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Influence of ethanol on the activity of glycosidases in rats exposed to cadmium (Cd^{2+})

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Inhibition by ethanol of the activities of lysosomal exoglycosidases in stomach, small intestine, liver and brain of rats exposed to cadmium (Cd^{2+}) was determined. Out of the glycosidases tested the most distinct effect of Cd^{2+} and ethanol administered to the rats *in vivo* was observed in the small intestinal mucosa in a decreasing order: *N*-acetyl-β-hexosaminidase, β-galactosidase and α-fucosidase.

Industrial areas are polluted mainly with heavy metals, among others with cadmium (Cd^{2+}) [1]. It is known that in humans cadmium ions interact with ethanol in their effect on enzymatic proteins causing disturbances in many metabolic processes [2, 3]. After consumption of ethanol the main toxic action is exerted by acetic aldehyde [4], which reacts with sulfhydryl, amino and hydroxyl groups of proteins and affects the activity of the enzymes taking part in both anabolic and catabolic processes [4–7]. The influence of ethanol consumption on the catabolism of glycoconjugates in rats exposed to cadmium is still unknown.

The aim of our work was to evaluate the effect of ethanol on the catabolism of glyconjugates in the organs of rats exposed to cadmium by determining the activity of *N*-acetyl-β-hexosaminidase, β-galactosidase, α-fucosidase and α-mannosidase in the gastric and small intestinal mucosa, liver and brain.

MATERIALS AND METHODS

Male Wistar rats (approx. 190 g body wt.) fed a standard diet were divided into six groups of 5 animals each. The animals received: group I, water; group II, 5 ppm cadmium chloride solution per day for 8 weeks; group III, 50 ppm cadmium chloride solution per day for 8 weeks; group IV, water and for 5 days intragastrically 25% ethanol (5 g/kg of body weight per day); group V, 5 ppm cadmium chloride solution per day for 8 weeks and for 5 days intragastrically 25% ethanol (5 g/kg of body weight per day) and group VI, 50 ppm cadmium chloride solution per day for 8 weeks and for 5 days intragastrically 25% ethanol (5 g/kg of body weight per day).

The rats were killed under ether anaesthesia 24 h after intoxication with Cd^{2+} and/or ethanol consumption. Tissues were homogenized in ice-cold 0.15 M KCl containing 0.2% Triton X-100. The homogenates were centrifuged at 10000 × *g* for 30 min at 4°C. The activity of *N*-acetyl-β-hexosaminidase, β-galactosidase, α-fucosidase and α-mannosidase in the supernatant were determined by the method of Chatteriee *et al.* [8] in the modification of Zwierz *et al.* [9].

Protein was determined according to Lowry *et al.* [10] using crystalline bovine serum albumin as a standard.

The results are presented as means ± S.D. of 5 identically treated animals. The statistical analysis was performed using Student's *t*-test ($P < 0.05$).

RESULTS AND DISCUSSION

Exposure to 5 ppm Cd^{2+} per day in our experimental model corresponds to exposure of the general human population [1], and 50 ppm Cd^{2+} per day in our model corresponds to industrial exposure [11]. Subacute intoxication by ethanol was simulated in our model by intragastric administration of 25% ethanol for five days.

Intoxication of rats (Fig. 1) with cadmium decreased the activity of β–galactosidase and α-fucosidase in stomach (5 and 50 ppm of Cd^{2+}), and of α-fucosidase and α-mannosidase

