Vapor From e-Cigarettes Renders Protective Lung Cells Defenseless, Study Finds

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A study that sought to replicate the effects of vaping on lung cells found that vapor from e-cigarettes boosts the production of inflammatory chemicals and disables key protective cells in the lung that engulf potentially harmful particles. Some of the effects were similar to those seen in regular smokers and people with chronic obstructive pulmonary disease.

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The study, published online in the journal *Thorax*, said e-cigarettes may be more harmful than believed, as some of the effects were similar to those seen in regular smokers and people with chronic obstructive pulmonary disease (COPD). Regular use of electronic cigarettes is associated with increased odds of having COPD, according to 1 recent study.

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The vapor impairs the activity of alveolar macrophages (AM), which engulf and remove dust particles, bacteria, and allergens that have evaded the other mechanical defenses of the respiratory tract.
Vaping is increasing in popularity, but most current research has focused on the chemical composition of e-cigarette liquid before it is vaped.

The researchers said this is the first study to report human AM responses to e-cigarette vapor condensate (ECVC) and demonstrates dose-dependent cytotoxicity, inducing cell death, with both nicotine-dependent and independent responses, which the vaping process accentuates. At sub-cytotoxic doses, ECVC enhances production of reactive oxygen species (ROS) release, inflammatory cytokines, chemokines, and metalloproteinases, although the response is less pronounced with nicotine-free ECVC. Bacterial phagocytosis by macrophages is inhibited acutely by ECVC, and the effects are attenuated by the antioxidant N-acetyl-cysteine, suggesting ROS and reactive aldehydes play a role in the effects of ECVC/nfECVC.

These effects appear to be partially dependent on phosphopinositol 3 kinase (PI3K). ROS-induced lung inflammation in COPD has been reported to be associated with activation with PI3K.

The researchers devised a mechanical procedure to mimic vaping and produce condensate from the vapor. They extracted alveolar macrophages from lung tissue samples provided by 8 nonsmokers who had never had asthma or COPD.

One-third of the cells were exposed to plain e-cigarette fluid, one-third to different strengths of the artificially vaped condensate with and without nicotine, and one-third to nothing for 24 hours. The results showed that the condensate was significantly more harmful to the cells than e-cigarette fluid and that these effects worsened as the dose increased.

After 24 hours of exposure, the total number of viable cells exposed to the vaped condensate was significantly reduced compared to the untreated cells. Condensate containing nicotine exaggerated this effect.

Exposure to the condensate increased cell death and boosted production of oxygen-free radicals by 50-fold. It significantly increased the production of inflammatory chemicals, even more so when the condensate contained nicotine.

The ability of cells exposed to vaped condensate to engulf bacteria was significantly impaired, although treatment with an antioxidant restored this function and helped lessen some of the other harmful effects.

The researchers conclude that the vaping process itself can damage vital immune system cells in a way similar to that observed in cigarette smokers and in patients with COPD, at least under laboratory conditions.

Reference

Scott A, Lugg ST, Aldridge K, et al. Pro-inflammatory effects of e-cigarette vapour condensate on human alveolar macrophages. [published online August 13, 2018]. *Thorax*. doi: 10.1136/thoraxjnl-2018-211663.

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