Air pollution exposure increases risk of severe COPD

October 19 2010

Long term exposure to low-level air pollution may increase the risk of severe chronic obstructive pulmonary disease (COPD), according to researcher s in Denmark. While acute exposure of several days to high level air pollution was known to be a risk factor for exacerbation in pre-existing COPD, until now there had been no studies linking long-term air pollution exposure to the development or progression of the disease.

The research was published online ahead of the print edition of the American Thoracic Society's American Journal of Respiratory and Critical Care Medicine.

"Our findings have significance on a number of levels," said lead researcher on the study, Zorana Andersen, Ph.D., post doctoral fellow at the Institute of Cancer Epidemiology of the Danish Cancer Society in Copenhagen. "Patients, primary care physicians, pulmonologists and public health officials should all take not of our findings."

Dr. Andersen and colleagues used data from the Danish Diet, Cancer, and Health Study, which consisted of more than 57,000 individuals between the ages of 50 and 64 who lived in Copenhagen or Aarhus, the first and second largest cities in Denmark, between 1993 and 1997. A self-administered questionnaire provided data on smoking, dietary habits, education, occupational history and lifestyle. They then used the unique personal identifiers to link the cohort to the Danish Hospital Discharge Register to identify hospital admissions and discharges due to COPD, and estimated pollution exposure by linking residential addresses

to outdoor levels of NO2 and NOx levels, which were used to approximate the overall level of traffic-related pollutants since 1971. They looked at exposures over 15-, 25- and 35-year periods to assess the effect of different exposure lengths on COPD incidence. Data for more than 52,000 were available from the start 1971 to the end of follow-up in 2006.

"We found significant positive associations between levels of all air pollution proxies and COPD incidence," said Dr. Andersen. "When we adjusted for smoking status and other confounding factors, the association remained significant, indicating that long-term pollution exposure likely is a true risk factor for developing COPD."

These associations were slightly stronger for men, obese patients and those eating less than 240 grams of fruit each day (approximately eight ounces, or just more than a single serving). But notably, the effect of air pollution on COPD was strongest in people with pre-existing diabetes and asthma.

"These results are in agreement with those of other cross-sectional studies on COPD and air pollution, and longitudinal studies of air pollution and lung function, and strengthen the conclusion that air pollution is a causal agent in development of COPD," said Dr. Andersen.

Because the study used hospital admissions for COPD to assess incidence, it is likely that the true incidence was underestimated, and that the cases represented severe COPD, as mild and moderate COPD does not often require hospitalization. This means that the reported increase in risk associated with air pollution is probably an underestimate of the true increase in risk for COPD in general. Furthermore, while smoking is known to be the primary cause of COPD in developed countries, and majority of COPD cases were smokers or previous smokers, the effect of pollution exposure was also observed in

the group of non-smokers. "This result refutes the possibility that the observed effect of air pollution was due to inadequate adjustment for smoking in our data and supports the idea that air pollution affects COPD risk, irrespective of smoking status," said Dr. Andersen.

The enhanced association between increased risk of COPD and air pollution in asthmatics and diabetics suggests the possibility of an underlying link. "It is plausible that airflow obstruction and hyper-responsiveness in people with asthma, or systemic inflammation in people with diabetes, can lead to increased susceptibility of the lung to air pollution, resulting in airway inflammation and progression of COPD, but more research is needed in this area." said Dr. Andersen.

"In any case, sufficient data, including the results of this study, provide evidence that traffic-related urban air pollution contributes to the burden of COPD and that reductions in traffic emissions would be beneficial to public health."

Provided by American Thoracic Society