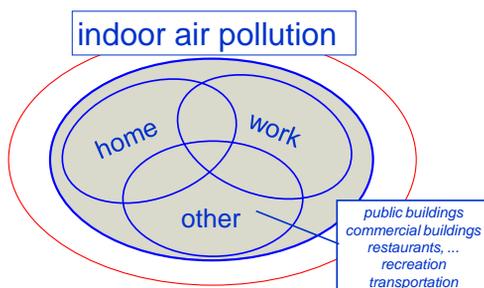
Air pollution



Air pollution



Air pollution



Air pollution



Effects of residential proximity to traffic in lung transplant patients

(Nawrot *et al.*, *Thorax* 2011, 66(9):748-54)

Downloaded from thorax.bmj.com on March 24, 2011. Published by group.bmj.com
 Thorax Online First, published on March 23, 2011 as 10.1136/thx.2010.155192

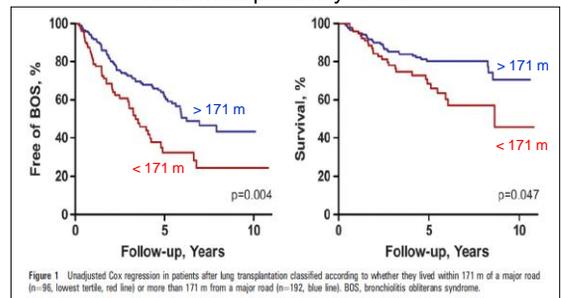
Lung transplantation

Press Release

The impact of traffic air pollution on bronchiolitis obliterans syndrome and mortality after lung transplantation

Tim S Nawrot,^{1,2} Robin Vos,^{3,4} Lotte Jacobs,⁷ Stijn E Verleden,^{3,4} Shana Wauters,⁴ Veerle Mertens,⁴ Christophe Dooms,³ Peter H Hoet,² Dirk E Van Raemdonck,^{4,5} Christel Faes,⁶ Lieven J Dupont,^{3,4} Benoit Nemony,² Geert M Verleden,^{3,4} Bart M Vanaudenaerde^{3,4}

BOS and mortality after lung transplantation and residential proximity to traffic



Occupation & respiratory disease

Some respiratory diseases are caused specifically by work exposures
 = occupational respiratory diseases

Occupational respiratory diseases

- Acute inhalation injuries
- Occupational infections
- Occupational asthma
- Chronic obstructive pulmonary disease
- Interstitial lung diseases
 - Pneumoconioses (silicosis, CWP, asbestosis, ...)
 - Berylliosis, hard-metal/cobalt lung disease, other metals, ...
 - Extrinsic allergic alveolitis
 - Other occupational ILD
- Bronchopulmonary cancer
- Pleural disease

“Emerging” lung diseases

1. Ardystil syndrome
2. Flock worker’s lung
3. Jeansblasting lung
4. Indium Tin Oxide
5. Nanomaterials

Sandblasting jeans

Eur Respir J 2008, 32, 1295-1303

An epidemic of silicosis among former denim sandblasters

M. Akgun*, O. Araz*, I. Akkurt[#], A. Eroglu¹, F. Alper*, L. Saglam*, A. Mirici¹, M. Gorguner* and B. Nemery¹



Akgun *et al.* ERJ 2008, 32, 1295-1303

- respiratory symptoms in 131 subjects (83%)
 - dyspnea 52%
 - chest pain 46%
- x-ray silicosis in 77 subjects (53%)
 - ILO Category 0 68 (47%) [0/- 52; 0/0 3; 0/1 13]
 - ILO Category 1 35 (24%) [1/0 19; 1/1 9; 1/2 7]
 - ILO Category 2 16 (11%) [2/1 4 ; 2/2 2; 2/3 10]
 - ILO Category 3 26 (18%) [3/2 8 ; 3/3 6; 3/+ 12]
 - Large opacities 14 (10%) [A 6; B 3; C 5]

Take home message

- “Sand” is one of the most toxic agents for the lungs
- Exposure to crystalline silica does not occur only in mining, tunnelling, foundries, ... but may occur in the “textile industry”
- Workers may die for futile reasons

Indium Tin Oxide (ITO)

Indium-Tin Oxide (ITO)

