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A possible connection between furnace dust exposure, plasma fibrinogen levels and cardiovascular disease

In a very well-written and informative study Hobbesland and his co-workers found an increased mortality from sudden death among ferromanganese/silicomanganese furnace workers (1).

In 1955 an interesting observation was made by Rodier (2) when studying manganese-exposed Moroccan miners. The clinical signs of manganese pneumonia were first those of acute alveolar inflammation. Dyspnea was marked, the respiration was shallow, and there was gasping. The illness was altered after the 3rd day from a frank case of pneumonia to less well-defined localization and discrete pleural involvement. Death from heart failure was possible between the 5th and the 10th day. It could also occur suddenly for patients regarded as cured. Thus it seems as if sudden death occurred in some of the patients regarded as cured.

During the last decade fibrinogen has emerged as an important risk factor for ischemic heart disease (3, 4). Increased concentrations of plasma fibrinogen have also been associated with myocardial infarction and sudden death among patients with angina pectoris (5).

Three deaths due to pneumonia occurred during active occupational exposure among Norwegian ferromanganese/silicomanganese furnace workers (6). Manganese pneumonia is an inflammatory disease and was associated with a marked rise in erythrocyte sedimentation rate among the Moroccan miners (2). The sedimentation rate can be influenced by several serum proteins, for example, fibrinogen, haptoglobin, immunoglobulins, and ceruloplasmin (7). In other words plasma fibrinogen may be increased in this condition.

In a Belgian factory producing manganese oxides and salts, the exposed workers more often had respiratory symptoms such as cough, sputum, and acute bronchitis than a reference group did. The median exposure of manganese airborne dust was 0.97 mg/m³. The exposed group also had an increased prevalence of chronic bronchitis, but the difference was not significant (8). It has recently been shown that persons with chronic bronchitis have an increased risk for coronary disease and coronary deaths (9).

The mentioned manganese-exposed Belgian workers had decreased lung function for several spirometric parameters, for example, forced vital capacity (FVC), forced expiratory volume in 1 second (FEV_{1.0}), and peak expiratory flow (PEF) (8). A relationship between decreased lung function (VC and FEV_{1.0}) and increased levels of fibrinogen was found in a study of 788 Swedish men (10). Several studies have observed a relationship between a decreased lung function

[expressed as VC (11), FEV_{1.0} (12), or PEF (13)] and ischemic heart disease.

A general hypothesis regarding inhaled particle exposure and the occurrence of IHD can be expressed in the following way. Long-term inhalation of particles retained in the lungs will create low-grade inflammation in association with an increase in plasma fibrinogen. The high levels of fibrinogen increase the likelihood for blood clotting and thereby the risk for myocardial infarction, ischemic heart disease, and possibly sudden death (14, 15). This hypothesis would be supported if furnace workers have a higher concentration of plasma fibrinogen than nonfurnace workers with control for other possible confounders (eg, smoking) (16).

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