

ROBERT HARRISON, MD MPH CALIFORNIA DEPARTMENT OF PUBLIC HEALTH OCCUPATIONAL HEALTH BRANCH

UNIVERSITY OF CALIFORNIA, SAN FRANCISCO DIVISION OF OCCUPATIONAL AND ENVIRONMENTAL MEDICINE



Learning Objectives:

- Understand the link between asthma and the work environment
- Identify strategies for recognizing and managing work-related asthma
- Familiarize yourself with the clinical resources related to work-related asthma

Produced under grant number DOL OSHA SH-27640-SH5 from the Occupational Safety and Health Administration, U.S. Department of Labor. It does not necessarily reflect the views or policies of the U.S. Department of Labor, nor does mention of trade names, commercial products, or organizations imply endorsement by the U.S. Government





Personnel

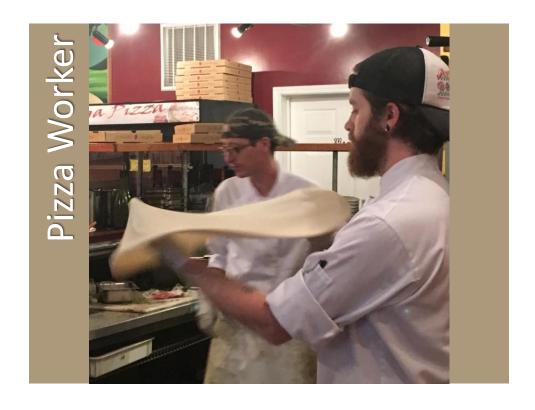
- Carolina Espineli (research assistant)
- Jennifer Flattery (epidemiologist)
- Eleana Martysh (research assistant)
- Debbie Shrem (health educator)
- Justine Weinberg (industrial hygienist)

Personnel

- Margaret Filios
- Patricia Schleif



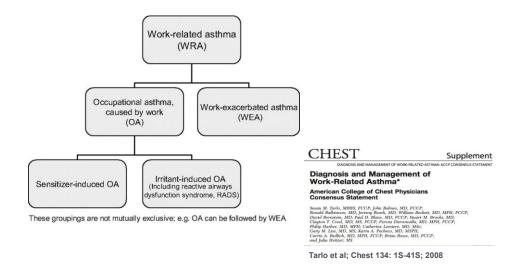




Clinical definition of WRA

- Variable airflow limitation and/or airway hyperresponsiveness due to exposure to a specific agent or conditions in the work environment
- Sensitizer and irritant-induced asthma

Clinical classification



Surveillance case definition

- Health care professional diagnosis consistent with asthma, AND
- An association between symptoms of asthma and work
- Includes both new onset (OA, RADS) and work-aggravated asthma

New onset asthma (I)

Occupational asthma

 Workplace exposure to an agent previously associated with occupational asthma?
 "Yes" or "No"

AND

• Objective evidence of work-relatedness?: "Yes" or "No"

New onset asthma (II)

- Reactive airways dysfunction syndrome (RADS)
 - New asthma symptoms within 24 hours after one-time high-level exposure, persists
 > 3 months

Work-aggravated asthma

Preexisting asthma that was symptomatic and/or treated with asthma medication within 2 years prior to entering the occupational setting

Epidemiology

- > 250 agents reported to cause OA (Chan-Yeung 1994)
- Most common type of occupational lung disease in population based studies (McDonald 2000)



Epidemiology

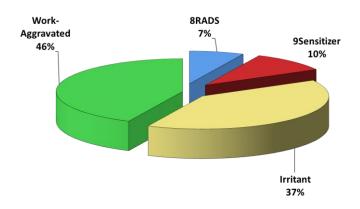
Synthesis of previously and currently reviewed studies regarding population attributable fraction (PAR) for occupational exposures and asthma.

ccupational exposures and asthma.					
Type of study	Studies Included	Ref	Range	Mean	Media
Current review					
Longitudinal	6	19–24	8.6%- 44%	19.3%	16.3%
Case-control	3	26-29	9.5%- 21.4%	14.8%	13.5%
Cross-sectional	7	31–37	7.0%- 31.3%	16.1%	13.6%
Current and earlier review					
Longitudinal	6	19-24	8.6%- 44.0%	19.3%	16.3%
Case-control	6	26-29, 41-43	9.5%- 36.0%	20.7%	12.2%
Cross-sectional	14	31-37, 44-51	7%-51%	21.2%	17.6%
All	26	See above	7%-51%	20.7%	17.6%
All, adult-onset	17	19-24, 26, 28-31, 34-36, 42, 43, 45	8.6%- 44.0%	18.8.5%	16.9%

