The Impact of Indoor Air Quality and Temperature on Symptom Burden for COPD Patients

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Background: Housing characteristics (heating and ventilation) may exacerbate effects of external cold and air pollution on respiratory health. Winter mortality in the UK has been linked to poor heating levels in homes especially of the elderly. Previous studies in predominantly non smoking households have found that indoor PM2.5 is close to outdoor levels. This study investigated the impact of indoor temperature and PM2.5 on symptom burden in COPD outpatients in Northeast Scotland. **Method:** Living rooms and bedrooms in homes of COPD outpatients were monitored during winter months. PM2.5 ugms/m3 was measured with Dustrak(TM) monitors for 14 hours and iLog(TM) temperature and humidity dataloggers for 168 hours. Participants completed the St George's Respiratory Questionnaire and a demographic questionnaire. Smoking and non smoking households were included. **Results:** 149 patients agreed to be monitored. Mean age was 68 (9.5) yrs, FEV1 1.06 (0.52). 40 patients and 65 households smoked. PM2.5 levels were highly determined by household smoking status. Median PM2.5 in non smoking households was 25 ugms/m3, in smoking households 270 ugms/m3. Median living room temperature over one week was 20°C (Range 7°C to 37°C). Median SGRQ Symptom score was 80.6, Impact 59.3, Activities 92.5. Symptom scores were significantly associated with PM2.5 levels and hours in which living room temperature was below 21°C. Impact and Activity scores were associated with age and FEV1 but not temperature or PM2.5. **Conclusions:** The impact of Environmental Tobacco Smoke on air quality in COPD patients' homes and the associated symptom burden may have been underestimated. PM2.5 levels were 3 to 4 times those found in previous studies, due to the greater number of smoking households in this study. Indoor temperatures below recommended levels also put patients at risk and were associated with low energy efficiency of homes.

Female Predominance of Severe Early-Onset COPD in Poland and the United States

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In the Boston Early-Onset COPD Study, severe early-onset COPD has demonstrated a marked female predominance, but it is unclear whether early-onset airflow limitation demonstrates a similar sex bias at the population level. We analyzed the prevalence of early-onset COPD defined as severe airflow limitation at age less than 53 years, in The Know the Age of Your Lung Study, a large national screening project in Poland, and the Third National Health Nutrition Examination Survey (NHANES III) in the United States. In the Polish cohort, 110,778 Caucasian individuals had spirometry data available. The mean pack-years of smoking was higher amongst the men (31 vs 21 pack-years, p=0.001). Of the 22,683 individuals with airflow limitation, there was a male predominance (24% males vs. 16% female,p=0.0001). However, amongst 811 individuals with severe early-onset COPD (FEV$_1$< 40%, FEV$_1$/FVC < .7) before age 53, the proportion of females was higher than the proportion of males (4.1% vs 3.4%, p<0.01). In NHANES III, 3,123 Caucasian individuals had spirometry data available for this analysis;due to lower numbers we categorized by GOLD stages.620 individuals with spirometry were categorized in GOLD stages 1-4. Of the 244 women, 46% were in GOLD stages 2-4; of the 376 men, 59% were in GOLD stages 2-4.When the analysis was further limited to those less than 53 years of age, 111 individuals (42 women, 69 men) were categorized in GOLD stage 1-4. Of the women, 48% were in GOLD stage 2-4 versus 42% of the men. In two large population screening evaluations, although men appear to have a higher rate of airflow limitation at the population level, women have proportionately more severe disease at an earlier age, supporting the hypothesis that women may be more susceptible to the pulmonary effects of tobacco smoking.

✳ COPD and Occupational Exposures: A Case-Control Study

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RATIONALE: Growing evidence suggests that COPD is causally associated with certain occupational exposures. Few studies have investigated the association using complete job histories and multiple types of occupational exposure.

METHODS: We conducted a case-control study among members of Kaiser Permanente Northwest to evaluate the associations between occupational exposure and COPD. We identified 388 COPD cases aged 45 and over; 356 controls without COPD were frequency matched to cases on age group, sex, and history of smoking. Detailed work and smoking histories were obtained by telephone interview. Certified Industrial Hygienists independently rated each job according to its likelihood of exposure to 8 agents thought to cause COPD. Odds ratios for the association of each exposure (ever-never) with COPD were computed using logistic regression, stratifying on smoking status and adjusting for age, sex, and, for smokers, pack years.

RESULTS: Among ever-smokers, occupational exposures most strongly associated with COPD were diesel exhaust (OR=1.7, 95% CI 1.1-2.7) and irritant gases and vapors (OR=1.7, 95% CI 1.2-2.4). Associations with metal dust (OR=1.5, 95% CI 0.95-2.5) and mineral dust (OR=1.5, 95% CI 0.95-2.3) were suggested. In the small group of never-smokers (42 cases and 58 controls), a similar pattern was evident though statistical power was low; the strongest associations found were with diesel exhaust (OR=6.4, 95% CI 1.3-31.6) and mineral dust (OR=3.5, 95% CI 0.94-13.3).