Homma S. *et al.* Pulmonary fibrosis in an individual occupationally exposed to inhaled indium-tin oxide. *ERJ* 2005, 25, 200-4

- Man, 30 y, light smoker (3 cig/d for 3 y)
- Exposure for 4 y to ITO (90% In₂O₃ / 10% SnO₂)
 - Manufacture of flat-panel displays (LCD, plasma screen)
- Dry cough and exertional dyspnoea; normal PFT
- Chest x-ray: reticulonodular shadows (right upper f)

Chonan T. *et al.* ERJ 2007, 29, 317-24

- ITO plant
 - 108 male workers (24 ex-workers)
 - mean age: 34 y [20-60 y]
 - mean duration of exposure: 3.6 y [0.8-17 y]
 - serum Indium: GM 8 ng.mL⁻¹ [0-127] ↑ with exposure duration; (control: GM 0.3 ng.mL⁻¹)
 - exposure to Indium: GM 0.01 – 0.05 mg.m⁻³ (max: 0.36); (particles Ø 2.5 µm [0.1-11 µm])
- HRCT: interstitial changes in 23 subjects
- Serum KL-6 > 500 U.mL⁻¹ in 40 subjects
Related to serum Indium
More disease in wet-surface grinding of ITO

Cummings *et al.* AJRCCM 2010, 181, 458-64

- Facility producing ITO (USA) (~ 15 workers)
- 2 cases of Pulmonary Alveolar Proteinosis
 - A. Male, nonsmoker, 49 y
 - September 2000 (after 9 month): dyspnea + dry cough
 - Diagnosis of PAP (HRCT, pathology)
 - October 2006: death in respiratory failure
 - B. Male, smoker, 39 y
 - 2005 (6 to 9 months after hire): dyspnea, dry cough, chest tightness
 - Diagnosis of PAP (HRCT, pathology)
 - 2009: partial improvement after bilateral whole lung lavage; autoAB against GM-CSF +

Take home message

- Indium Tin Oxide is a new cause of pulmonary alveolar proteinosis (in addition to SiO₂, ...)
- Hi-tech materials are not necessarily produced or applied with hi-tech safety and hygiene!

Engineered nanomaterials

Nanomaterials

Technical reason for developing and using nanomaterials:
large surface-to-mass ratio → better use of surface properties

→ change in biological properties, including toxicity

“Nanotoxicology”

Oberdörster G, Oberdörster E, Oberdörster J.
Nanotoxicology: An Emerging Discipline Evolving from Studies of Ultrafine Particles.
Environmental Health Perspectives, 2005, 113, 823-39

nano-size → Giga-numbers

Particle diameter (nm)	Particle no. (cm ⁻³)	Particle surface area (µm ² /cm ³)
5	153,000,000	12,000
20	2,400,000	3,016
250	1,200	240
5,000	0.15	12

Exposure

- Occupational environment
 - Metal fumes (welding, smelting, plasma spraying, ...)
 - Combustion processes (engine exhaust, carbon black, ...)
 - Aerosolized materials
 - Organic aerosols
 - Production (+handling & packaging) of nanomaterials
 - + cleaning/maintenance/repair
 - + leaks/accidents/waste
 - Secondary users of nanomaterials
- Ambient environment (urban, indoor)

Engineered nanomaterials

“A cause waiting for a disease”

Eur Respir J 2009; 34: 559-567
DOI: 10.1183/09041939.00170009
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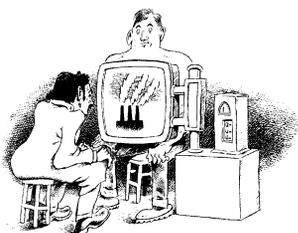
Exposure to nanoparticles is related to pleural effusion, pulmonary fibrosis and granuloma

Y. Song*, X. Li* and X. Du*

- Beijing
- 7 women (18-47 y) working in small print plant (5-13 months)
- Workplace 70 m², no windows, exhaust broken down, no PPE
- 1 machine to airspray coating material onto polystyrene boards
 - Coating material = “mixture of polyacrylic ester”: GC/MS “butanoic acid, butyl ester, N-butyl ether, acetic acid, toluene, di-tert-butylperoxide, 1-butanol, acetic acid ethenyl ester, isopropyl alcohol, ethylene dioxide”
- Electron microscopy of paste and accumulated dust: “nanoparticles ~ 30 nm”

