

TABLE VI-8.—STUDIES ON TOXICOLOGICAL EFFECTS OF DPM EXPOSURE, 2000–2002—Continued

| Authors, Year | Description | Key results | Agent(s) of toxicity |
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| Fujimaki <i>et al.</i> , 2001 | In Vitro: assessment of cytokine production, spleen cells used. In Vivo: assessment of cytokine production profile following IP sensitization to OA and subsequent exposure to 1.0 mg/mg ³ DE for 12 hr/day, 7 days/week over 4 weeks, mouse inhalation model used. | Adverse effects of DE on cytokine and antibody production by creating an imbalance of helper T-cell functions. | DE generated from diesel engine DPM, CO ₂ , SO ₂ NO/NO ₂ /NO _x measured. |
| Gilmour <i>et al.</i> , 2001 | In Vivo: assessment of infectivity and allergenicity following exposure to woodsmoke, oil furnace emissions, or residual oil fly ash, mouse inhalation model used, IT instillation used in rats. | Exposure to woodsmoke increased susceptibility to and severity of streptococcal infection, exposure to residual oil fly ash increased pulmonary hypersensitivity reactions. | Woodsmoke, oil furnace emissions, and residual oil fly ash (ROFA) used |
| Hsiao <i>et al.</i> , 2000 | In Vitro: assessment of cytotoxic effects (cell proliferation, DNA damage) of PM _{2.5} (fine PM) and PM _{2.5} –10 (coarse PM), rat embryo fibroblast cells used. | Seasonal variations in PM, in their solubility, and in their ability to produce cytotoxicity. Long-term exposure to non-killing doses of PM may lead to accumulation of DNA lesions. | PM collected Hong Kong area and solvent-extractable organic compounds used. |
| Kuljucka-Rabb <i>et al.</i> , 2001 | In Vitro: assessment of adduct formation following exposure to DPM, DPM extracts, benzo[a]pyrene, or 5-methylchrysene, mammary carcinoma cells used. | Temporal and dose-dependent DNA adduct formation by PAHs. Carcinogenic PAHs from diesel extracts lead to stable DNA adduct formation. | Some DPM purchased from NIST, some DPM collected on filters from diesel vehicle, and solvent-extractable organic compounds used. |
| Moyer <i>et al.</i> , 2002 | In Vivo: 2-phase retrospective study, review of NTP data from 90-day and 2-yr exposures to particulates, use of mouse inhalation model. | Induction and/or exacerbation of arteritis following chronic exposure (beyond 90-day) to particulates. | Indium phosphide, cobalt sulfate heptahydrate, vanadium pentoxide, gallium arsenide, nickel oxide, nickel subsulfide, nickel sulfate hexahydrate, talc, molybdenum trioxide used. |
| Saito <i>et al.</i> , 2002 | In Vivo: assessment of cytokine expression following exposure to DE (100 µg/m ³ or 3 mg/m ³ DPM) for 7-hrs/day × 5 days/wk × 4 wks, mouse inhalation model used.. | DE alters immunological responses in the lung and may increase susceptibility to pathogens, low-dose DE may induce allergic/asthmatic reactions. | DE generated from diesel engine DPM, CO, SO ₂ , and NO ₂ measured. |
| Sato <i>et al.</i> , 2000 | In Vivo: assessment of mutant frequency and mutation spectra in lung following 4-wk exposure to 1 or 6 mg/m ³ DE, transgenic rat inhalation model used. | DE produced lesions in DNA and was mutagenic in rat lung. | DE generated from light-duty diesel engine Concentration of suspended particulate matter (SPM) measured, 11 PAHs and nitrated PAHs identified and quantitated in SPM. |
| Van Zijverden <i>et al.</i> , 2000 | In Vivo: assessment of immunomodulating capacity of DPM, carbon black, and silica particles, mouse model used (sc injection into hind footpad). | DPM skew immune response toward T helper 2 (Th2) side, and may facilitate initiation of allergy. | DPM, carbon black particles (CBP) and silica particles (SIP) used. DPM donated by Nijmegen University, CBP and SIP purchased from BrunswichChemie and Sigma Chemical Co., respectively. |
| Vincent <i>et al.</i> , 2001 | In Vivo: assessment of cardiovascular effects following 4-hr exposure to 4.2 mg/m ³ diesel soot, 4.6 mg/m ³ carbon black, or 48 mg/m ³ ambient urban particulates, rat inhalation model used. | Increases in endothelin -1 and -3 (two vasoregulators) following ambient urban particulates and diesel soot exposure. Small increases in blood pressure following exposure to ambient urban particulates. | Diesel soot, carbon black and urban air particulates used. Diesel soot purchased from NIST, carbon black donated by University of California, urban air particulates collected in Ottawa. |
| Walters <i>et al.</i> , 2001 | In Vivo: assessment of airway reactivity/responsiveness, and BAL cells and BAL cytokines following exposure to 0.5 mg/mouse aspirated DPM, ambient PM, or coal fly ash. | Dose and time-dependent changes in airway responsiveness and inflammation following exposure to PM. Increase in BAL cellularity following exposure to DPM, but airway reactivity/ unchanged. | DPM, PM, and coal fly ash used. DPM purchased from NIST, PM collected in Baltimore, and coal fly ash obtained from Baltimore power plant. |