P24. The effect of lead exposure on thiol disulfide homeostasis and lipid peroxidation in human tissue

Ceylan Bal\textsuperscript{1}, Murat Büyükşekerci\textsuperscript{2}, Utku Serkant\textsuperscript{3}, Murat Alişık\textsuperscript{1}, Meşide Gündüzöz\textsuperscript{4}, Ömer Hınç Yılmaz\textsuperscript{5}, Engin Tutkun\textsuperscript{6}, Özcan Erel\textsuperscript{1}

\textsuperscript{1}Department of Biochemistry, Yıldırım Beyazıt University, Ankara, Turkey
\textsuperscript{2}Department of Pharmacology, Occupational Diseases Hospital, Ankara, Turkey
\textsuperscript{3}Department of Biochemistry, Golbaşı Hasvak Hospital, Ankara, Turkey
\textsuperscript{4}Department of Family Medicine, Occupational Diseases Hospital, Ankara, Turkey
\textsuperscript{5}Department of Public Health, Yıldırım Beyazıt University, Ankara, Turkey
\textsuperscript{6}Department of Public Health, Bozok University, Yozgat, Turkey

Objective: Several molecular and cellular mechanisms are proposed for lead induced toxicity yet oxidative stress is the most prominent of them. In this study we evaluated the redox state in occupationally lead exposed workers by assessing the dynamic serum thiol-disulfide homeostasis using a novel method.

Methods: The study group consisted 46 male battery workers who admitted to our clinic for periodic medical examination and the control group was composed of 44 healthy male administrative workers. We determined the thiol-disulfide homeostasis parameters of subjects using a novel automated colourimetric assay. Catalase, ceruloplasmine and myeloperoxidase activities were measured as oxidative stress indicators and 8-isoprostane level as marker of oxidative tissue damage.

Findings: Median blood lead and 8-isoprostane levels of exposed group were significantly higher than control group (8.0(4.1-35.3) μg/dl vs. 1.7(0.30-3.60) μg/dl, p<0.001 and 4.02(1.30-9.27) ng/ml vs. 3.17(0.65-7.72) ng/ml, p<0.001, respectively). Among the thiol disulfide homeostasis parameters, disulfide level and disulfide/native thiol ratio were significantly higher in exposed group compared to control group. The mean ceruloplasmine, myeloperoxidase and catalase activities of Pb exposed group and control group did not differ significantly higher than (94.99±28.45 U/L vs. 88.90±20.66 U/L, p=0.189; and 147.74±60.21U/L vs. 139.91±51.74U/L, p=0.456 and 250(50-476.30) (kU/L) vs. 102.80(18.60-307.1) kU/L, p= 0.606, respectively).

Result: We conclude that the thiol-disulfide homeostasis in Pb exposed workers is disrupted in favor of disulfide which is indicates oxidative stress. Furthermore Pb induced oxidative stress contributes to elevation of 8-isoprostane.

Keywords: lead exposure, thiol disulfide homeostasis, lipid peroxidation