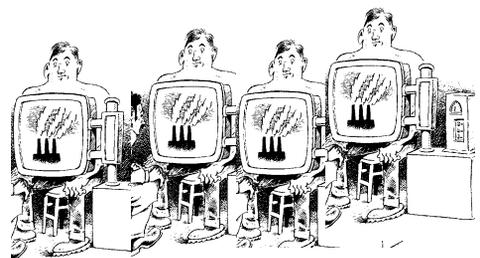
Engineered nanomaterials

“A cause waiting for a disease”



Contrary to this drawing, there is no simple test. The suspicion and the determination of work-relatedness depend primarily on a [careful occupational history](#)

From LEVY BS, WEGMAN DH. Occupational health (3rd ed), p.60



However, when you find one case of occupational disease, there are likely more around ...
[In occupational medicine, n is nearly always >1](#)

Modified From LEVY BS, WEGMAN DH. Occupational health (3rd ed), p.60

Nearly all respiratory diseases are influenced to some extent by the environment, especially the work environment

Pneumonia and occupation

- Coggon *et al.* Lobar pneumonia: an occupational disease in welders. *Lancet* 1994, 41-43
 - Excess mortality (SMR 182-157) from pneumonia in metal-working occupations (welders) (< 65 y)
- Coggon *et al.* Exposure to metal fume and infectious pneumonia. *Am J Epidemiol* 2003, 157, 227-33
 - 1996-1999, 11 hospitals in West-Midlands, UK
 - 525 cases of CAP / 1,122 controls
 - Occupational exposure in past 6 months?
 - Exposure to metal fumes: adjusted O.R. **1.6**
 - Recent exposure to Fe: adj O.R. **2.3** (lobar pneumonia)

COPD and occupation

- American Thoracic Society Statement: Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med* 2003, 167, 787-797
- Literature-based estimation of **population attributable risk (PAR)** for asthma and COPD due to occupational exposures
 - Asthma: median PAR **15%** (21 studies)
 - COPD: PAR **~15%**
 - Chronic bronchitis (8 studies): median 15% [4-24%]
 - Airflow obstruction (5 studies): median 18% [12-55%]

COPD and occupation

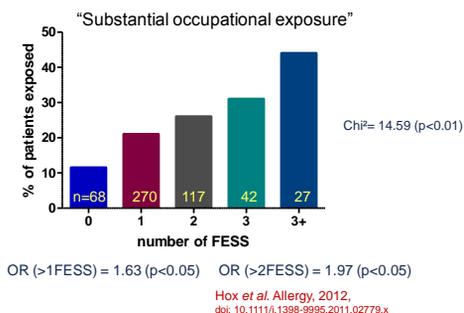
- Blanc *et al.* Occupational exposures and the risk of COPD: dusty trades revisited. *Thorax* 2009, 64, 6-12
- subjects (40-65 y) from Kaiser Permanente
 - 1202 with COPD (742 with GOLD 2-4)
 - 302 matched control subjects
- Telephone interview, then Spirometry
- Self-reported exposure to vapours, gas, dust or fumes (VGDF) + Job-Exposure Matrix (JEM) [longest held job]
 - COPD subjects: lower educational attainment, less \$, more smokers (13% NS), more « dirty » jobs

COPD and occupation

- Blanc *et al.* Occupational exposures and the risk of COPD: dusty trades revisited. *Thorax* 2009, 64, 6-12
- VGDF exposure: adj. OR **2.11** [1.59-2.82] → PAF **31%**
- Joint influence of smoking and VGDF:

	adj. OR COPD	GOLD 2+
• Never S / no VGDF	1.0	1.0
• Never S / VGDF	1.98 [1.26-3.09]	1.69
• Ever S / no VGDF	6.71 [4.58-9.82]	8.31
• Ever S / VGDF	14.1 [9.33-21.2]	18.7

Chronic rhinosinusitis and occupation



Asthma severity and occupation

- Le Moual N. *et al.* Asthma severity and exposure to occupational asthmogens. *Am J Respir Crit Care Med* 2005, 172, 440-5
 - Retrospective study of tertiary referral centres (France): 148 asthmatics
 - 8 grade score of severity (frequency of attacks, persistence of symptoms, hospitalizations)
 - Asthma more likely to be “severe” if exposure to known asthmogens (HMW & LMW sensitizers; irritants)

How much asthma is work-related?