CONCLUSIONS: In this study, COPD was associated with exposure to diesel exhaust and irritant gases and vapors.

Chronic Obstructive Pulmonary Disease (COPD) in the Framingham Heart Study (FHS)

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Introduction: Prior reports have estimated that 10-20% of smokers have COPD. FHS is a cohort study, designed to improve our understanding of cardiovascular disease. Two cohorts have been followed for extended periods, the Original Cohort since 1948 and the Offspring since 1971. Subjects underwent spirometry at most examinations. Thus FHS offers a unique opportunity to understand the risk of developing COPD. **Methods:** Using data from the most recent examinations with spirometry (19th and 7th examinations of Original and Offspring cohorts, respectively), we explored the prevalence of COPD, defined by GOLD stage 2 or higher. **Results:** Among 2484 Offspring and 1998 Original cohort participants, 15% of ever smokers had COPD. There were differences between the cohorts: 12% of ever smokers in the Original cohort had COPD, versus 17% in the Offspring. COPD prevalence was concentrated among heavier smokers (≥30 pack years): 20% of participants in the Original cohort and 35% in the Offspring cohort who were heavier smokers had COPD. This appears to be related to differences between cohorts in the prevalence of COPD among female participants: 38% of the female Offspring heavier smokers had COPD, as compared to 13% of the female Original cohort heavier smokers. **Conclusions:** The prevalence of COPD among ever smokers was somewhat higher than is often cited; approximately 17% of ever smokers (excluding the female participants in the Original cohort) had COPD. Among heavier smokers, nearly 1/3 had COPD. The differences between the female participants in the Original cohort and the rest of the sample may be explained by secular changes in smoking habits among women.

Early Life Symptoms Predict Chronic Bronchitis in Middle-Age: A Longitudinal Study from Age 7 to 44 Years

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Background: Identification of early symptoms for chronic bronchitis (CB) in middle age provides an opportunity to identify subjects at risk of COPD. **Aim:** To identify early life symptom predictors of CB in middle age. **Methods:** The Tasmanian Asthma Study is a longitudinal study of respiratory disorders in a population based birth cohort of 8,500 that was followed from age 7 to 44 years. Information on respiratory symptoms was collected at ages 7, 14, 21, 32 and 44 years either on the total cohort or samples. CB was defined as cough and sputum at least for three months on two consecutive years. Multiple Logistic Regression analysis was used to identify the predictors. **Results:** The prevalence of CB was 12.7%, being 12.9% in males and 12.5% in females. Of these CB subjects 34% reported coexisting asthma. 76% of CB subjects were smokers compared to 57% of those without CB (p=0.0001) and those with CB started smoking significantly younger than those without CB (p=0.002). Of the subjects with CB 24% and 12.3% had wheeze at ages 7 and 14 respectively, which was significantly different to those without CB (19.9%, 7.6%)(p=0.06 & 0.002 respectively). Cough at ages 7 and 14 was not significantly related to CB. In non-smokers, wheeze at age 13 predicted CB at age 44 years (OR 1.71, 95%CI 1.32, 2 76). In smokers, neither wheeze nor cough predicted CB. **Conclusions:** The prevalence of CB is high among middle age adults, which can be predicted by symptoms in early life and smoking. Further follow-up and lung function testing are needed to establish how many of them will go on to develop chronic obstructive pulmonary disease.

The Effect of Cigarette Smoke Exposure on Immune Inflammatory Responses to Influenza A Is Dependent on Viral Dose

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Viral infection is a major etiologic factor in the development of COPD exacerbation. Evidence also suggests that cigarette smoke (CS) impacts both innate and adaptive components of the immune system. Exacerbation of disease may therefore be attributed in part to an impaired ability to appropriately deal with infectious agents. The purpose of this study was to investigate the impact of CS on immune inflammatory responses toward pulmonary infection with influenza A. Mice were exposed to CS for 12-16 weeks and infected with a mouse adapted influenza A virus. Compared to sham exposure, CS exacerbated the airway inflammatory response following infection with a high dose of influenza A, increasing the number of both mononuclear cells and neutrophils. The heightened inflammatory response in CS-exposed mice was associated with augmented production of type 1 interferon and IL-6 in the airway and increased morbidity and mortality. In sharp contrast, infection with a low dose of influenza A led to an attenuated airway inflammatory response in CS-exposed mice compared to sham-exposed animals, decreasing the number of mononuclear cells and neutrophils. CS exposure also decreased the percentage of granzyme B expressing CD8 T cells in the lung. Despite the blunted immune inflammatory response, virus was cleared more rapidly from the lungs of CS-exposed animals compared to sham-exposed mice. Furthermore, CS exposure did not impair the establishment of influenza-specific memory responses since sham- and CS-exposed animals were equally protected from secondary exposure to the virus. Our studies demonstrate that the effect of CS on anti-viral immunity in the lung may be to some extent dependent on the magnitude of the infection.

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ABSTRACTS
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