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EFFECTS OF PHENYLMERCURY ON HEMOGLOBIN AND REDOX STATE LEAD TO DESTABILIZATION IN HUMAN ERYTHROCYTES

Sheila O. Souza (PG),[1*] Marcos V. S. Sales (PG), [1] Maiara I.C. Queiroz (PG), [1] Ana F. S. Lima (PG),[2] Wallinson S. Dias (PG), [1] Laís F. A. M. Oliveira (PG), [3] Júlio C. S. Silva (PQ),[1] Eduardo J. Fonseca (Prof),[3] Letícia Bassi (Prof), [1] Alexandre U. Borbely (Prof), [2] Josué Carinhanha C. Santos (Prof), [1] Ana Catarina R. Leite (Prof) [1]

sheila.souza@iqb.ufal.br

[1]Institute of Chemistry and Biotechnology; [2]Institute of Biological and Health Sciences; [3]Institute of Physics, UFAL, Alagoas, Brazil

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ABSTRACT

Phenylmercury (PM) is an organomercurial compost used in fungicides and pesticides. Human blood comprises various cells, including erythrocytes (Ery), which contain hemoglobin, a protein vital for transporting oxygen to the body's tissues. Exposure to mercury species can induce oxidative stress and cause alterations in biological systems. This study explores the effects of PM exposure on human Ery, investigating their functionality, structural alterations, and impact on redox status. Human Ery from volunteers (*n* = 5, CAAE 02840318.2.0000.5013) were exposed to PM concentrations varying from 0.125 to 1.0 μM. The results showed a 34% reduction in oxygen uptake levels in Ery when exposed to PM 1 μM. The generation of global reactive oxygen species (EROs) increased by more than 100% compared to the control. The production of H_2O_2 increased by 11%, while the production of $^{\bullet}NO$ increased by 90%. Membrane fragility and structural alterations were observed in Ery exposed to PM, revealing damage to the cell membrane. Evaluating the activities of antioxidant enzymes, we found a decrease of about 25% in catalase (CAT) and 49% in glutathione peroxidase (GPx). On the other hand, the activity of superoxide dismutase (SOD) increased by 13%. Furthermore, when evaluating secondary markers of oxidative stress, we observed a reduction of 34% in the GSH/GSSG ratio and a reduction of 44% in the sulfhydryl groups. The levels of carbonylated proteins increased by 100%, but there was no significant alteration in lipid peroxidation. Furthermore, atomic force microscopy (AFM) revealed that exposure to PM caused significant morphological deformations in human cells, including a reduction in cell height and an increase in diameter. Through Tung's modulus, higher elasticity was observed in cells exposed to PM (1 μM). Investigations of the direct impact of PM on commercial hemoglobin, through Soret band analysis, reveal structural alterations in the heme group of hemoglobin. The electrophoresis assay also identified a delay in hemoglobin migration in the presence of PM. Molecular docking simulations indicate that PM can bind to the heme group and cysteine 93 of hemoglobin, probably altering its capacity to act as an oxygen carrier. Based on these results, we conclude that PM modifies Ery functionality, redox state, and morphology, directly affecting hemoglobin structure.

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