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Redox interactions of epinephrine with iron at physiological pH

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Epinephrine ((R)-4-(1-hydroxy-2-(methylamino)ethyl)-benzene-1,2-diol (Epi) is catecholamine that is released by the sympathetic nervous system and adrenal medulla. It is a physiologically important molecule that acts as a hormone, neurotransmitter, and medication with a broad range of effects [1-3]. Coordinate and redox interaction of Epi with iron affects the interactions with other molecules and its biological effects [4]. In this study, we reported details of redox interactions of Epi with Fe^{2+} at pH 7.4, which correspond to the pH value of human plasma Epi and Fe^{2+} form a complex that acts as a strong reducing agent. Cyclic voltammetry showed that the positions of E_{pa} and E_{pc} potentials were at approximately -480 and -1100 mV. This implies that Epi and Fe^{2+} build a complex with unique redox properties. $E_{1/2}$ was significantly lower compared to E_0' for $O_2/O_2^{\cdot-}$ (-350 mV). It is important to point out this because superoxide radical anion is produced via spontaneous Fe^{2+} reaction with O_2. In other words, Epi-Fe^{2+} complex should be capable of reducing transition metals in (patho)physiologicaly relevant complexes that are not susceptible to reduction by O_2. Our results confirmed that Epi-Fe^{2+} is capable of reducing the S-S group of glutathione disulfide. On the other hand, Epi acted in a catalyst-like fashion to promote Fe^{2+} oxidation by molecular oxygen, and to a facilitated formation of the Epi–Fe^{3+} complexes, at physiological pH. In addition, we examined the effects of epinepfrine and Epi/Fe^{3+} system on glioma cells. Epinephrine alone evokes changes in the membrane currents of glioma cells, but such effects were not observed for the complex with Fe^{3+}. This implies that Epi-Fe^{3+} might modulate neural activity of Epi in CNS.

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