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**Effects of Diesel Exhaust on Epithelial Cells: Potential Interactions with Viral Infections**

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The respiratory epithelium is a potential target for many inhaled particles, including diesel exhaust particles (DEP). Exposure to DEP increases markers of inflammation and oxidative stress in several cell types, including respiratory epithelial cells. The respiratory epithelium is also the primary host tissue for viral infection and replication of numerous respiratory viruses, including influenza virus. Thus, interactions between inhaled DEP and influenza virus at the level of the respiratory epithelium are likely. Previous studies in mice indicated that exposure to DE can modify the susceptibility to respiratory viruses, yet the mechanisms of these effects are poorly understood. Using both human in vitro and mouse in vivo experimental approaches, we investigated whether exposure to DEP modifies the susceptibility to influenza virus and the cellular mechanisms mediated these effects. Briefly, human respiratory epithelial cells were exposed to an aqueous-trapped solution of diesel exhaust prior to infection with influenza virus. We examined markers of viral replication, antiviral defense responses, and oxidative stress 24 hours post-infection. Our data demonstrate that exposure to diesel exhaust increases the number of influenza-infected cells not by suppressing interferon-related antiviral mediator production but by increasing the ability of the virus to attach and enter the host cell. In addition our data suggest that exposure to DE increases cellular oxidative stress and that the effects of DE on influenza virus infections are mediated by the ability of DE to induce oxidative stress in epithelial cells. Similarly, our in vivo data demonstrate that repeated exposures of mice to moderate levels of DE, increased the susceptibility to influenza virus and consequently influenza-induced inflammation and lung injury. Similar to our in vitro experiments, interferon-related antiviral defense response were not suppressed by DE. However, preliminary analysis suggests that the increased susceptibility to influenza virus may be caused by the effects of DE on host innate antiviral defense mediators, such as surfactant proteins. Taken together, our data indicate that exposure to DE “primes” the respiratory epithelium for subsequent infection with respiratory viruses, such as influenza virus. Considering the likelihood of concurrent exposures to ambient particulate matter, such as DE, and community acquired influenza virus infections, interactions between DE and influenza virus at the level of the respiratory epithelium could have significant public health implications.

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