

TABLE VI-5 STUDIES OF HUMAN RESPIRATORY AND IMMUNOLOGICAL EFFECTS, 2000-2002—Continued

Authors, year	Description	Key results
Svartengren <i>et al.</i> , 2000 .....	Twenty nonsmoking subjects with mild allergic asthma were exposed for 30 minutes to high and low levels of engine exhaust air pollution on two separate occasions at least four weeks apart. Respiratory symptoms and pulmonary function were measured immediately before, during and after both exposure periods. Four hours after each exposure, the test subjects were challenged with a low dose of inhaled allergen. Lung function and asthmatic reactions were monitored for several hours after exposure.	Subjects with PM <sub>2.5</sub> exposure 100 µg/m <sup>3</sup> exhibited slightly increased asthmatic responses. Associations with adverse outcome variables were weaker for particulates than for NO <sub>2</sub> .

TABLE VI-6.—REVIEW ARTICLES ON RESPIRATORY AND IMMUNOLOGICAL EFFECTS, 1999-2002

Authors, Year	Description	Key results
Gavett and Koren, 2001 .....	Summarizes results of EPA studies done to determine whether PM can enhance allergic sensitization or exacerbate existing asthma or asthma-like responses in humans and animal models.	Studies indicate that PM enhances allergic sensitization in animal models of allergy and exacerbate inflammation and airway hyper-responsiveness in asthmatics and animal models of asthma.
Pandya <i>et al.</i> 2002 .....	Reviews human and animal research relevant to question of whether DPM is associated with asthma.	Evidence indicates that DPM is associated with the inflammatory and immune responses involved in asthma, but DPM appears to have a far greater impact as an adjuvant with allergens than alone. DPM appears to augment IgE, trigger eosinophil degranulation, and stimulate release of numerous cytokines and chemokines. DPM may also promote the cytotoxic effects of free radicals in the airways.
Patton and Lopez, 2002 .....	Review of evidence and mechanisms for the role of air pollutants in allergic airway diseases.	Evidence suggests that air pollutants (including DPM) "affect allergic response by different mechanisms. Pollutants may increase total IgE levels and potentiate the initial sensitization to allergens and the IgE response to a subsequent allergen exposure. Pollutants also may act by increasing allergic airway inflammation and by directly stimulating airway inflammation. In addition, it is well known that pollutants can be direct irritants of the airways, increasing symptoms in patients with allergic syndromes."
Peden, 2002 .....	Review of "studies that exemplify the impact of ozone, particulates, and toxic components of particulates on asthma."	DPM "may play a significant role not only in asthma exacerbation but also in T <sub>H</sub> 2 inflammation via the actions of polyaromatic hydrocarbons on B lymphocytes." "PM in which the active agents are biologically active metal ions and organic residues may have significant effects on asthma, especially modulating immune function, as demonstrated by the role of polyaromatic hydrocarbons from diesel exhaust in IgE production."
Sydbom <i>et al.</i> 2001 .....	Review of scientific literature on health effects of diesel exhaust, especially the DPM components.	The epidemiological support for particle effects on asthma and respiratory health is very evident; and respiratory, immunological, and systemic effects of DPM have been documented in a wide variety of experimental studies. Acute effects of DPM exposure include irritation of the nose and eyes, lung function changes, and airway inflammation. Exposure studies in healthy humans have documented a number of profound inflammatory changes in the airways, notably, before changes in pulmonary function can be detected. Such effects may be even more detrimental in subjects with compromised pulmonary function. Ultrafine particles are currently suspected of being the most aggressive particulate component of diesel exhaust.