[Urol. Int., **50**, 33-35 (1993)] [Lab. of Pharmaceutics]

Immunopathology of alkaline phosphatase isozymes in seminoma.

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An immunohistopathological study using monoclonal antibodies for alkaline phosphatases demonstrated placental alkaline phosphatase (PLAP)-like substance in the tumor cells of 11 pure seminomas, 1 seminoma with embryonal carcinoma and 1 seminoma metastasis. Liver alkaline phosphatase (LAP) could also be demonstrated in all seminomas but a third intestinal alkaline phosphatase (IAP) was not demonstrable in any tumor. The PLAP-like substance and LAP had considerable enzyme activities. This provides two tumor markers of seminomas detectable in histopathological specimens.

[Seibutsubutsurikagaku, 37, 75-82 (1993)] [Lab. of Pharmaceutics]

Altered form of alkaline phosphatase produced by Hep.2 substrain derived from HeLa cells.

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We purified an alkaline phosphatase (AP) from Hep.2 substrain derived from HeLa cells, by immunoaffinity chromatography using the combination of anti-placental and anti-intestinal enzyme antibodies. It was separated into two bands on SDS-polyacrylamide gel electrophoresis. The N-terminal amino acid sequence indicates that the AP was an intermediate from of the placental and intestinal isozyme. The treatment of the Hep.2 cell with phosphatidylinositol-specific phospholipase C stoichiometrically released the two different subunits of AP. These results clearly indicated that Hep.2 cell can produce an unique AP consisting of the placental and intestinal subunits.

[Biochem. J., **289**, 523-527 (1993)] [Lab. of Pharmaceutics]

Binding of human xanthine oxidase to sulphated glycosaminoglycans on the endothelial-cell surface.

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Purified human xanthine oxidase (h-XOD) had an affinity for heparin-Sepharose. Exposure of h-XOD to the lysine-specific reagent trinitrobenzenesulphonic acid or the arginine-specific reagent phenylglyoxal caused it to lose its affinity for heparin-Sepharose. The binding of h-XOD to heparin is apparently of electrostatic nature, and both lysine and arginine residues are involved in the binding. h-XOD was found to bind to porcine aortic endothelial cells, and this binding was inhibited by the addition of heparin or pretreatment of the cells with heparinase and/or heparitinase. These results suggest that XOD localizes on the outside surface of endothelial cells by association with polysaccharide chains of heparin-like proteoglycans on the endothelial-cell membranes.