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[Lab. of Pharmaceutics]

The nature of heterogeneous components of extracellular-superoxide dismutase purified from human umbilical cords.

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Extracellular-superoxide dismutase (EC-SOD) has been purified with a high yield from human umbilical cords. Purified native EC-SOD (n-EC-SOD C) showed a single band with enzymatic activity on polyacrylamide gel electrophoresis. It showed two bands with apparent molecular masses of 29.3 and 32.0 kDa on SDS-PAGE, while recombinant EC-SOD C (r-EC-SOD C) showed only one band with 32.0 kDa. The two components could be clearly separated from each other by C4 reverse-phase high-performance liquid chromatography (HPLC). The recovered peak corresponding to the lower molecular component of SDS-PAGE lacked a peptide composed of 11 amino acid residues in the carboxyterminal region, Lys-212 to Ala-222, and had lost the heparin affinity.

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Pedigree of serum extracellular-superoxide dismutase level.

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Serum extracellular-superoxide dismutase (EC-SOD) levels from healthy persons without any abnormality in a thorough physical and clinical examination are clearly divided into two groups: a lower concentration group (Group I, below 120 ng/ml) and a higher concentration group (Group II, above 400 ng/ml). This report describes family studies of serum EC-SOD and its heparin affinity. Five propositi and their parents were studied. Although the number of observations is limited, it was found that only one parent of all the propositi showed high EC-SOD levels. Family studies in this report are highly indicative of a genetic transmission of EC-SOD level.

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Extracellular superoxide dismutase level in chronic hemodialysis patients.

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We studied the levels of serum extracellular-superoxide dismutase (EC-SOD) in patients treated with hemodialysis (HD) and in conservatively managed patients with chronic renal failure (CRF). Results show that serum levels of EC-SOD are significantly higher in patients with CRF than in controls, and are highest in HD patients. Serum EC-SOD levels measured before HD were significantly higher than those after HD. The increase in serum EC-SOD in patients with renal dysfunction may be caused by a reduced affinity of EC-SOD for vascular endothelial cells, a delay in its metabolic clearance, or the inductive influence of heparin.