

TABLE VI-4.—COMPLIANCE WITH 400 $\mu\text{g}/\text{m}^3$ TC LIMIT AND/OR PROPOSED 308 $\mu\text{g}/\text{m}^3$ EC LIMIT.
[Numbers in parentheses are percentages.]

EC > 308 $\mu\text{g}/\text{m}^3$	TC > 400 $\mu\text{g}/\text{m}^3$		Total
	No	Yes	
no	239 (66.8)	7 (2.0)	246 (68.7)
yes	6 (1.7)	106 (29.6)	112 (31.3)
Total	245 (68.4)	113 (31.6)	358 (100.0)

C. Health Effects Literature Update

We have identified additional scientific literature pertaining to health

effects of fine particulates in general and DPM in particular published subsequent to the January 19, 2001 final rule.

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

Authors, year	Description	Key results
Frew <i>et al.</i> , 2001	25 healthy subjects and 15 subjects with mild asthma were exposed to diesel exhaust (108 $\mu\text{g}/\text{m}^3$) or filtered air for 2 hr, with intermittent exercise. Lung function was assessed using a computerized whole body plethysmograph. Airway responses were sampled by bronchial wash (BW), bronchoalveolar lavage (BAL), and mucosal biopsies 6 hr. after ceasing exposures.	Both the asthmatic and healthy subjects developed increased airway resistance after exposure to diesel emissions, but airway inflammatory responses were different for the 2 groups. The healthy subjects showed statistically significant BW neutrophilia and BAL lymphocytosis 6 hr after exposure. The neutrophilic response of the healthy subjects was less intense than that seen in a previous study using a DPM concentration of 300 $\mu\text{g}/\text{m}^3$.
Fusco <i>et al.</i> , 2001	Analysis of daily hospital admissions for acute respiratory infections, COPD, asthma, and total respiratory conditions in Rome, Italy.	Respiratory admissions among adults were significantly correlated with CO and NO ₂ levels, but not with suspended particles. The authors noted that since CO and NO ₂ are good indicators of combustion products in vehicular exhaust, the detected effects may be due to unmeasured fine and ultrafine particles.
Holgate <i>et al.</i> , 2002	25 healthy and 15 asthmatic subjects were exposed for 2 hours to 100 $\mu\text{g}/\text{m}^3$ of DPM and to filtered air on separate days. Another 30 healthy subjects were exposed for 2 hours to DPM concentrations ranging from 25 to 311 $\mu\text{g}/\text{m}^3$ and compared to 12 different healthy subjects exposed to filtered air. Exposure effects were assessed using lung function tests and biochemical tests of bronchial tissue samples.	Healthy and asthmatic subjects exhibited evidence of bronchoconstriction immediately after exposure. Biochemical tests of inflammation yielded mixed results but showed small inflammatory changes in healthy subjects after DPM inhalation.
Oliver <i>et al.</i> , 2001	Pulmonary function tests and questionnaire data were obtained for 359 "heavy and highway" (HH) construction workers. Intensity of DPM exposure was estimated according to job classification. Duration of exposure was estimated based on length of union membership.	After adjusting for smoking and some other potential confounders, HH workers showed elevated risk of asthma. One subgroup (tunnel workers) also showed elevated risk of both undiagnosed asthma and chronic bronchitis, compared to other HH workers. Respiratory symptoms appeared to decline with exposure duration as measured by length of union membership. The authors interpreted this as suggesting that HH workers tend to leave their trade when they experience adverse respiratory symptoms.
Salvi <i>et al.</i> , 2000	15 healthy nonsmoking volunteers were exposed to 300 $\mu\text{g}/\text{m}^3$ DPM and clean air for one hour at least three weeks apart. Biochemical analyses were performed on bronchial tissue and bronchial wash cells obtained six hours after each exposure.	Diesel exhaust exposure enhanced gene transcription of IL-8 in the bronchial tissue and airway cells and increased IL-8 and GRO- α protein expression in the bronchial epithelium. This was accompanied by a trend toward increased IL-5 mRNA gene transcripts in the bronchial tissue. Study showed effects on chemokine and cytokine production in the lower airways of health adults. These substances attract and activate leukocytes. They are associated with the pathophysiology of asthma and allergic rhinitis.