Clinical research

Serum iron concentrations, lipid peroxidation and superoxide dismutase activity in Turkish iron miners

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Abstract

The effects of iron exposure, on serum iron, serum superoxide dismutase (SOD), a free radical scavenger, and plasma malondialdehyde (MDA), a lipid peroxidation parameter, were investigated in Turkish iron miners, office workers and healthy control subjects. Serum iron levels in both miner and office workers groups were higher than those of healthy controls ($p < 0.05$). There were higher mean values of plasma MDA levels in both iron miners and office workers compared to controls ($p < 0.05$). Serum SOD activity in the miner group was lower than that of controls ($p < 0.05$). These results suggested that elevated MDA levels in both miners and office workers were the result of an increased production and/or decreased catabolism of MDA in chronic iron exposure. These changes in MDA metabolism may be due to iron-induced lipid peroxidation in the blood and related body compartments.

Introduction

Iron overload is usually associated with increased serum iron levels and decreased iron binding capacity (Nichols & Bacon 1989). Generation of the reactive oxygen species (ROS) by iron-catalyzed Fenton reactions (Frenkel 1992) have been implicated in the pathogenesis of many diseases including cancer, atherosclerosis and ischemia/reperfusion injury and conditions of iron overload such as haemochromatosis (Dabbagh et al. 1994). Epidemiological studies indicated an excess of lung cancer deaths among iron ore miners and steel industry workers (Haguenoer et al. 1996). Under conditions of chronic iron overload, there is evidence that excess iron induces cellular injury and functional abnormalities, especially in hepatocytes by the process of lipid peroxidation of cell membrane and some intracellular organelles such as lysosomes, mitochondria and microsomes. Lipid peroxidation is a likely outcome of oxidative stress in biological systems, and its measurement is often used as a method of assessing the degree of oxidative damage (Rao et al. 1978; Dabbagh et al. 1994).

Bioavailable iron levels in coal mine may be responsible for the observed regional differences in the prevalence and severity of disease among coal workers. Zhang observed (Zhang & Huang 2002) that bioavailable iron, ferritin and lipid peroxidation were significantly higher in lung epithelial cells treated with various coals than in control cells. The findings of their study provide further evidence that metals, particularly iron, play important roles in coal dust-induced cellular damage and contributing to the regional differences in the prevalence of the disease (Zhang et al. 2002). In case of acute (Hallaway et al. 1989) or chronic overload in human body, iron can be a hazardous material because of its redox potential. The toxicity of iron arises from its ability to catalyze the formation of oxygen-derived free radical species that can interact with cellular membranes and cytoplasmic constituents (Balla et al. 1990; Chau et al. 1993). Such interactions, per se, can not only potentially modify the functional integrity of the cells and their membranes but also generate toxic products that further modify various cellular functions. Such oxidants usually proceed by a free radical chain mechanism.
and its initial event is the generation of free radicals which initiate the chain reaction. The transition metal, iron, is strongly implicated in the generation of free radicals both in vitro and in vivo. Although other occupational disease groups have been studied in regarding iron and other element exposures, there has been no detailed evaluation of mine workers and office workers together in the same study. An increased mortality from lung and stomach cancer was found in iron miners in the previous studies (Edling 1982; Pham et al. 1983; Rice-Evans & Baysal 1987; Darby et al. 1995). Bacon et al. (1983) demonstrated the occurrence of hepatic mitochondrial and microsomal lipid peroxidation in vivo in rats with chronic dietary iron overload. In the literature, we did not encounter any research on lipid peroxidation and antioxidant status of iron miners and office workers, except Odinaev (1992) who found increased lipid peroxidation and decreased antioxidant defense in miners.

In this study, we investigated the effects of iron exposure from mining on an endogenous antioxidant enzyme superoxide dismutase (SOD), and the end product of lipid peroxidation malondialdehyde (MDA) in the serum humans. Occupational risks involve that increased lipid peroxidation by the hypothesis of redox activity on the surface of dust particles might be different for miners and office workers in a mining area yet this does not seem to have been investigated in the literature.

Materials and methods

Subjects and samples

The study consisted of 32 iron miners aged from 31 to 55 (mean ± SD; 42.2 ± 5.9) and 26 office workers aged from 30 to 52 (39.6 ± 6.4) working in an iron mining company at Divrigi, Sivas, Turkey for at least 10 years. They were all male. A questionnaire was circulated among 120 miners: 55 were invited to take part until the study end. Thirty-nine consented, however, seven miners did not complete the study. Iron miners worked in relays in the mine excavation process. Office workers worked at the same mine in surface buildings from 8 am to 5 pm. Thirty-two office workers were invited to take part in the study but six of them were excluded from the study because of chronic diseases. None of the office workers were previously miners given desk jobs. Eighteen healthy males (40.5 ± 7.1) living in Divrigi, Sivas formed the control group. None of the subjects in the miner group and healthy controls was a smoker. This was also a selection criteria for miner and healthy groups. On the other hand, the office workers group contained five smokers. To measure serum SOD activity and iron level, fasting blood samples were obtained from subjects’ cubital vein without any anticoagulant in the morning. For MDA measurement, heparinized tubes were used. After plasma was obtained, glutathione and EDTA at the concentrations of 0.65 and 1.34 mmol L⁻¹, respectively, were added to inhibit production MDA by auto-oxidation post sampling.

Analyses

MDA was estimated according to the method of Wasowicz et al. (1993) which is based on the coupling of MDA with thiobarbituric acid (TBA) at 95 °C with minor changes. Following the reaction, fluorescence intensity was measured in the n-butanol phase with a fluorescence spectrophotometer (Hitachi Model F-4010, Japan), excitation at 525 nm, emission at 525 nm, by comparing with a standard solution of 1,1,3,3-tetramethoxypropane. Results were expressed as µmol per liter plasma. SOD activity was determined according to the method of Sun et al. (1988), based on the inhibition of nitroblue tetrazolium (NBT) reduction with the xanthine–xanthine oxidase system as a superoxide generator. One SOD unit was defined as the enzyme amount causing 50% inhibition in the NBT reduction rate, and SOD activity was also expressed as IU mL⁻¹ plasma. All samples were assayed in duplicate. Serum iron concentrations were determined by an autoanalyser (Kodak Ektachem 500) using the method of colorimetric slide and commercial kits (Johnson and Johnson Company), and results were expressed as µg dL⁻¹.

Statistical analysis

Data were analyzed by using SPSS® for Windows computing program. Results were expressed as mean ± SD. One-way ANOVA test were used to detect significant differences initially. At the second step, Tukey’s B posthoc test was used to detect differences between groups. Bivariate comparisons were examined using Pearson’s rank correlation coefficients (r). Differences were considered significant at \( p < 0.05 \).