[Balmes J. (chair) *et al.*] American Thoracic Society Statement. Occupational contribution to the burden of airway disease. *Am J Respir Crit Care Med* 2003, 167, 787-97

Literature-based estimation of **population attributable risk (PAR)** for asthma «due» to occupational exposures: median **15%** (21 studies: 4% to 58%)

How much asthma is work-related?

<<1%

- workers' compensation agencies
- physician-based voluntary reporting schemes
- prevalence and incidence studies in the population

25%

Mannino DM. *Occup Med* 2000, 15, 359-68

Under-recognition of occupational asthma

- Insufficient awareness among clinicians
 - Own experience (undocumented)
 - Shofer *et al.* *Chest* 2006, 130, 455-62
 - Academic medical center (USA)
 - Clinical notes of 197 adults with newly diagnosed asthma
 - Job title in 75%; rarely other details (exposures, duties, prior job, ...)

Under-recognition of occupational asthma

- Diagnosing occupational asthma is not (always) easy
 - Many pitfalls (Nemery. *Breathe* 2004, 1, 25-32)
 - Clinicians are unfamiliar with workplace exposures
- Diagnosing occupational asthma often leads to (more) administrative work

Asthma risk by occupation

- Karjalainen *et al.* *AJRCCM* 2001, 164, 565-8
SJWEH 2002, 28, 49-57

Finland

- reimbursement of medication for asthma only if persistent asthma is confirmed by a chest physician (“Reimbursement Register”)
- match with individual employment data (Social Security Register)

Asthma risk by occupation

- Karjalainen *et al.*
 - 3 cohorts of all employed Finns (25 - 59 y) without pre-existing asthma in 1985, 1990, 1995
 - followed for **incident** asthma for 4 years
 - **49,575** incident cases of adult asthma in Finland
 - 1.65 (M) - 2.47 (F) / 1,000 / year
 - 2,464 cases of recognized occupational asthma

Asthma risk by occupation

- Karjalainen *et al.*
 - **attributable fraction** of occupation for adult-onset asthma (controls = administrative workers):
 - **29 %** (men) - **17 %** (women)
 - not confounded by smoking
 - known sectors (agriculture, manufacture, services) and occupations (bakers, ...), but also less known jobs (cleaners, ...)
 - share of recognised cases of OA << 50 %

How much asthma is work-related?

Kogevinas M. *et al.* Exposure to substances in the workplace and new-onset asthma: an international prospective population-based study (ECRHS-II). *Lancet* 2007, 370, 336-341

- ECRHS-I (1990-95), 28 centres, 13 countries, 20-44 y
 - ECRHS-II (1998-2003): follow-up of 6,837 subjects without asthma or respiratory symptoms
 - New-onset asthma (symptoms or medication): n=134
 - Occupational exposures (high-risk job; job-exposure matrix; inhalation accidents)
- PAR due to occupation: **10-25%** (250-300 cases/10⁶/y)

Occupational asthma Definitions

- **Occupational asthma**
Asthma that is **caused** (specifically) by exposure to an agent present at work
- **Work-aggravated asthma**
Pre-existing asthma that is **aggravated** (non-specifically) by work (cold, exercise, irritants)

Occupational asthma Definitions

- **Occupational asthma**
 - **Work-aggravated asthma**
- ! in practice the difference may be difficult to make
- ! pre-existing asthma does not exclude the occurrence of occupational asthma
- ! work-aggravated asthma also needs appropriate individual + collective measures

Work-aggravated asthma

- Exposures at work are frequently evoked as causes of exacerbations
 - Henneberger *et al.* *OEM* 2006, 63, 551-7
 - 598 adult asthmatics (HMO), telephone questionnaire
 - workplace exacerbation in **23%**
 - Berger *et al.* *JOEM* 2006, 48, 833-9
 - 301 working asthmatics (low-income minority, NY)
 - workplace exacerbation of respiratory symptoms reported by **51%** (current job) - **71%** (ever)

