*Project S E.N.S.O.R. News

Volume 21, No. 3 **Summer 2010**

COPD AND WORK

The causal relationship between cigarette smoking and the development of COPD is well known. What is less appreciated is the important role that workplace exposures have in causing COPD, either alone or in conjunction with cigarette smoking.

In 2003, a committee of the American Thoracic Society (ATS) reviewed the medical literature on COPD and estimated that 15% of the burden of COPD in the general population was attributable to occupational exposures (ATS, 2003). The percentage of COPD attributable to a work exposure is greater among individuals within specific cohorts of workers (such as silica or diesel exhaust exposed individuals). Table 1 summarizes the workplace exposures which increase the risk of COPD.

Further studies since the ATS consensus document have addressed the role that workplace exposures have in the etiology of COPD. This newsletter highlights studies published in the last two years showing the importance of considering workplace exposures in the etiology and management of COPD.

Table 1. Workplace Exposures Associated With Increased Risk of COPD

Asbestos
Cadmium Oxide
Chlorine Gas
Cotton Dust
Diesel Exhaust
Non-specified vapors, gases, dusts or fumes
Silica

Non Specified Vapors, Gases, Dusts or Fumes

In a case-control study from California of patients 55-75 years of age with a physician diagnosis of COPD, 233 patients were interviewed about their cigarette smoking and work exposure to vapors, gases, dust or fumes. The population attributable risk for work exposures was 32% using physician diagnosis of COPD and 17% using spirometric results of a reduced FEV_1/FVC ratio. Odds ratios were 2-3 for work exposures alone, 3-5 for smoking alone and largest at 5-9 for those with both a history of work exposures and cigarette smoking (Blanc et al, 2009a).

The same authors conducted a larger case-control study of patients 40-65 years old treated for COPD in the Kaiser Program in Northern California. Here they reported that joint exposure to smoking and work exposures increased the risk of COPD 14.1 times with a 95% confidence interval (C.I.) of 9.33 – 21.2 (Blanc et al, 2009b). Another case-control study of patients with COPD 45 years or older from the Kaiser program in Oregon found a similar association with workplace exposures (Weinmann et al, 2008).

A cohort of 2,734 workers, ages 18-58, was followed over 10 years in southern Italy. Independently, both cigarette smoking and workplace exposures were risk factors for the development of COPD with the risk being greatest in those individuals who had both workplace exposures and were smokers (Boggia et al, 2008).

A longitudinal study of 3,208 Danish men found that men who were smokers and had exposure to solvents had a 7.0 fold (95% C.I. 3.4-14.5) increased risk for developing chronic bronchitis, versus 3.7 (95% C.I. 2.8 – 4.8) for smokers alone and no increased risk among solvent exposed workers who were non-smokers (Ebbehoj et al, 2008).

Severity of COPD assessed by different parameters including FEV_1 <30%, respiratory symptoms or work inactivity were associated independent of cigarette smoking with workplace exposures in a study of 194 patients with COPD treated at a specialty clinic in Spain (Rodriguez et al, 2008).

COAL

An autopsy study of 722 coal miners found that emphysema severity was significant in both non-smoking and smoking miners compared to non-smoking and smoking *non-miners*. The amount of coal in the autopsied lungs correlated with the severity of emphysema (Kuempel et al, 2009). These results are consistent with previous reports of increased COPD in coal miners including a recent study reporting increased mortality from COPD among coal miners (Attfield and Kuempel, 2008).

DIESEL EXHAUST

Since 1959, 95% of the locomotives in the United States have been powered by diesel fuel. A study of diesel exhaust-exposed railroad workers which included engineers, firemen, brakemen and hostlers found a 2.1% increase in mortality from COPD for each year of work in a diesel exposed job while controlling for cigarette smoking (Hart et al, 2009).

Management of COPD and Work Exposures

Despite the contribution of work exposure to COPD reported in the medical literature some physicians do not consider work exposures when evaluating a patient with COPD. A review of 6,150 medical records on 54 patients with chronic bronchitis from a Veteran's Administration hospital in California found three patients (5%) where the medical record stated that work exposures potentially contributed to the etiology of the chronic bronchitis and six (10%) where avoidance of workplace exposures was recommended (Kuschner et al, 2009).

The consequences from a lack of attention to workplace exposures in patients with COPD has been documented in the Lung Health Study, a longitudinal follow-up of 5,724 individuals with early COPD. One of the conclusions of that study was that "In men with early COPD, each year of continued fume exposure was associated with a 0.25% predicted reduction in post-bronchodilator FEV_1 % predicted" (Harber et al, 2007).

Statistical models have been developed that predict that COPD could be reduced by 20% by an 8.8% decrease in the prevalence of occupational exposures. In comparison, these models predict the same 20% reduction in COPD with a 5.4% reduction in smoking (Blanc et al, 2009c).

The absence of studies on specific levels of exposure and/or a specific irritant can make clinical decisions on managing a patient with emphysema difficult. We know it is important to counsel such a patient to stop smoking. If such patients have exposures to chemicals and dusts at work, should physicians advise their patients to leave their job? Given the adverse economic consequences of losing a job, this advice cannot be given too lightly. The use of a peak flow meter to indicate acute changes and/or serial spirometry showing chronic loss would allow the decision to be made on objective evidence.

It is also difficult to decide in the context of workers' compensation whether a patient's occupational exposure is a contributing factor in the development of their emphysema. All too often a worker's respiratory symptoms are summarily attributed to past or current cigarette smoking. While there is no generalizable answer as to the importance of a person's workplace exposure,

the individual's smoking habits, duration of occupational exposure and types of exposure need to be considered. In addition, cigarette smoking does not cause the x-ray changes of fibrosis, the large opacities of progressive massive fibrosis or pleural thickening. The presence of the above x-ray findings should heighten the physician's concern about work-place exposures.

The bottom line is that a patient's cigarette smoking habits are not necessarily a sufficient explanation for the patient's respiratory condition.

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The Project SENSOR News is published quarterly by Michigan State University-College of Human Medicine with funding from the National Institute for Occupational Safety and Health and is available at no cost. Suggestions and comments are welcome.

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