The influence of $HgCl_2$ on the rate of oxygen uptake and release by red blood cells

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Mercury compounds possess the expressed toxic action on any kind of living matter. Pathological action of mercury compounds is based primarily on its interaction with sulfhydric groups of proteins. It was shown that approximately half of mercury injected into a human organism is bounded by red blood cells. In this work the influence of mercuric chloride ($HgCl_2$) on the rate of oxygen uptake and release by human red blood cells was investigated. Kinetic measurements were carried out by stopped-flow technique with dual-wavelength photometric registration (dead time - 6 ms). The dual-wavelength mode was used to decrease the influence of lightscattering on kinetic curves. Registration of hemoglobin transition from desoxy- to oxy- form was carried out by measuring simultaneous absorbance changes at 560 and 577 nm. Kinetics of oxygen uptake by red cells was registered upon mixing of an anaerobic red cells suspension with the buffer containing oxygen. Kinetics of oxygen release was registered upon mixing an aerobic suspension of cells with a solution of a reducing agent sodium dithionite ($Na_2S_2O_4$).

Incubation of red blood cells (Hct 0,2% before mixing) with $HgCl_2$ causes essential decrease of the oxygen uptake and release rates. At concentration of 160 μM $HgCl_2$ was shown to cause the 2.7 fold decrease of oxygen uptake rate. At concentration of 200 μM $HgCl_2$ was shown to cause the 2.3 fold decrease of oxygen uptake rate in comparison with the control.

The effects of $HgCl_2$ can not be solely explained by change of red blood cell hemoglobin properties upon $HgCl_2$ binding, since it was shown, that mercuric chloride causes increase of hemoglobin's oxygen affinity and, hence, it should increase the rate of oxygen uptake and decrease the rate of oxygen release [1]. However $HgCl_2$ does not change microviscosity of red cell membranes and viscosity of cell cytoplasm significantly [2]. Thus, the effects of $HgCl_2$ can be due to action on membrane proteins [3, 4].

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