- Median PAR = 17.6% (Toren and Blanc, 2009)
- Occupations at high risk: painters, bakers, woodworkers, welders, chemical workers
- Most common exposures: isocyanates, flour/grain, wood, latex, glutaraldehyde, lab animals

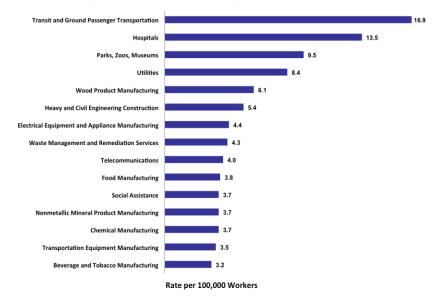
Classification of Confirmed Cases

Work-related asthma in California, 1993-2012 (N=2,991)



Top Industries with Highest Rates of Work-related Asthma

California Work-related Asthma Prevention Program (WRAPP) Surveillance Data, 1993-2011 (N=6,577) Occupational Health Branch, California Department of Public Health

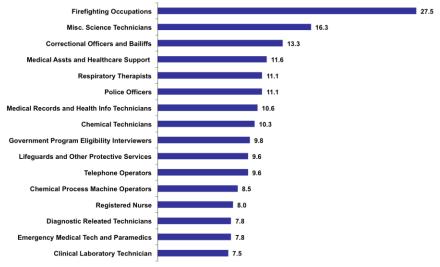




Select occupation with highest rate of WRA.

Top Occupations with Highest Rates of Work-related Asthma

California Work-related Asthma Prevention Program (WRAPP) Surveillance Data, 1993-2011 (N=6,577)
Occupational Health Branch, California Department of Public Health



Rate per 100,000 Workers

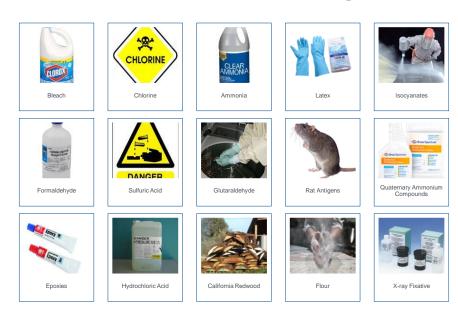
Exposures among Occupations with the Highest Rates

Occupation	Most Common Exposures		
Firefighters	Smoke		
Science Technicians	Acids, chemicals, indoor air, rat antigens, glues, dust		
Medical Assistants & Support	Glutaraldehyde, chemicals, smoke, latex, dust, perfume, paint		
Correctional Officers & Bailiffs	Smoke, chemicals, pepper spray, mace, cleaning chemicals		
Respiratory Therapists	Cleaning chemicals, latex, pharmaceuticals		
Medical Records Technicians	Dust, smoke, perfume		
Police Officers	Smoke, pepper spray, dust, indoor air, mold, animal antigens		
Telephone Operators	Chemicals, perfume, paint, carpet dust		
Chemical Technicians	Solvents, acids, chemicals		
Govt Program Eligibility Workers	Roofing tar, chemicals, indoor air, toner, perfume, dust		

15 Most Common Exposures Reported by Cases

Dust
Unspecified Chemicals
Smoke
Mold
Indoor Air Pollutants
Cleaning Agents
Paint
Indoor Air Pollutants from Building Renovation
Perfume
Pesticides
Glues
Bleach
Diesel Exhaust
Asphalt
Cigarette Smoke

Most Common Asthmagens



Pathogenesis: sensitizer-induced

High molecular weight (≥ 5,000 Da)

- IgE-mediated (ex: flour dust, latex)
- Bind to specific IgE on mast cells, basophils
- Act as complete antigens

Pathogenesis: sensitizer-induced

High molecular weight [continued]

- Release of cytokines/chemokines
- Activation of inflammatory mediators (histamine, leukotrienes, prostaglandins)
- Antibodies detected by circulating IgE (RAST) or skin prick testing

Pathogenesis: sensitizer-induced

Low molecular weight

- IgE-mediated (Ex: acid anhydrides, platinum) or non IgE-mediated (ex: isocyanates)
- React with proteins to produce complete antigen, mechanism poorly characterized
- Key role for T lymphocytes in inflammatory process

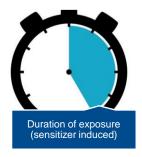
Pathogenesis: sensitizer-induced

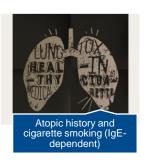
- Effector cells (eosinophils, mast cells, epithelial cells, neutrophils) cause smooth muscle contraction, mucus hypersecretion, airway inflammation, and epithelial injury
- Genetic polymorphisms for major histocompatibility complex class II proteins may determine specificity of response

