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An environmental quinoid polycyclic aromatic hydrocarbon, acenaphthenequinone (AcQ), modulates COX-2 expression through ROS generation and NF-κB activation in A549 cells

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Abstract

Diesel exhaust particles (DEP) contain oxygen-containing polycyclic aromatic hydrocarbons (PAHs) called quinoid PAHs. Several quinoid PAHs generate free radicals as they undergo enzymatic and nonenzymatic redox cycling with their corresponding semiquinone radicals. Reactive oxygen species (ROS) produced by these reactions can cause severe oxidative stress connected with inflammatory processing. Although humans and animals are continuously exposed to these chemicals in the environment, little is known about which quinoid PAHs are active. In this study, we estimated the intracellular ROS production and NF-κB translocation in A549 cells exposed to isomers of quinoid PAHs having 2 to 4 rings. We found that both acenaphthenequinone (AcQ) and 9,10-phenanthrenequinone (PQ) enhanced ROS generation and that AcQ translocated NF-κB from the cytosol to the nucleus. However, PQ, which was reported to induce apoptosis, did not influence NF-κB activation. In addition, AcQ induced cyclooxygenase-2 (COX-2) expression which is a key enzyme in the inflammatory processing involved in the activation of NF-κB. Up-regulation of NF-κB and COX-2 expression by AcQ treatment was suppressed by the antioxidant N-acetylcysteine (NAC). These results provide that AcQ might play an important role in human lung inflammatory diseases as an air pollutant.