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Respirators

OCCUPATIONAL EXPOSURE AND 25-YEAR INCIDENCE RATE OF NON-SPECIFIC LUNG DISEASE - The Zutphen Study. D.J.J. Heederik, H. Kromhout, J. Burema, K. Biersteker, D Kromhout. University Wageningen, Wageningen, The Netherlands. Department of the National Institute of Public Health and Environmental Protection, Bilthoven, The Netherlands.

Information gathered in the 'Zutphen Study', the Dutch contribution to the Seven Countries Study that started in the sixties, was used for the present study. 878 men participated in the physical examination in 1960 and they were followed for twenty five years till July 1, 1985. During this follow-up, their morbidity status was verified regularly. With this information the occurrence of CNSLD at a specific time was coded by one physician, using strict criteria. The CNSLD diagnosis was based on the following criteria:

-episodes of regular cough and/or wheezing for longer than three months reported to the survey physician, or:

-diagnosis of CNSLD, including chronic bronchitis or emphysema by a clinical specialist.

Occupation in 1960 was coded and used to generate specific occupational exposures with a Job Exposure Matrix. Because the exact time of diagnosis of CNSLD was known, Incidence Densities could be calculated. For 804 men a complete set of data was available. A Poisson regression analysis was used to analyse the relationships between the Incidence Density and independent variables like age, calendar period, occupation and specific occupational exposure. Blue collar workers had a significantly elevated Incidence Density Ratio (IDR) compared to white collar workers (1.82, 95% confidence limits: 1.35, 2.46). Subgroups of blue collar workers, wood and paper workers, textile workers and tailors, construction workers and transport workers had significantly elevated IDRs also. Of the specific exposures heavy metals, mineral dust and adhesives had a significantly elevated IDR. Thirty percent of the population had at least one exposure to dusts, fumes or gases in their occupation and they had a significantly elevated IDR of 1.4 compared to non-exposed workers (95% confidence limits: 1.07, 1.85). These results are in concordance with a previous cross-sectional analysis of a sample of the Zutphen population and confirm the relationship between occupational exposures and CNSLD incidence.

ROLE OF SUBJECTIVE RESPONSE IN EVALUATING RESPIRATORS. P.Harber. J. Luo, J. Beck, C. Brown. University of California, Los Angeles.

Industrial respirators are often evaluated by assessing their physiologic effects, but subjective responses may affect compliance with proper use. We therefore assessed subjective response during moderate field exercise using 4 experimental periods on two separate occasions: No respirator (N), Full Mask without deflector (Fn), Full Mask with nasal deflector (Fd), Powered Air Purifying Respirator (Pa). Visual analog scales were used to rate "Exert" ("How long can you continue?) and "Disc" (How uncomfortable is it?"). Resp inductive plethysmography measured tidal volume (Vt), Inspiratory time (Ti), and Duty Cycle (DC). ANOVA measured effect of respirator type (R) and Week (W). Results, summarized below, show that both subjective scales were affected by R but not by W. Pa had the greatest adverse effect on "exert" and also affected "Disc", possibly reflecting the weight of the blower assembly. N was, as expected, best tolerated subjectively. The physiologic results showed that Pa had minimal adverse effect, Pa and N being quite similar. This reflects the lack of deadspace and of inspiratory resistance in Pa. Thus, subjective response can be measured reliably, as shown by good week to week consistency. Further, physiologic response may be disparate from subjective response. These results suggest that subjective response as well as physiologic response can and should be determined.

	Disc	Exert	Ti	DC	Vt (means)
N	2.31	5.11	0.89	0.45	0.86
Fn	2.94	4.31	1.05	0.44	1.21
Fd	3.09	4.24	1.11	0.45	1.19
Pa	3.03	4.07	0.88	0.39	0.81

(Anova showed $p < 0.01$ for R for variables except DC. It was n.s. for all W effects.)