Work-aggravated asthma

Henneberger *et al.* The occupational contribution to severe exacerbation of asthma. *ERJ* 2010, 36, 743-50

- ECRHS-I & II
- 966 working adults with current asthma
- 74 (7.7%) at least 1 self-reported severe exacerbation in past year
- If high exposure to dust, gas or fumes: RR **3.1** → PAR **14.7%** among workers with asthma

American Thoracic Society Documents

An Official American Thoracic Society Statement: Work-Exacerbated Asthma

Paul K. Henneberger, Carrie A. Redlich, David B. Callahan, Philip Harber, Catherine Lemière, James Martin, Susan M. Tarlo, Olivier Vandenas, and Kjell Torén, on behalf of the ATS Ad Hoc Committee on Work-Exacerbated Asthma

THIS OFFICIAL AMERICAN THORACIC SOCIETY (ATS) STATEMENT WAS APPROVED BY THE ATS BOARD OF DIRECTORS, MARCH 2011

Am J Respir Crit Care Med Vol 184, pp 368-378, 2011

- Work-exacerbated asthma (WEA)
- Median prevalence: **21.5%** among adults with asthma
- "WEA should be considered in any patient with asthma that is getting worse or who has work-related symptoms"
- "Management of WEA should focus on reducing work exposures and optimizing standard medical management, with a change in job only if these measures are not successful"

Occupational asthma Types

1. Occupational asthma caused by **immunological** sensitisation
2. Occupational asthma not caused by immunological sensitisation

Occupational asthma Types

1. Occupational asthma caused by **immunological** sensitisation (occupational asthma "*stricto sensu*")
 - symptom-free latency period "**occupational asthma with latency**"*
 - reaction to (extremely) low amounts
 - "minority" of exposed workers

* Bernstein IL, Chan-Yeung M, Malo JL, Bernstein DI. (Eds) *Asthma in the workplace* (2nd Ed.) Marcel Dekker, 1999

Occupational allergic asthma Causes

- High molecular weight (HMW) agents (macromolecules of biologic origin)
 - **IgE mechanisms**
- Low molecular weight (LMW) agents ("chemicals" < 1500 Dalton)
 - **not (necessarily) via IgE mechanisms**

Occupational asthma Types

2. Occupational asthma "without" immunological sensitisation
 - caused by irritants ("irritant-induced")
 - single exposure (**RADS**)
 - multiple peaks
 - caused by organic dust and microbial contaminants (**asthma-like syndrome**)

Occupational asthma Types

2. Occupational asthma caused by irritation: “irritant-induced asthma”
 - 2.1. after a single inhalation accident
= RADS (“Reactive Airways Dysfunction Syndrome”)*

* Brooks S, Weiss MA, Bernstein IL. *Chest*, 1985, 88, 376-84

RADS

Brooks SM, Weiss MA, Bernstein IL. Reactive airways dysfunction syndrome (RADS): persistent asthma syndrome after high level irritant exposure. *Chest*, 1985, 8, 376-84

= *de novo* asthma caused by an acute inhalation injury

RADS - criteria

1. Documented absence of preceding respiratory complaints
2. Onset of symptoms after a single specific exposure incident
3. Exposure to gas, smoke, fume or vapour present in very high concentration and with irritant properties
4. Onset of symptoms within 24 h after exposure
5. Persistence of symptoms for at least 3 months
6. Symptoms simulate asthma (cough, wheezing, dyspnoea)
7. Pulmonary function tests may show airflow obstruction
8. Positive methacholine/histamine test
9. Other disease ruled out

RADS

- Inhalation accidents and RADS may occur
 - at work: industry, agriculture, services
 - specific risk (petrochemical industry, firefighters, maintenance workers, industrial cleaning, ...)
 - nonspecific risk
 - at home:
 - cleaning (e.g. bleach + acids or NH₃), DIY, ...
 - in the community:
 - transportation accidents
 - fires & explosions

RADS - criteria (2')

2. Onset of symptoms after a single specific exposure incident
 - yes, in typical cases
 - also after repeated high-level respiratory irritant exposures
- Tarlo SM, Broder I. *Chest*, 1989, 96, 297-300

RADS - criteria (3')

