

# Miller School Researchers Find Link between E-Cigarettes and Lung Infections

A team of researchers at the University of Miami Miller School of Medicine has found a link between the use of electronic cigarettes and the risk of development of mycobacteria-related lung infections.



Introduced to the U.S. marketplace in 2007, e-cigarettes have become the most commonly used tobacco product among American youth.

According to their study, the chemicals found in e-cigarettes enhance the replication of mycobacteria, causing a significant

type of lung infection that is difficult to treat. The findings were reported in an [article](#) in the *American Journal of Respiratory Cell and Molecular Biology*.

“Our findings are alarming because e-cigarettes are becoming increasingly popular. Dr. Jerome Adams, Surgeon General of the U.S. Public Health Service, officially announced that e-cigarettes are an epidemic among youth,” said Mehdi Mirsaeidi, M.D., M.P.H., assistant professor in the Miller School Department of Medicine’s Division of Pulmonary and Critical Care, who was senior author of the journal article. “E-cigarettes were introduced to the U.S. marketplace in 2007, and they have become the most commonly used tobacco product among U.S. youth.

“According to the Food and Drug Administration, currently more than 8 million people smoke e-cigarettes in the U.S., including a growing number of adolescents seeking social approval. Other research has shown that non-smoker adolescents who try e-cigarettes – which are marketed as a safer alternative to traditional cigarettes – are likely to initiate cigarette smoking in the future.”

Prior studies have shown that e-cigarettes vaporize a mixture of compounds, including nicotine, propylene glycol, and glycerin, to the lungs. Smoking traditional cigarettes and e-cigarettes disrupts lung homeostasis, inducing inflammation. In a healthy human lung, exposure to nicotine and aerosol components, including vegetable glycerin, propylene glycol and flavoring chemicals, activates and alters the expression of the nicotine

receptor pathway genes found in the small airway epithelium and alveolar macrophages.

Nonetheless, few studies have been able to shed light on the mechanisms of how mycobacteria infects the respiratory epithelia, and there has been a general lack of knowledge about how e-cigarettes interact with pulmonary epithelial cells infected with mycobacteria. To learn more, Dr. Mirsaeidi and his team developed a bilayer lung model with human bronchial epithelial cells and human lung endothelial cells to evaluate the effect of e-cigarettes on mycobacteria replication. Their analysis showed definitively that e-cigarettes enhanced replication and significantly increased key proinflammatory cytokines in the lung model.

The researchers called their findings “alarming,” because of the growing popularity of e-cigarettes among adolescents, and the finding that e-cigarette users show increased levels of crucial cytokines, which puts them at a higher risk for pulmonary infections and diseases. The next step will be to request funding from private and governmental institutes, including the National Institutes of Health, for a larger study in defining the mechanism effect of e-cigarettes in cellular and animal models.

Dr. Mirsaeidi’s Miller School faculty co-authors were Michael Campos, M.D., associate professor of medicine, Gregory Conner, Ph.D., associate professor of cell biology, Nevis Fregien, Ph.D., associate professor of cell biology, and Gregory Holt, M.D., Ph.D., assistant professor of medicine.

