Medical Xpress

Wood smoke exposure multiplies damage from smoking, increases risk of COPD

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Smokers who are exposed to wood smoke, either through home heating and cooking or through ambient neighborhood pollution, are not only at increased risk of COPD, but are also more likely to have epigenetic changes in the DNA that further increase their risk of COPD and related pulmonary problems.

Together, smoking, wood smoke exposure and these epigenetic changes can increase an individual's risk of COPD fourfold.

"When cigarette smokers are exposed to wood smoke their risk of having reduced lung function increases," explained lead author Yohannes Tesfaigzi, Ph.D. senior scientist and director of COPD Program at the Lovelace Respiratory Research Institute, where the research was completed. "Cigarette smokers who have both changes in sputum DNA and are exposed to wood smoke have a synergistically increased risk of having reduced lung function and other indicators of COPD such as chronic mucous hypersecretion. "

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

Dr. Tesfaigzi and colleagues administered questionnaires to more than 1800 current and former smokers between 40 and 75 years old, and obtained demographic and smoke exposure information, as well as sputum samples which were analyzed for epigenetic changes to eight

genes known to be associated with lung cancer.

They found that wood smoke exposure was significantly and independently associated with an increased risk of respiratory disease, especially among current smokers, non-Hispanic whites and men. Furthermore, wood smoke exposure was associated with specific COPD outcomes in people who had aberrantly methylated p16 or GATA4 genes, and both factors together increased the risk more than the additive of the two risk factors together. They also found that people with more than two of the eight genes analyzed showing methylation were also significantly more likely to have a lower than predicted FEV1 than those with fewer than two methylated genes.

"Because exposure to wood smoke appears to increase the risk of reducing lung function, cigarette smokers should try to avoid heating their homes or cooking with wood stoves and try to avoid environments where wood smoke is likely (for example, neighborhoods where wood smoke is common)," said Dr. Tesfaigzi. "Because the same gene changes were associated with increased risk for lung cancer one would assume that wood smoke exposure also increases the risk of developing lung cancer. Future studies may show that it would be appropriate to screen patients for lung cancer if these exposures were present for prolonged periods."

Based on these findings, Dr. Tesfaigzi and colleagues established an animal model that will be able to further test whether both wood and tobacco smoke exposure cause more damage to the lung than either one of the exposures alone. "We observed increased inflammatory response in mice that were exposed to both cigarette smoke and low concentrations of wood smoke compared to those exposed to cigarette smoke only. We would like to use this animal model to determine the mechanisms underlying this exacerbation," said Dr. Tesfaigzi.

Because wood smoke exposure was documented by self-report and was not quantified in this study, in the future Dr. Tesfaigzi also intends to characterize the type and amount of wood smoke the participants were exposed to. Such studies will help to further refine the analysis and provide intervention strategies.

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