

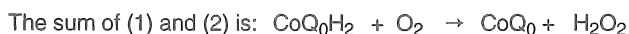
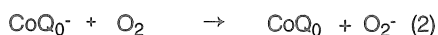
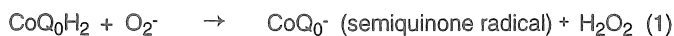
Effects of salts and minerals on SOD inhibition of ubiquinol oxidation

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Summary

Ubiquinone (CoQ₀ = 2,3-Dimethoxy-5-methyl-1,4-benzoquinone) and hydrogen peroxide (H₂O₂) were formed in the process of oxidation of ubiquinol (CoQ₀H₂ = 2,3-Dimethoxy-5-methyl-1,4-hydroquinone) in a phosphate buffer. Among eight mineral salts investigated, FeSO₄, MnSO₄ and CuSO₄ increased the ubiquinone formation. NaCl had no effect. Chelators such as DTPA (diethylenetriaminepentaacetic acid) decreased the amount of ubiquinone and H₂O₂. This suggests that trace amount of Fe ion in the buffer increased the ubiquinol oxidation. The amount of H₂O₂ was almost equal to that of CoQ₀, indicating that the H₂O₂ formation was coupled with the CoQ₀ formation. Copper-zinc superoxide dismutase (CuZn-SOD) and manganese superoxide dismutase (Mn-SOD), which accelerate the dismutation of superoxide ($2O_2^- + 2H^+ \rightarrow H_2O_2 + O_2$), inhibited both the CoQ₀ formation and the H₂O₂ formation in the presence of DTPA. We propose that CoQ₀H₂ oxidation occurs as a chain reaction with superoxide (O₂⁻) as the chain carrier and that SOD inhibits this reaction by lowering the superoxide concentration.



It is interesting that the active sites of SODs consist of the minerals which increased the ubiquinol oxidation. In animal cells, Mn-SOD is located in the mitochondria, and CuZn-SOD is located in the cytoplasm. Ubiquinols are also located in both the mitochondria and the cytoplasm. Similar inhibitory effects of Mn-SOD on the CoQ₀ formation and the H₂O₂ formation at the same enzymatic activity as in the case of CuZn-SOD imply that these enzymes have a common antioxidative role in different parts of the cells.