



## Effects of Occupational Inorganic Dust Exposure on Plasma Glutathione

**\*Gospodinka R. Prakova<sup>1</sup>, Pavlina L. Gidikova<sup>2</sup>, Gergana N. Sandeva<sup>2</sup>,  
Kamelia H. Haracherova<sup>3</sup>, Tanya T. Tacheva<sup>4</sup>, Tatyana I. Vlaykova<sup>4</sup>**

1. Department of Internal Medicine, Medical Faculty, Trakia University, Stara Zagora, Bulgaria
2. Department of Hygiene, Epidemiology, and Infectious Diseases, Medical Faculty, Trakia University, Stara Zagora, Bulgaria
3. Occupational Medical Service "Zagora Medical" Ltd, Stara Zagora, Bulgaria
4. Department of Chemistry and Biochemistry, Medical Faculty, Trakia University, Stara Zagora, Bulgaria

**\*Corresponding Author:** Email: prakova@hotmail.com

(Received 10 Nov 2019; accepted 27 Nov 2019)

### Dear Editor-in-Chief

“Glutathione (GSH) is an important antioxidant involved in reducing various lipid and other peroxides, thus protecting cells from oxidative damage” (1). In a study on 40 individuals exposed to coal dust, for both pneumoconiosis and non-pneumoconiotic respiratory disease, increased enzymatic antioxidants indicated reduced risk (1). The aim of this study was to assess plasma levels of total and reduced glutathione in workers exposed to inorganic dust.

The study was approved by the Trakia University's Ethics Committee, and written informed consent was obtained from all studied individuals. The study was performed in 2016 in Stara Zagora, Bulgaria. Forty workers from an iron casting factory occupationally exposed to inorganic dust (24 men and 16 women) and 44 non-exposed controls (29 men and 15 women) were examined. The mean age of the exposed was  $41.50 \pm 8.09$  years and of the control group  $47.89 \pm 11.61$  years.

Determination of mineral dust in the factory work environment showed that the inhalable dust fraction ( $12.1 \text{ mg/m}^3$ ) exceeded more than twice the limit value ( $5.0 \text{ mg/m}^3$ ). Total and reduced

glutathione were determined using Glutathione Colorimetric Detection Kit (Ray Biotech Inc., USA) in mg/ml. The obtained results for glutathione were statistically analyzed according to age, sex, duration and type of dust exposure, and smoking.

A trend for elevated total ( $0.46 \pm 0.19 \text{ mg/ml}$ ) and reduced ( $0.32 \pm 0.17 \text{ mg/ml}$ ) glutathione was observed in the group exposed to inorganic dust compared with the control group ( $0.44 \pm 0.02 \text{ mg/ml}$ ,  $P=0.301$  and  $0.31 \pm 0.14 \text{ mg/ml}$ ,  $P=0.993$  respectively). Total glutathione in exposed women ( $0.49 \pm 0.02 \text{ mg/ml}$ ) was significantly higher ( $P=0.01$ ) than in the control females ( $0.41 \pm 0.02 \text{ mg/ml}$ ). The lower level of total and reduced glutathione found in exposed men appears to be related to higher dust concentrations in the work environment and the resulting depletion of those individuals' compensatory capacity, which increases the occupational risk compared to women.

Similar findings were reported where rise in nonenzymatic antioxidants (i.e. vitamin E, GSH) was indicative of reduced health risk (1). Genetic predisposition may be the underlying cause of the



different glutathione levels and one of the reasons that only about 10% of the exposed individuals developed manifest farmer's lung (2). Changes in GSH occurring due to smoking are currently not sufficiently studied.

Our results for total and reduced glutathione in smokers and non-smokers of both genders showed significantly higher levels of total glutathione only in non-smoking women compared to the corresponding control group ( $0.52 \pm 0.083$  mg/ml vs.  $0.39 \pm 0.057$  mg/ml,  $P=0.013$ ). The oxidant burden in the lungs is further enhanced in smokers by the release of ROS from macrophages and neutrophils (3, 4). Smoking results in decreased antioxidant capacity in plasma associated with depleted protein thiol groups (5). GSH plays a critical role in maintaining epithelial membrane integrity and the protection against cigarette smoke or oxidant-mediated epithelial injury. Mice exposed to cigarette smoke initially lowered GSH levels by 50%, but within 2 hours GSH increased to 3 times above base levels. Smoking caused a potent GSH adaptive response, both systemic and locally in the lungs (6). Cell GSH and GSH blood concentrations are influenced by some habits but not by gender or age of healthy adults, whereas decreased glutathione plasma concentration does not correlate with any of these variables. These changes should be evaluated for a possible use of glutathione in prevention and treatment of clinical disorders (7).

In our study we found a significant negative correlation between total glutathione and length of service ( $r = -0.48$ ,  $P=0.02$ ) only in men exposed to inorganic dust, confirming the higher occupational risk of men exposed to inorganic dust, as well as the likelihood of a faster depletion of the body's antioxidant capacity than in exposed women. Serum GSH level can be considered a potential biological marker for workers exposed to silica containing inorganic dust (8). Exposure to inorganic dust affects antioxidant mechanisms in the body and is dependent on workers' gender and smoking habits.

Our results imply that further studies are needed to properly assess the changes in the antioxidant status of workers exposed to inorganic dust.

## Acknowledgements

This work was supported by Research Project No16/2014, Medical Faculty, Trakia University, Stara Zagora, Bulgaria.

## Conflict of interest

The authors declare that there is no conflict of interests.

## References

1. Schins RP, Keman S, Borm PJ (1997). Blood antioxidant status in coal dust induced respiratory disorders: a longitudinal evaluation of multiple biomarkers. *Biomarkers*, 2(1): 45-50.
2. Behr J, Degenkolb B, Beinert T, Krombach F, Vogelmeier C (2000). Pulmonary glutathione levels in acute episodes of farmer's lung. *Am J Respir Crit Care Med*, 161(6): 1968-71.
3. Hoidal RR, Fox RB, LeMarbe PA, Perri R, Repine JE (1981). Altered oxidative metabolic responses in vitro of alveolar macrophages from asymptomatic cigarette smokers. *Am Rev Respir Dis*. 123: 85-89.
4. Schaberg T, Haller H, Rau M, Kaiser D, Fassbender M, Lode H (1992). Superoxide anion release induced by platelet activating factor is increased in human alveolar macrophages from smokers. *Eur Respir J*. 5: 387-393.
5. Rahman I, MacNee W (1996). Role of oxidants/antioxidants in smoking-induced airways diseases. *Free Radic Biol Med*. 21: 669-681.
6. Gould NS, Min E, Gauthier S, Martin RJ, Day BJ (2011). Lung glutathione adaptive responses to cigarette smoke exposure. *Respir Res*, 12: 133.
7. Michelet F, Gueguen R, Leroy P, Wellman M, Nicolas A, Siest G (1995). Blood and plasma glutathione measured in healthy subjects by HPLC: relation to sex, aging, biological variables, and life habits. *Clin Chem*, 41(10): 1509-1517.
8. Mohammadi H, Dehghan SF, Alireza Tahamtan A, Golbabaie F (2018). Evaluation of potential biomarkers of exposure to crystalline silica: A case study in an insulator manufacturer. *Toxicol Ind Health*, 34(7): 491-498.