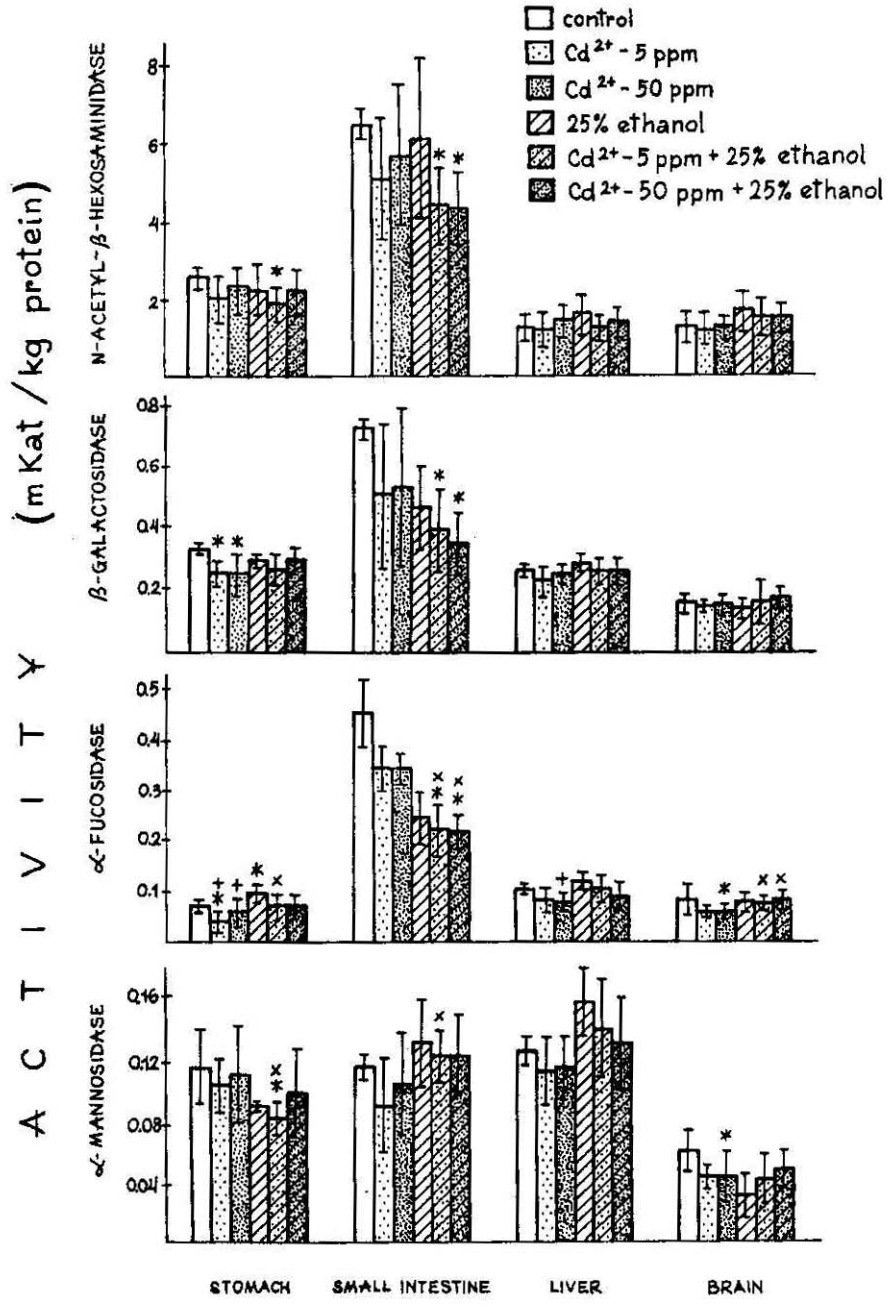

Fig. 1. Activity of glycosidases in rat organs exposed to cadmium (Cd^{2+}) and subacutely intoxicated with ethanol.

*, Significant differences in comparison to the control group; +, significant differences in comparison to the group treated with 25% ethanol; x, significant differences between the group exposed to Cd^{2+} and that exposed to Cd^{2+} and treated with 25% ethanol.

in brain (50 ppm of Cd^{2+}). Rats consuming ethanol without cadmium did not show any changes in glycosidase activities (except the increase of α-fucosidase in stomach). Rats intoxicated with cadmium and additionally given ethanol showed a significant decrease in the activities of *N*-acetyl-β-hexosaminidase, β-galactosidase and α-fucosidase in small intestine (5 and 50 ppm Cd^{2+}) and α-mannosidase in stomach (only after 5 ppm Cd^{2+}).

Our results raise the possibility that cadmium combined with ethanol may decrease the catabolism of glycoconjugates in the digestive tract, i.e. in the tissues exposed to the highest concentration of the above mentioned toxic substances. The absence of response to cadmium exposure and ethanol intoxication of the systems catabolising glycoconjugates in liver, may indicate that either the stomach and small intestinal mucosa are the main lines of defence which lower the concentration of toxic substances reaching the liver or, alternatively, liver exoglycosidases are less sensitive to cadmium and ethanol than the exoglycosidases in other tissues. The latter possibility is supported by the fact that the activity of α-mannosidase in brain decreased after exposure to cadmium, which reaches the brain *via* vena porta and liver.

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