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Lung disease in cement factory workers: Implications of metals in cement dust

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Introduction: Exposure to cement dust is one of the most common occupational hazards. It has been associated with the development of laryngeal cancer and other metal related diseases. The mechanism of injury to lung cells (alveolar macrophages and type II epithelial cell) and disease development by this particulate is still unclear.

Objectives: We evaluated the immunotoxicity of two cement dust samples (CDN and CDU) and clinker (CN) using alveolar macrophages and type II epithelial cell relative to their metal contents.

Methods: Metals (chromium, copper, lead, manganese, nickel, cadmium and mercury) were quantified using graphite furnace atomic absorption spectrophotometry, while hexavalent chromium (Cr (VI)) was determined by colorimetric method. Endocytosis of particles was assessed using transmission electron microscope. Additionally, apoptosis (annexin-V-PI), intracellular reactive oxygen species (iROS) generation and reduced glutathione (GSH) were determined using flow cytometry. Tumor necrosis factor-α (TNF-α), interleukin-1β (IL-1β) and macrophage inflammatory protein-2 (MIP-2) secretion from NR8383 were evaluated by ELISA technique.

Results: Our results indicated that Cu, Ni and Mg were significantly higher in CDU relative to CDN. Both total Cr and Cr (VI) were also higher in CDU than in CDN. Cadmium was higher in both CDN and CN. Mercury was more in both CDN and CN, while lead (Pb) was only significantly higher in CN. Alveolar epithelial cells internalised clinker predominantly at the membrane bound vacuoles. The CDU induced more apoptosis, intracellular ROS generation (22% higher) and reduced GSH compared with control, which may be related to the significant Cr (VI) level in CDU. Increase in IL-1β and TNF-α secretion were consistent in both CDN and CDU, while MIP-2 was not significantly increased in cells exposed to both CDU and CDN but significant in cells exposed to clinker.

Conclusion: The data suggest that the high metals and Cr (VI) concentrations may be contributory to the pathologic basis of cement dust toxicity. Endocytosis of cement dust particulates, oxidative stress induced-apoptosis and induction of pro-inflammatory cytokines may be the key mechanisms of cement dust immunotoxicity in lung cells. This study revealed that cement dust exposure is a public health threat to both cement factory workers and people located near such factories.

Biography

Anthony O Okorodudu has been with the University of Texas System (UTMB) since 1989. He is a Distinguished Professor of Pathology and Director for services/programs. He has obtained his Doctorate degree in Pathology (University of Medicine and Dentistry of New Jersey) following an undergraduate degree from Rutgers University, New Jersey. He also holds an MS degree in Management, Computing and Systems (Houston Baptist University, Texas), MBA degree (Rice University, Texas), and completed his Fellowship training in Clinical Chemistry at Hartford Hospital in Hartford, CT. He is a Diplomate of the American Board of Clinical Chemistry and a Fellow of National Academy of Clinical Biochemistry.

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