Development of a system for the detection of the inflammatory response induced by airborne fine particulate matter in rat tracheal epithelial cells

Abstract

Exposure to airborne particulate matter (PM) is related to the increased risk of several diseases, including chronic and allergic rhinitis. We have previously shown that atmospheric endotoxin level was positively associated with the number of emergency department visits for asthma even after adjusting for meteorological factors, suggestive of the significant association between atmospheric endotoxin level and asthma exacerbation. Whether atmospheric endotoxin level is related to inflammatory response induction is, however, unclear. Here, we established stable cell lines to determine the promoter activity of the genes encoding pro-inflammatory cytokines such as tumor necrosis factor alpha, interleukin 6 (IL6), and IL33 by transfection of each reporter plasmid into rat tracheal epithelial EGV-4 T cells. These cells could measure the inflammatory response induced by endotoxin treatment more easily, rapidly, and sensitively than the conventional system using immunodetection assays. Furthermore, we revealed a relationship between atmospheric endotoxin level and inflammatory response induction. Thus, the system established herein may serve as a promising tool to monitor inflammatory response induced upon PM exposure.