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Editorial

Silicosis-Associated Tuberculosis

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Experimental and epidemiological studies have demonstrated that silica is not only a cause of chronic inflammatory-interstitial lung diseases, but is also a potential carcinogen or lung cancer, particularly silica in the forms of cristobalite and quartz. Workers exposed to silica dust have increased morbidity and mortality from pulmonary tuberculosis, evidenced by several epidemiological and clinical studies. Several studies in China demonstrated that several cytokines, such as tumor necrosis factor-alpha, interferon-gamma, interleukin-10, and transforming growth factor-beta express changes in individuals with silicotuberculosis. These cytokine-associated genes express altering cytokine production. Other diseases associated with silica exposure include chronic obstructive pulmonary disease (COPD), pulmonary fibronodular disease, non-tuberculous mycobacterial (NTM) disease, other pneumoconiosis (alveolar lipoproteinosis, progressive massive fibrosis (PMF)), mineral dust airways disease (MDAD), and renal and autoimmune diseases (scleroderma, systemic lupus erythromatosis, rheumatoid arthritis) which had been reviewed by the National Institute of Safety and Health (NIOSH) and the American Thoracic Society (ATS). A large number of industries generate respirable crystalline silica, such as ceramics, glass manufacture, construction, foundry, stone-working, monumental masonry, furnace masonry, abrasive blasting with sand (sandblasting) or siliceous material, craft work, jewellery, diatomaceous earth, and drilling or crushing or moving sand or stones or rocks. Frequent exposure to sand storms is a source of non-occupational exposure. Silica-associated tuberculosis (silicotuberculosis) is of particular concern in the low-income countries. This concern contributed to the establishment of a Global Elimination of Silicosis Campaign by the International Labour Organization (ILO) and the World Health Organization (WHO).

The pathophysiology of silicotuberculosis has not been well understood. Both pulmonary and extra-pulmonary tuberculosis risk in the individuals with advanced simple silicosis in high background tuberculosis settings can be up to three-fold higher than those in the same workforce without silicosis, and the tuberculosis risk increases with the severity of silicosis. Greaves indicated that the risk of silicosis following a lifetime of respirable crystalline silica exposure at 0.05 mg/m^3 is most likely to be 20 - 40%, whereas we widely use current standards of exposure of 0.05 - 0.1 mg/m^3. The American Conference of Government Industrial Hygienists (ACGIH) has recommended a threshold limit value (TLV) of 0.025 mg/m^3, even this low level may not be protective against silicotuberculosis.

In conclusion, achieving legislated crystalline-silica exposure levels is challenging, particularly in low-income countries and in small enterprises.

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