[*Spctrosc. Lett.*, **31**, 1217-1231 (1998)] [Lab. of Instrumental Center]

Molecular Structure and Dimerization of D-Cycloserine in the Solid State.

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D-Cycloserine (CS) is transformed into *cis*-3,6-bis(aminooxymethyl)-2,5-piperazinedione (CS-dimer) in the solid state under a humid atmosphere. This dimerization process was followed by measureing the IR bands characteristic of CS and CS-dimer. The reaction was accelerated by the presence of increased water vapor. The X-ray analysis of CS monohydrate ($CS \cdot H_2O$) revealed that the CS molecules exist as a zwitter ion where the α-amino N atom is protonated and the amide N atom is deprotonated in the crystal. Participation of water molecules was suggested in the dimerization of CS.

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Association of Extracellular-Superoxide Dismutase Phenotype with the Endothelial Constitutive Nitric Oxide Synthase Polymorphism.

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The distribution of extracellular-superoxide dismutase (EC-SOD) levels in healthy Australian subjects was consistent with two distinct phenotype in which the smaller group of subjects (3.3%) had 15-fold higher levels. The EC-SOD levels in individuals homozygous for endothelial constitutive nitric oxide synthase 4a (ecNOS4a), a rare allele for ecNOS repeat polymorphism at intron 4, were significantly lower than those in ecNOS4A/a and ecNOS4A/A subjects. Furthermore, NO levels were negatively correlated with the EC-SOD levels in common EC-SOD phenotype subjects. Whilst the mechanism remains speculative, it is possible that there is a significant interaction between EC-SOD and ecNOS, or that common factor(s), either genetic or environmental, influence both of them.

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Plasma Extracellular Superoxide Dismutase Levels in an Australian Population with Coronary Artery Disease.

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We explored factors that may affect plasma extracellular-superoxide dismutase (EC-SOD) levels measured by ELISA and assessed the association between plasma EC-SOD and coronary artery disease documented angiographically in 590 white Australian patients. Plasma EC-SOD in female patients was significantly higher than in male patients, and all 19 patients with levels >400 ng/mL were heterozygous for the Arg213 to Gly mutation at the EC-SOD gene; there was also a positive correlation with age. Plasma EC-SOD in current smokers was much lower than in nonsmokers, and ex-smokers had intermediate levels. Levels were significantly lower in patients with than in those without a history of acute myocardial infarction (MI), and low plasma EC-SOD was independently associated with an increased likelihood of a history of MI; higher EC-SOD levels also tended to be associated with delayed onset of MI. These findings are consistent with EC-SOD's being protective and contributing to reduced coronary risk.

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Changes in the Heparin Affinity of Extracellular-Superoxide Dismutase in Patients with Coronary Artery Atherosclerosis.

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Plasma extracellular-superoxide dismutase (EC-SOD) is heterogeneous in heparin affinity and can be divided into five fractions, I to V, by heparin-HPLC. It has been suggested that EC-SOD form V is the primary form synthesized in the body and that EC-SOD forms with reduced heparin affinity are the result of proteolytic truncation of the C-terminal end of EC-SOD form V which is responsible for the binding with heparin. The heparin affinity of plasma EC-SOD in patients with coronary atherosclerosis (CA+ patients) was compared in this study. The increase of plasma EC-SOD form V after heparin injection in CA+ patients was significantly less than that in subjects without evidence of stenosis in their major coronary arteries (CA- subjects). On the other hand, in CA+ patients, EC-SOD forms I to III, with low heparin affinity, were significantly increased compared to those in CA- subjects. The decrease of bound EC-SOD on the endothelial cell surface might cause in part the development of CA.