Motorcycle Exhaust Particles Induce IL-8 Production Through NF-kB Activation in human Airway Epithelial Cells

鄭幼文
Lee CC;Cheng YW;Kang JJ

Abstract

Motorcycle exhaust particles (MEP) are among the major air pollutants, especially in urban area of Taiwan. In our previous study, data showed that MEP induce proinflammatory and proallergic response profiles in BALB/c mice. Effects of MEP on interleukin (IL)-8 production in A549 human airway epithelial cells were further investigated in this study. It was found that MEP enhanced IL-8 protein and mRNA expression in human epithelial cells. Pretreatment with an NF-kappaB inhibitor (1 mM PDTC), extracellular signal-regulated kinase (ERK) inhibitor (50 microM PD98059), JNK inhibitor (25 microM SP600125), p38 inhibitor (2 microM SB203580), and three antioxidants (500 U/ml superoxide dismutase [SOD], 50 microM vitamin E, 10 mMN-acetylcysteine [NAC]) attenuated the MEP-induced increase in IL-8 production. Through further, direct detection of nuclear factor (NF)-kappaB activation in epithelial cells using immunoblotting of nuclear p65 and NF-kappaB reporter assay, data showed that MEP induced nuclear translocation of p65 and enhancement of NF-kappaB luciferase gene expression. MEP also induced activation of ERK, JNK, and p38 signaling pathways and produced an increase of oxidative stress in A549 cells. By using mitogen-activated protein kinase (MAPK) inhibitors and antioxidant, it was demonstrated that ERK inhibitor, JNK inhibitor, and antioxidants but not p38 inhibitor attenuated the MEP-induced increase in NF-kappaB reporter activity. In conclusion, evidence shows that filter-trapped particles emitted from unleaded gasoline-fueled, two-stroke motorcycle engines induce an increase in IL-8 production by activation of NF-kappaB in human airway epithelial cells.