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# Air Pollution, Respiratory Infections and Innate Immunity

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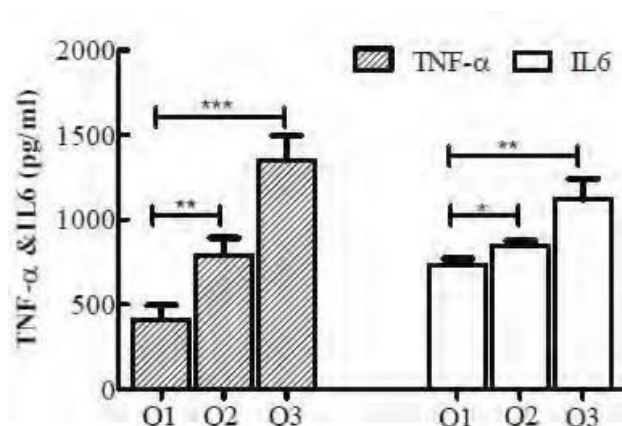
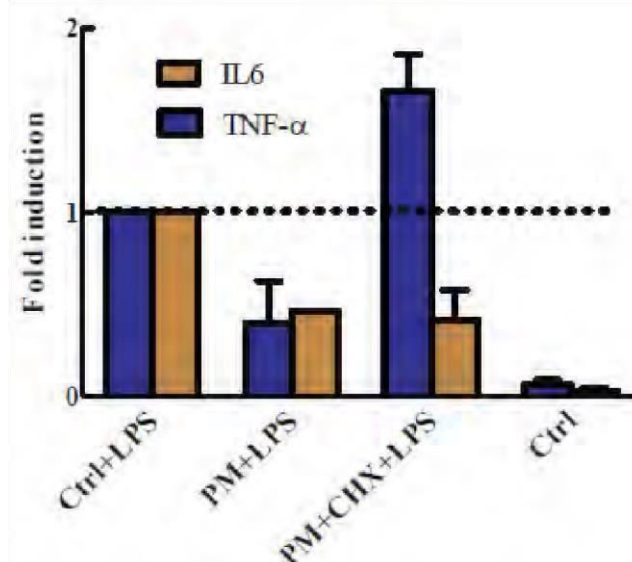
**ABSTRACT :** According to a World health organization report, acute lower respiratory infections associated with ambient air pollution exposure causes significant mortality among children under five years of age. Similarly, exposure to indoor biomass fuel burning smoke causes respiratory diseases, especially respiratory infections among children. Several studies in India have identified *S. pneumoniae* is the major cause of respiratory infections and is followed by *H. Influenzae* type b and *Staphylococcus aureus*. Innate immune defense is important during initial stages of bacterial infection in the lung. Signaling molecules such as  $TNF\alpha$  and IL6 produced by macrophages plays an important role in host immune response. Our experiments have shown that human macrophages exposed to air pollution particles are unable to produce cytokines *de novo* and are anergic to subsequent endotoxin or bacterial challenge. In summary, air pollution exposure induces high basal inflammation and inhibits a robust response of macrophage or monocyte to respiratory pathogens.

**KEYWORDS:** Air pollution, respiratory infections, inflammatory cytokines

## I. INTRODUCTION

According to a World health organization<sup>1</sup> report, acute lower respiratory infections (ALRI) associated with ambient air pollution exposure causes significant mortality among children under five years of age. Similarly, exposure to indoor biomass fuel burning smoke causes respiratory diseases, especially respiratory infections among children. Lower respiratory tract infection is one of the leading causes of death among children under five years of age in India<sup>2</sup>. Several studies in India have identified *S. pneumoniae* is the major cause of respiratory infections and is followed by *H. Influenzae* type b and *Staphylococcus aureus*<sup>3</sup>. Viral isolation from respiratory samples is a difficult process compared to isolation of bacteria and this lead to underestimation of viral contribution to respiratory infection. Viral infection was identified in 49% of pneumonia cases in children.<sup>4</sup> In many studies, respiratory syncytial virus is identified as the most common organism followed by influenza viruses, parainfluenza viruses and adenovirus<sup>4,5</sup>.

Innate immune defense is important during initial stages of bacterial infection in the lung<sup>6,7</sup>. Alveolar macrophages and recruited monocytes are the dominant immune cells during the initial stages of bacterial infection. Signaling molecules such as  $TNF\alpha$  and IL6 produced by macrophages plays an important role in host immune response. Inhibition of  $TNF\alpha$  by vaccination promotes growth and survival of pneumococci<sup>8</sup>. When the bacterial count increases in the host tissues, macrophage and monocytes must send a signal ( $TNF\alpha$  and IL6) of sufficient clarity to overcome the normal quiescent tissue homeostasis. However, high basal expression of inflammatory mediators due to air pollution reduces the clarity and effectiveness (Figure 1) of the macrophage and monocytes signaling. In Figure 1, THP1 cells were differentiated with PMA and were stimulated with air pollution particles (PM) for 5hrs. The supernatant was separated and  $TNF-\alpha$  and IL6 were measured with ELISA. Endotoxin was measured using kinetic LAL assay. Quantiles, q1, q2 and q3 represents endotoxin levels in lowest 1/3- quantile ( $< 26.75 \text{ Eu/m}^3$ ), endotoxin levels in middle 1/3 (between 26.75 and  $47.67 \text{ Eu/m}^3$ ) and endotoxin levels in last 1/3 ( $> 47.67 \text{ Eu/m}^3$ ). We have shown that human macrophages exposed to PM are unable to produce cytokines *de novo* and are anergic to subsequent endotoxin or bacterial challenge (Figure 2). In Figure 2, monocytes were stimulated with PM extracts for 24hrs. Cells were washed and rested for 2hrs. The cells were again stimulated with LPS (Sigma, India. 10ng/ml) alone or with cyclohexamide (Himedia, India) for 24hrs. Total RNA were isolated from cells and IL6 and  $TNF-\alpha$  were estimated using RTPCR (Life technology, India). However, interferon- $\gamma$  treatment reverses the anergic response of monocytes. We have also studied the expression of host genes associated with viral entry and fusion in lung epithelial cells. Our results show that PM exposure enhances expression of host viral entry genes. In summary, air pollution exposure induces high basal inflammation and inhibits a robust response<sup>9</sup> of macrophage or monocyte to respiratory pathogens.

FIGURE 1. Airborne endotoxin levels influence TNF- $\alpha$  and IL6 production from differentiated THP-1 cells.FIGURE 2. Cycloheximide treatment partially inhibits PM induced hypo-response (TNF- $\alpha$ ) in peripheral monocytes.

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