[J. Biochem., **117**, 586-590 (1995)] [Lab. of Pharmaceutics]

Heparin-induced release of extracellular-superoxide dismutase form (V) to plasma.

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EC-SOD in serum is heterogeneous with regard to heparin-affinity and can be divided into five fractions (I) to (V), whereas fibroblast-secreted EC-SOD consists mainly of form (V). An intravenous injection of 50 i.u. of heparin/kg body weight into two healthy volunteers led to an immediate rise of serum EC-SOD level by 2.4-2.8-fold. The half-life of serum EC-SOD after the prompt rise was about 90 min. The *in vivo* experiment using rats and an *in vitro* experiment strongly suggested the EC-SOD released into the plasma reconstituted the interaction with glycocalyx on the vascular endothelial cell surface in accordance with the elimination of heparin from the vascular system.

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The heparin-binding site of human xanthine oxidase.

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The enzyme xanthine oxidase (XOD) has an affinity for heparin and can bind to cultured porcine aortic endothelial cells. The heparin-binding sites in human XOD (h-XOD) are studied. From a chymotryptic digest of cyanogen bromide fragmented h-XOD, two peptides with an affinity for heparin-HPLC, A-1 and A-2, were isolated. Amino acid sequence analysis showed that both peptides had lysine and/or arginine residues. The A-1 region may direct its charged side chains toward the solvent while burying its hydrophobic side chains against the hydrophobic inside, because the A-1 peptide forms a highly amphipathic structure. Peptide A-2 contains triple lysine residues and constitutes a hydrophilic region.

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Molecular analysis of extracellular-superoxide dismutase gene associated with high level in serum.

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By molecular analysis of the EC-SOD coding region from the group Ⅱ individuals in Sweden, a single nucleotide substitution of G to C generating an amino acid change of arginine-213 to glycine has been identified in the region associated with the heparin affinity of the enzyme. The same mutation was detected in the Japanese as a homozygote in both alleles of 2 hemodialysis patients and as a heterozygote in one allele of all the healthy group Ⅱ individuals and 17 hemodialysis patients. The amino acid substitution may result in the decrease of the heparin affinity which is favorable for the existence of EC-SOD in the serum.