3. Exposure to gas, smoke, fume or vapour present in very high concentration and with irritant properties
 - yes, in typical cases
 - inhalation injury requiring medical treatment (emergency room admission, infirmary, ...)
 - some cases do not appear to involve “very high” concentrations, nor clinically severe injury needing (immediate) medical attention

RADS and severity of initial injury

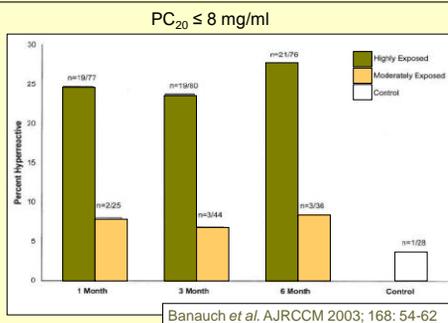
- Cohort studies do *not* indicate that RADS only occurs after a clinically severe inhalation injury *
 - Kern. *ARRD* 1991, 144, 1058-64
 - spill of **glacial acetic acid** in hospital
 - Cone *et al.* *Chest* 1994, 106, 500-8
 - derailment → metam sodium in river → **MITC** (CH₃NCS)
 - Banauch *et al.* *AJRCCM* 2003, 168, 54-62
 - NYFD after 9/11 WTC collapse

* Nemery B. (Editorial). *AJRCCM* 2003, 168, 2-3

“WTC 9/11”

- Prezant *et al.* *NEJM*, 2002, 347, 806-15
 - 11,336 FDNY firefighters
 - 343 died - 10,116/10,993 evaluated
 - 1636 (16%) high exposure (present at WTC collapse)
 - 6958 (69%) moderate exposure (within first 2 days)
 - 1320 (13%) low exposure (3-7 days after collapse)

Bronchial hyperreactivity



RADS at WTC

- RADS = « bronchial hyperreactivity with respiratory symptoms at 6 months »
 - 17/83 (20%) of highly exposed
 - 3/40 (8%) of moderately exposed
 - all nonsmokers, except one
 - **no evidence of clinically severe initial injury**
 - occurrence of RADS predicted only by hyperreactivity at 1 or 3 months

RADS - Prognosis

Malo *et al.* Long-term outcomes of acute irritant-induced asthma. *AJRCCM* 2009, 179, 923-8
 35 subjects with RADS, 13.6 y [4 – 24 y] after accident

- All had respiratory symptoms (34% inhaled steroids)
- No improvement in spirometry
- Methacholine test (n=23): normal or improved in 6+3 (better starting values)
- Induced sputum (n=27):
 - eosinophils >2% (n=6), pmn >60% (n=8);
 - ↑ mediators of inflammation & remodelling (~ occupational asthma)
- Abnormal depression score: n=12

Occupational asthma Types

- Occupational asthma caused by irritation: “**irritant-induced asthma**”
 - after **multiple peaks** of chemical irritants
 - Cl₂, SO₂, formaldehyde, ...

Asthma and irritants?

Asthma and cleaning agents

- Higher risk of asthma in female cleaners
 - Zock *et al. SJWEH* 2001; 27: 76-81: **P.R. 1.7**
 - Karjalainen *et al. ERJ* 2002; 19: 90-5: **R.R. 1.50**
 - Medina-Ramón *et al. Thorax* 2003; 58: 950-4: **O.R. 1.46**
- “hidden sensitizers”?
 - Quaternary ammonium cpds (disinfectants / preservatives)
 - Isothiazolinones (preservatives)
 - Ethanol amines (wax-removal agents)
 - d-Limonene, terpenes (perfumes)
- exposure to irritants and sprays ?

Asthma and cleaning agents

Medina-Ramón *et al. OEM* 2005, 62, 598-606

- (Nested) case-control study of female cleaners (30-65 y)
- 40 cases (asthma or chronic bronchitis) – 155 controls
- Higher risk of asthma if use of bleach (dose-related)
- Higher risk of asthma if reported inhalation incident (frequent!)