Pathogenesis: irritant-induced

- Localized inflammatory response with subepithelial fibrosis, eosinophils and T cells infiltration
- Activation of nonadrenergic, noncholinergic pathways via axon reflexes and mast cell degranulation
- Recruitment of inflammatory cells
- Altered epithelial permeability

Risk factors for WRA



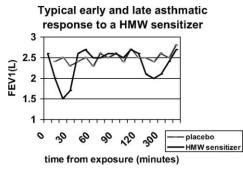




Diagnosis of WRA

- Asthma = intermittent respiratory symptoms and reversible/variable airways obstruction
- Cough, chest tightness, shortness of breath, dyspnea on exertion
- Rhinoconjunctivitis (more common with HMW substances)

Diagnosis: sensitizer-induced



Tarlo et al; Chest 134: 1S-41S; 2008

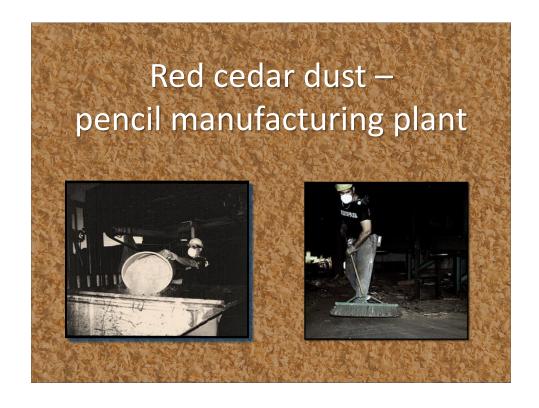
Symptoms occur months to years after exposure onset

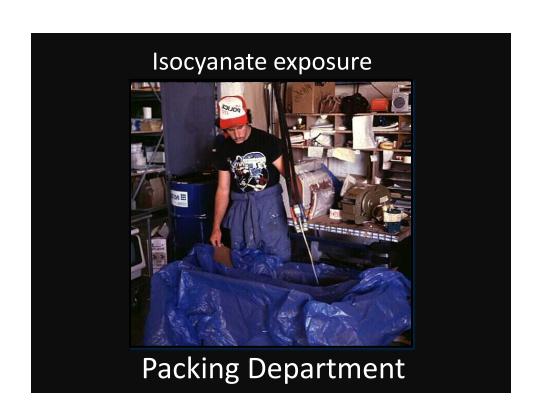
- Early, late or biphasic responses
 - HMW: early and biphasic
 - LMW: late and biphasic



Which of the following tests is LEAST useful for the diagnosis of work-related asthma?









Egg protein exposure - breaking room



Cleaning agent exposure - hospital







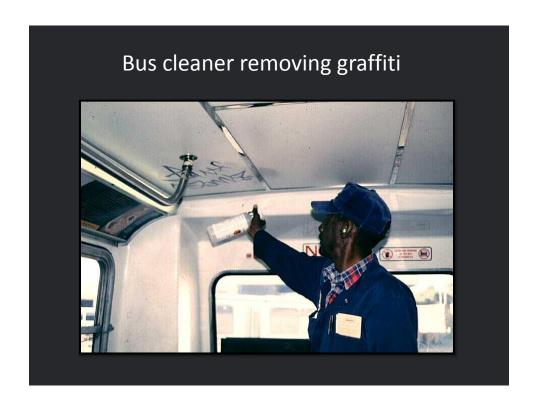


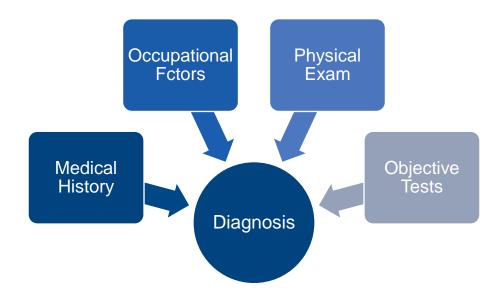




Diagnosis: irritant-induced







Medical history

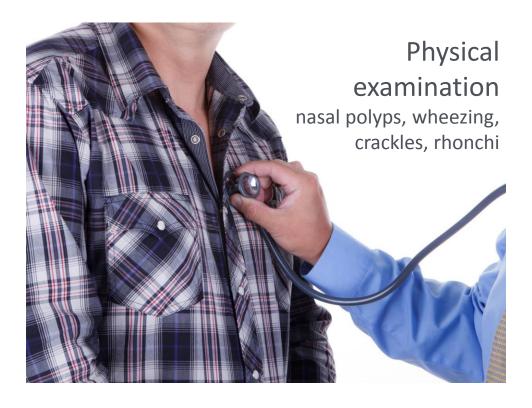
- Asthma, allergies, atopic dermatitis, cardiac history
- Tobacco and medication use



