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Does Cu/Zn-superoxide dismutase exhibit a nondismutase activity?

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Cu/Zn-superoxide dismutase (SOD) catalyses the dismutation of superoxide anion radical to the nonradical molecules, oxygen and hydrogen peroxide. At the physiological conditions there are defense systems destroying H_2O_2 in the organism such as glutathione peroxidase and catalase. If the removal of H_2O_2 by catalase and peroxidases is insufficient, the product of the dismutation reaction, hydrogen peroxide can be turned into the substrate for SOD. During this nondismutase activity of Cu/Zn-SOD, copper can be liberated from the active centre of this enzyme. Liberated copper can be chelated with low molecular weight chelators and in this form it can be a substrate for the formation of prooxidant metabolites. In this case, especially when the activity of Cu/Zn-SOD is non-physiologically elevated, it is questionable what the real function of SOD is.

Down syndrome is known to have increased gene dose for Cu/Zn-SOD due to the trisomy of chromosome 21, where the gene for Cu/Zn-SOD is located. We confirmed an increased activity of Cu/Zn-SOD in leukocytes of persons with Down syndrome as compared with the control group. We determined a phenanthroline-detectable low molecular weight copper (chelated with low molecular weight chelators) (LMW-Cu) and we found that LMW-Cu was significantly increased as compared with the controls as well as there was a positive correlation between SOD activity and LMW-Cu in these subjects. On the base of these results we can assume that nondismutase activity of Cu/Zn-SOD can be involved in the potential oxidative stress in persons with Down syndrome.