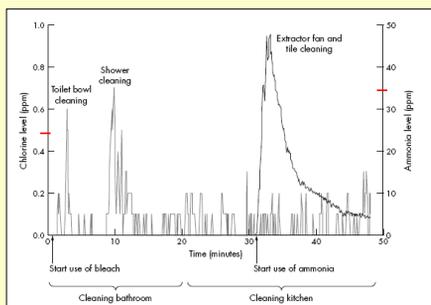
Medina-Ramón *et al. OEM* 2005, 62, 598-606

Table 4 Multivariate associations (adjusted odds ratios and 95% confidence intervals) between asthma/chronic bronchitis symptoms, and risk factors

	Controls (n=152)	All cases (n=40)	Asthma (n=24)	Chronic bronchitis without asthma (n=16)
		OR (95% CI)	OR (95% CI)	OR (95% CI)
Bleach (both undiluted and diluted)				
<344 times/year	56	8	1.0	5
344-640 times/year	53	11	2.3 (0.9 to 11)	2
>640 times/year	42	21	4.9 (1.5 to 15)	9
Use of liquid multi-use cleaning products				
<344 times/year	50	20	1.0	7
244-480 times/year	51	12	0.3 (0.1 to 0.8)	6
>480 times/year	51	8	0.2 (0.1 to 0.6)	3
Washing dishes				
<276 times/year	64	10	1.0	2
276-520 times/year	37	12	3.2 (1.0 to 10)	6
>520 times/year	51	18	3.1 (1.1 to 8.9)	10
Inhalation of an important quantity of vapours, gas, or fumes related to cleaning agents				
Never	73	9	1.0	4
Ever	79	31	2.3 (0.9 to 6.1)	12
Employment in non-domestic cleaning				
Never	101	9	1.0	4
Ever	51	31	8.5 (3.2 to 23)	12
Smoking				
Never	127	25	1.0	6
Currently	15	11	4.1 (1.1 to 15)	9
Formerly	10	4	5.3 (1.1 to 23)	1

Multiple logistic regression analyses adjusted for all listed variables and age, tenure.
*Three controls had missing values for one or more of the exposure variables and were not included in this multivariate model.

Medina-Ramón *et al. OEM* 2005, 62, 598-606



Asthma and cleaning

Review: Jaakkola JJK, Jaakkola MS. Professional cleaning and asthma. *Curr Opin Allergy Clin Immunol* 2006, 6, 85-90

- Systematic Medline literature search 2003-2005 (12 relevant publications)
- Occupational exposures
- Case reports and series
- Occupational disease registry reports & registry-linkage studies
- Epidemiologic studies (n=6)

Asthma and cleaning agents

Zock *et al.* *AJRCCM* 2007, 176, 735-741

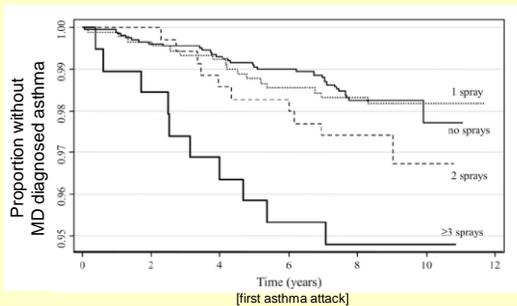
- ECRHS-I → ECRHS-II [+ 9 y]
- N = 3,503 (69% women), 20-48 y [→ 28-57 y]
 - ECRHS-I: free of asthma at baseline
 - ECRHS-II: “doing cleaning at home”
 - face-to-face interview
 - use of 15 products for domestic cleaning and washing?
 - never, <1 d/w, 1-3 d/w, 4-7d/w

Asthma and cleaning agents

Zock *et al.* *AJRCCM* 2007, 176, 735-741

- use of cleaning sprays ≥ 1 d/w: RR **1.49** for incidence of asthma symptoms/medication
- use of cleaning sprays ≥ 4 d/w: RR **2.11** for incidence of physician-diagnosed asthma
- sprays for glass-cleaning, furniture and air-refreshing
- no association with cleaning products not applied as sprays
- no modification of risk by atopy

Zock *et al.* *AJRCCM* 2007, 176, 735-741



How much asthma is work-related?

“In adults, asthma is caused (directly or indirectly) by work in approximately **15 %** of cases”

- Work-aggravated asthma? **some?**
- Occupational allergic asthma? **minority**
- Irritant-induced asthma? **many?**

Thank you for your
attention

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