Environmental polycyclic aromatic hydrocarbons (PAHs) enhance allergic inflammation by acting on human basophils.

Abstract:
Diesel exhaust particles (DEPs) have been implicated in the worldwide increased incidence of allergic airway diseases over the past century. There is growing evidence that DEP-associated polycyclic aromatic hydrocarbons (PAHs) participate in the development and maintenance of immunoglobulin (Ig) E-mediated allergic diseases. To address this issue we investigated the impact of U.S. Environmental Protection Agency (EPA) priority PAHs as well as of PAH-containing airborne extracts on antigen-induced CD63 upregulation and mediator release from human basophils. Whole blood samples from birch pollen allergic and control subjects were incubated in the presence of organic extracts of urban aerosol (AERex) or EPA-PAH standard with or without rBet v 1. Basophils were analyzed for CD63 expression as a measure of basophil activation by using multiparameter flow cytometry. In addition, purified basophils from birch pollen allergic donors were incubated for 2 h in the presence of 1 μM benzo[a]pyrene (BaP) or phenanthrene (Phe) and then stimulated with rBet v 1 for 45 min. Supernatants were assayed for histamine, interleukin (IL)-4, and IL-8 by means of enzyme-linked immunosorbent assay (ELISA). Basophils exposed in vitro simultaneously to AERex or EPA-PAH
standard and rBet v 1 expressed CD63 significantly more than with antigen alone. PAHs synergized 
with rBet v 1 dose dependently, but did not activate basophils from nonallergic donors. BaP and Phe 
significantly enhanced cytokine secretion (IL-4, IL-8) and histamine release from purified basophils 
without antigen added, and secretion was not further enhanced by rBet v 1 stimulation. In conclusion, 
PAHs from roadside emissions can directly activate sensitized basophils to cytokine secretion and 
drive proallergic processes through enhanced Fcepsilon RI-coupled mediator release from human 
basophils.

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