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CLINIC BASED SURVEILLANCE FOR HAZARDS: QUESTIONNAIRE, EXPERT, AND EXPERT SYSTEM APPROACHES. P. Harber, M. Levin, J.M. McCoy, K. Bailey, K. Howard, C. Brown. University of California, Los Angeles

Clinic based surveillance for occupational hazards requires that the patient or health care worker identify the possibly causative agents. Three approaches are described in this pilot study. The Occupational Questionnaire (OQ) involved a standard interviewer administered questionnaire, asking patients to list "significant" exposures. The Occupational Expert Interview (OE) was performed by an experienced industrial hygienist in three stages: 1. Ask patient to list jobs, industries, and possible exposures; 2. Based on "expert" knowledge, suggest additional exposure possibilities; 3. Ask patient to confirm or deny the suspected exposures. The Expert System (ES) approach applied an artificial intelligence expert system to the basic interviewer data from OQ to determine possible paths from job or industry to exposure to disease. The system is a C-based expert system including a knowledge base of pairwise relationships and an inference engine (for search, predicate calculus, and selection of relevant paths). 30 successive unselected pulmonary patients participated in OE: 55 chemical, 10 product, and 41 activity items were added by the OE to those stated by the patient; confirmation by the patient was in 56%,80%, and 56% of instances respectively. 36 other pulmonary patients (mainly asthmatics) were assessed by OQ and ES. The OE results above and the table show: 1. highly standardized questionnaires elicit fewer possible exposure agents than less structured human interview (stage 1 of OE); 2. expert knowledge can add significantly, inferring from job title to exposures; 3. artificial intelligence (ES) can define a large number of possible paths from job to possible disease. ES may therefore provide occupational health expertise to primary clinicians. Clinic based surveillance can help raise the possibility of exposures for clinical or public health investigation.

Results of ES use: mean (s.d.),range:

	mean	(s.d.)	range
Agents stated by Subject	0.19	(0.46)	0-2
Activities (jobs) stated	2.83	(1.88)	0-8
Matched Activity/Subject	2.61	(1.72)	0-8
Paths/Subject	20.1	(26.3)	0-127
Paths/Activity	6.97	(8.91)	0-41

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THE ROLE OF LUNG INFLAMMATION IN METAL FUME FEVER. P.D. Blanc, B. Sicby, M.S. Bernstein, M. Wong, H.A. Boushey. University of California San Francisco, San Francisco, CA.

We carried out human exposures to study Metal Fume Fever (MFF), a flu-like illness that occurs 6 to 18 hours after inhaling zinc oxide fume. We hypothesized that a pulmonary inflammatory response was linked to the systemic syndrome of MFF. Subjects (N=7) welded galvanized steel in a controlled exposure chamber; breathing zone zinc ranged from 23 to 171 mg/M^3 over 15 to 30 minutes. We measured peripheral polymorphonuclear leukocytes (PMNs), lung volumes, flow rates, diffusing capacity for carbon monoxide (D_{Lco}), specific airway resistance (S_{raw}), and reactivity to inhaled methacholine at baseline and again 22-23 hours after challenge. We also performed bronchoalveolar lavage (BAL) at 24 hours. In addition to BAL cell type and count, we determined lymphocyte subtype in 5 subjects. Two subjects with the heaviest zinc exposure developed chills, fever and myalgia typical of MFF; the other 5 subjects were without such symptoms. In all 7 subjects the BAL yielded numerous PMNs, with a mean of 31% (range 18 to 48%). The concentration of post-challenge peripheral PMNs doubled. Zinc fume dose (air concentration* minutes of exposure) was correlated with the concentration of BAL PMNs ($r=.89$, $p<0.01$), macrophages ($r=.90$, $p<0.01$), increasing proportion of BAL PMNs ($r=.75$, $p=0.05$), and post:pre exposure peripheral PMNs ($r=.75$ $p=0.05$). Zinc dose was also correlated with the proportion of T cells identified as activated lymphocytes ($r=.98$, $p<0.01$) or inducer lymphocytes ($r=.94$, $p<0.05$). There was a slight overall fall in D_{Lco} (mean change -7%) and little to no post-challenge change in lung flow rates, volumes, S_{raw}, or reactivity. These data demonstrate a dose-dependent pulmonary inflammatory response to zinc oxide inhalation involving increases in PMNs, macrophages and certain lymphocyte sub-types. These changes did not correspond to a marked functional change in the airways. The clinical response indicated an apparent threshold in manifesting MFF, consistent with the hypothesis that MFF may be mediated by the degree and cellular characteristics of the pulmonary inflammatory response to zinc oxide inhalation.