The Synergistic Effects of World Trade Center Dust and Chronic Intermittent Hypoxia on Pulmonary Surfactant

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Exposure to dust created by the collapse of the world trade center (WTC dust) resulted in a number of pulmonary complications. There is a high prevalence of obstructive sleep apnea (OSA) within this population as well. It is unknown how these two insults interact in terms of lung function. In this study, we used WTC dust installation and chronic intermittent hypoxia (CIH), a model of OSA, to examine the effects on surfactant production. Surfactant regulates surface tension and immune function in the lung lining. It consists of phospholipids and surfactant proteins (SPs). Here, we examined phospholipid and SP-D levels in response to CIH (for 5, 14, & 28d) and WTC dust. CIH was simulated by cycling the inhaled gas between room air and a FiO2 of 5% 20 times per hour for 8 hours a day. WTC dust was intratracheally administered in saline. Western blot analysis was conducted on bronchoalveolar lavage (BAL) fluid to test for SP-D. Phospholipid within the BAL samples was also measured. CIH at 5, 14, & 28d resulted in an increase in phospholipid, irrespective of dust exposure. This increase was greatest at 5d. WTC in the absence of CIH produced a significant loss of phospholipid. Increased SP-D production was seen at 5 and 14d but not at 28d, indicating a greater acute than chronic effect. These results show an increase in surfactant production in response to CIH and immune function with WTC dust. Supported by the NIH R25ES020721 and U01OH012072 Grants, and the ASPET SURF Program.