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Inhibition of purified human sucrase and isomaltase by ethanolamine derivatives.

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Sucrase-isomaltase complex was purified from human intestinal mucosa. Immunostaining shows that sucrase-isomaltase is confined to the area of the striated cell borders of human small intestinal absorptive cells of the villus. Inhibition of sucrase and isomaltase activity by ethanolamine derivatives was investigated. Tris inhibits both types of enzyme activity and is the strongest inhibitor of the ethanolamine derivatives investigated. Bis-Tris inhibited sucrase more than isomaltase. On the other hand, mono-, di- and tri-ethanolamine were weak inhibitors of sucrase but not isomaltase.

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An arginine-213 to glycine mutation in human extracellular-superoxide dismutase reduces susceptibility to trypsin-like proteinases.

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Molecular genetic studies of extracellular-superoxide dismutase (EC-SOD) have shown that individuals with high serum EC-SOD content have a single base substitution generating the exchange of glycine for arginine-213 (R213G) in the heparin-binding domain of this enzyme. The IC_{50} of trypsin for the heparin affinity of mutant EC-SOD (m-EC-SOD) was fivefold that for normal EC-SOD (n-EC-SOD). m-EC-SOD is more resistant to also neutrophil-release trypsin-like proteinases than n-EC-SOD, which causes the heparin affinity of serum EC-SOD to differ in individuals with and without the R213G mutation.

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Substitution of glycine for arginine-213 in extracellular-superoxide dismutase impairs affinity for heparin and endothelial cell surface.

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Extracellular-superoxide dismutase (EC-SOD) levels in sera divide into two discontinuous groups. Molecular genetic studies have shown that the donors in the high-level group have a single base substitution generating the exchange of glycine for arginine-213 (R213G) in the heparin-binding domain of EC-SOD. The binding of mutant EC-SOD (m-EC-SOD) to bovine aortic endothelial cells was about 50-fold less than that of normal EC-SOD. This result suggests that the binding of m-EC-SOD to vascular endothelial cells is much decreased *in vivo*, which causes a high level of serum EC-SOD.