Occupational factors

- Temporal relationship between work and symptoms
- Identification of work processes, job duties, chemicals (MSDS), PPE



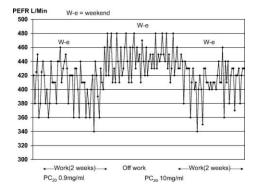


Objective tests



- CXR: bronchial wall thickening
- Spirometry: >12% improvement or absolute increase > 200 ml in FEV₁ after bronchodilator (ATS 1991)

Diagnosis of WRA



Tarlo et al; Chest 134: 1S-41S; 2008

- Decrease of 10% in FEV₁ across work shift
- > 20% diurnal variability in peak expiratory flow (PEF)
- Airway
 hyperresponsiveness with
 inhalation challenge testing
 (histamine or
 methacholine)

Diagnosis of WRA

Objective tests [continued]

- Specific inhalation challenge tests (precise etiology, test new agent)
- Skin prick tests for HMW aeroallergens
- Specific IgE antibodies against HMW and some LMW sensitizers (ex: diisocyanates, acid anhydrides)



After the diagnosis of workrelated asthma, what is the BEST management strategy?

Management of WRA

- Prompt diagnosis and removal from exposure (especially in sensitizer-induced asthma)
- Follow published guidelines for asthma management (NHLBI)
- Inhaled corticosteroids improve outcome following removal from exposure (Malo 1996)

Management of WRA

- Incomplete recovery common with sensitizerinduced asthma (Chan-Yeung 1999)
- Early removal increases likelihood of recovery (Cote 1990)
- Improved PFTs 1-2 years following removal from sensitizer exposure
- Persistent sxs > 2 years following irritantinduced asthma (Bherer 1994)

WRA and disability

- High rates of job loss/job change determined by working conditions (Blanc 1996)
- Substantial income reduction after 3 years in >50% affected (Ameille 1997)
- Impaired quality of life: increased symptoms, activity limitation, emotional dysfunction (Gassert 1998)

Preventing WRA

- Primary prevention
 - Substitute with less hazardous substances
 - Change work processes
 - Reduce exposure: engineering controls, PPE as last resort
 - Worker education and training







Preventing WRA

Secondary prevention

Detect early to minimize severity and duration

Tertiary prevention

- Provide appropriate health care: workers compensation claims
- Early removal from exposure
- Permanent impairment guidelines (ATS 1993)
- Long-term follow-up

When is illness or injury work related?

Any injury or illness resulting from or sustained in the course of any occupation or employment.

More than 50% likely due to work

USE THESE WORDS:

- "More likely than not" due to work
- Work "most likely" cause of the condition
- "But for the work" the condition would not exist

Preventing WRA: workplace surveillance

- Early detection works to prevent morbidity and disability (Tarlo 1999)
- Medical surveillance: symptom questionnaires, spirometry, PEF records, skin-prick testing for HMW antigens (flours, proteolytic enzymes, animal proteins)
- Exposure monitoring of hazards

Chemical policies

- "Green chemistry" initiatives
- "cradle to grave" or "life cycle" and the workplace
- Occupational and environmental advocacy



Green Seal – third party certification for cleaning



Before



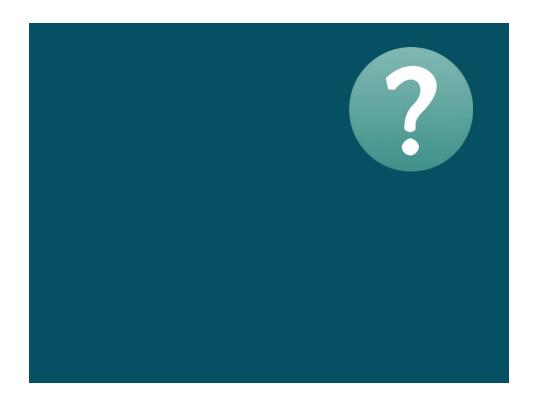
After







http://tinyurl.com/WorkRelatedAsthma 800-970-6680





Robert Harrison, MD MPH
California Department of Public Health
Occupational Health Branch
and
University of California, San Francisco

University of California, San Francisco
Division of Occupational and Environmental Medicine

TEL 415 717 1601 Email: robert.harrison@ucsf.edu

Produced under grant number DOL OSHA SH 27640 SH5 from the Occupational Safety and Health Administration, U.S. Department of Labor. It does not necessarily reflect the views or policies of the U.S. Department of Labor, nor does mention of trade names, commercial products, or organizations imply endorsement by the U.S. Government.