E-cigarettes may boost resistance of drug-resistant pathogens

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Despite being touted by their manufacturers as a healthy alternative to cigarettes, e-cigarettes appear in a laboratory study to increase the virulence of drug- resistant and potentially life-threatening bacteria, while decreasing the ability of human cells to kill these bacteria

Researchers at the VA San Diego Healthcare System (VASDHS) and the

University of California, San Diego (UCSD), tested the effects of e-cigarette vapor on live methicillin-resistant Staphylococcus aureus (MRSA) and human epithelial cells. MRSA commonly colonizes the epithelium of the nasopharynx, where the bacteria and epithelial cells are exposed constantly to inhaled substances such as e-cigarette vapor and cigarette smoke.

"The virulence of MRSA is increased by e-cigarette vapor," said lead investigator Laura E. Crotty Alexander, MD, VA researcher and assistant professor of medicine in pulmonary and critical care at UCSD. Exposure to e-cigarette vapor increased the virulence of the bacteria, helping MRSA escape killing by antimicrobial peptides and macrophages. However, she added, the vapor did not make the bacteria as aggressive as cigarette smoke exposure did in parallel studies her group conducted.

To conduct the e-cigarette vapor experiment, the researchers grew MRSA (USA 300 strain) in culture with vapor concentrations similar to inhalers on the market. They tested first for biochemical changes in the culture known to promote pathogen virulence and then introduced epithelial cell- and alveolar macrophage-killing assays.

The study was presented at the 2014 American Thoracic Society International Conference.

The researchers looked at five factors that contribute to MRSA virulence: growth rate, susceptibility to reactive oxygen species (ROS), surface charge, hydrophobicity and biofilm formation. In particular, e-cigarette vapor led to alterations in surface charge and biofilm formation, which conferred greater resistance to killing by human cells and antibiotics.

Crotty Alexander said that one possible contribution to the increased

virulence of MRSA was the rapid change in pH induced by e-cigarette vapor. Exposure changed the pH from 7.4 up to 8.4, making the environment very alkalotic for both bacterial and mammalian cells. This alkalosis stresses the cells, giving them a danger signal, leading to activation of defense mechanisms. The bacteria make their surface more positively charged, to avoid binding by the lethal antimicrobial peptides produced by human innate immune cells. The bacteria also form thicker biofilms, increasing their stickiness and making MRSA less vulnerable to attack.

These changes make MRSA more virulent. However, when MRSA is exposed to regular cigarette smoke, their virulence is even greater. Cigarette smoke induces surface charge changes 10-fold greater than that of e-cigarette exposure, alters hydrophobicity and decreases sensitivity to reactive oxygen species and antimicrobial peptides. In a mouse model of pneumonia, cigarette smoke exposed MRSA had four-times greater survival in the lungs, and killed 30% more mice than control MRSA. E-cigarette vapor exposed MRSA were also more virulent in mice, with a three-fold higher survival.

Unfortunately, while e-cigarette vapor is increasing bacterial virulence, Crotty Alexander has found that the vapor is also decreasing the ability of human epithelial cells to kill pathogens.

"As health care professionals, we are always being asked by patients, "Would this be better for me?" Crotty Alexander said. "In the case of smoking e-cigarettes, I hated not having an answer. While the answer isn't black and white, our study suggests a response: even if e-cigarettes may not be as bad as tobacco, they still have measurable detrimental effects on health."

More information: Electronic Cigarette Vapor (ECV) Exposure Decreases Staphylococcus Aureus Susceptibility To Macrophage And

Neutrophil Killing, Late Breaking Abstract, 10.06 - Host Defenses and Pathogenic Mechanisms Related to Respiratory Pathogens (MTPI), by L.E. Crotty Alexander1, S. Enany1, H. Hwang1, K. Sladewski1, V. Nizet2; 1VASDHS, University of California at San Diego - San Diego, CA/US, 2University of California at San Diego - La Jolla/US