

Abstract

beta-Glucuronidase from human lung neoplasms of various histological types and from uninvolved tissues was studied. A significant elevation of beta-glucuronidase activity was observed in adenocarcinoma and squamous cell carcinoma of the lung as compared with the corresponding uninvolved tissues (P less than 0.01). Saccharo-1,4-lactone, a strong inhibitor of the enzyme, exhibited a substantially greater stabilizing effect on the adenocarcinoma enzyme than on the other enzymes. However, removal of the carbohydrate moiety from the adenocarcinoma enzyme by treatment with endo-beta-N-acetylglucosamidase H (endoglycosidase H) brought about a decrease in the stabilizing effect. Tumor beta-glucuronidase showed considerable negative charge heterogeneity in the pI range from 4.2 to 6.2 in isoelectric focusing on polyacrylamide gel. Upon treatment with exogenous alkaline phosphatase or endoglycosidase H, the heterogenous variant forms of the tumor enzyme appeared to partly or completely lose their negative charge and to be converted into forms similar to those of the normal lung enzyme. These data strongly suggest that the variants are highly phosphorylated on the oligosaccharide chains of the enzyme. An experiment on the labelling of beta-glucuronidase with [32P]-phosphoric acid provided further evidence that the acidic variants found in lung cancers are extensively phosphorylated forms of the enzyme.

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