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SILICOSIS- AN OCCUPATIONAL HAZARD

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ABSTRACT

Silicosis is a form of occupational lung disease is caused by the inhalation of crystalline silica (silicon dioxide, SiO2) in various forms. Exposure to silica occurs at workplaces in factories like quartz crushing facilities (silica flour milling), agate, ceramic, slate pencil, glass, stone quarries and mines, etc, This inflammatory and fibrotic events involved in cell-mediated, and humoral, immune responses also produces silicosis. Public concern regarding non-occupational or ambient exposure to crystalline silica has emerged making it important to gather information available on non-soccupational exposures to silica dust and non-occupational silicosis. The concept that interactions between silica and pulmonary macrophages are the events in the pathogenesis of silicosis. The silica probably exerts its effects on the macrophages that ingest it by altering their function while they are alive, rather than merely by disrupting them. The macrophage appears to be stimulated to secrete mediator substances, such as interleukin-1 (IL-1), which alter the function and behavior of other cells. Lymphocytes and macrophages appear in close proximity to one another in developing silicotic nodules, and increased proportions of lymphocytes are found in bronchoalveolar lavage specimens from animals and humans with silica dust exposure. The macrophage has been implicated as a major cause for the fibrosis that accompanies silicosis. The products of activated T-lymphocytes, particularly interferon-gamma (IF-g), are potent stimulators of secretion from macrophages of a fibroblast growth competence factor, macrophage-derived growth factor (MDGF). IL-1 may have an additional stimulatory effect on fibroblasts. Silicosis provides a model of chronic diffuse interstitial immunologic and fibrotic lung disease in which the cause is known, can be applied in defined doses, and tracked in the lung throughout the course of the disease.

KEYWORDS: Silicosis, Quartz, Silica, Macrophages, Occupational Exposure, Lymphocytes, Pulmonary Fibrosis, Interleukin