those obtained by using fluorimetric[5,11], gas chromatographic[12,13] and radioenzymatic[6,7,14] procedures. On the other hand, DOPAC values in the caudate nucleus were found to be about twice higher than those reported by others[5,13]. The difference might be due to the fact that we dissected an area restricted to the head of the caudate nucleus.

Pargyline, a MAO inhibitor, enhanced DA content and produced a disappearance of DOPAC levels in 3 brain structures studied within 60 min after treatment, indicating a rapid turnover of DOPAC in 3 brain areas. Reserpine caused a marked decrease of DA in these brain areas while surprisingly enhancing the DOPAC content only in the caudate nucleus. Haloperidol increased DOPAC levels in the caudate nucleus, substantia nigra, but failed to do so in the medial basal hypothalamus. Our results confirm previous observations[15] that different dopaminergic areas may respond to psychotropic drugs in a different manner.

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Effect of chronic alloxan diabetes and insulin administration on intestinal brush border enzymes

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Summary. Brush border sucrase and lactase activities are significantly elevated in alloxan-induced chronic diabetes and are restored to control levels after insulin treatment. Alkaline phosphatase and Mg-ATPase levels remain unchanged in diabetes, compared to a control group. Insulin treatment alone to control animals also led to enhanced activities of these enzymes.

Morphological and functional alterations in the intestine of diabetics have been well documented. Increase in the intestinal absorption of sugars and amino acids in diabetic animals has been described[1-4]. Similar changes in the activities of various disaccharidases in the intestine, following an acute dose of alloxan to rats, have been observed[5-6], which could be because of the specific induction of these enzymes in response to alloxan, or because of the metabolic disturbances associated with alloxan-induced diabetes. In order to differentiate between these 2 possible effects, and in view of the fact that there are no reports available on the effect of chronic diabetes on brush border enzymes, the present study was undertaken. In addition to its effects on brush border sucrase, lactase and alkaline phosphatase (AP), the effect of chronic alloxan diabetes on intestinal Mg-ATPase was also investigated.

Materials and methods. Male albino rats (120-140 g) bred in the Institute colony were used. The procedure for the induction of diabetes and insulin treatment of the animals was essentially the same as described by Chauhan and Sarkar[7]. Animals in control (A), diabetic (B), diabetic + insulin (C) and control + insulin (D) groups were observed for 120 days and sacrificed after giving ether anesthesia. Intestines were removed, washed with chilled normal saline, and brush border membranes prepared according to Schmitz et al.[8]. The membrane fragments were suspended in 10 nM sodium maleate, pH 6.8, containing 0.02% sodium azide. Sucrase and lactase activities were measured using glucose oxidase peroxidase system[9,10]. Alkaline phosphatase was determined as described by Eiccholz[11] Mg-ATPase activity was assayed in the intestinal homogenates as previously reported[12]. Blood sugar was measured by Somogyi's method[13]. Protein estimation was done according to Lowry et al.[14].

Results and discussion. Results on the effect of chronic alloxan diabetes and of insulin administration to rats are shown in table 1. There is a 2fold increase in the activities of both sucrase and lactase in alloxan-treated animals

Table 1. Effect of chronic alloxan diabetes and insulin administration on brush border disaccharidases

Group		Blood sugar at the time of sacrificing (mg/100 ml)	Sucrase μmoles glucose/min g protein at 37 °C	Lactase
A	Control	85 ± 11	318.3 ± 20.6	44.5 ± 1.5
B	Diabetic	357 ± 28	714.1 ± 27.6	86.3 ± 3.9
C	Diabetic + insulin	126 ± 12	347.3 ± 13.8	52.9 ± 5.3
D	Control + insulin	78 ± 10	536.2 ± 11.6	94.7 ± 4.6

Values are mean ± SD of 6-8 determinations.

Table 2. Effect of chronic alloxan diabetes and insulin administration on intestinal alkaline phosphatase and Mg-ATPase activities

Group		Alkaline phosphatase μmoles phenol/min g protein at 37 °C	Mg-ATPase μmoles Pi/min g protein at 37 °C
A	Control	13.37 ± 1.49	5.31 ± 0.45
B	Diabetic	13.29 ± 2.31	5.55 ± 0.58
C	Diabetic + insulin	14.42 ± 2.78	6.75 ± 0.84*
D	Control + insulin	20.43 ± 4.36	6.59 ± 0.53*

Values are mean ± SD, n = 8. * $p < 0.05$ compared to control.

compared to controls. These results are in agreement with those reported by Younoszai and Schedl[5], who found a similar increase in the disaccharidase levels on 5th day of alloxan administration. Our results with chronic alloxan diabetic animals obviously suggest that increase in the sucrase and lactase activities may not be due to toxic effects of alloxan, which could be prevalent in short-term experiments. Furthermore, the enhanced activity of these enzymes cannot be attributed to increase in intestinal cell mass or its surface area observed in diabetes[15], since there is no change in the activity of brush border AP (table 2), which is known to be located on the same site of mucosal membrane as the disaccharidases[16]. Insulin administration to diabetic animals restored the activity of these enzymes to almost control levels. The specific increase in the level of these enzymes is most likely due to elevated levels of glucagon, cortisone or catacholamines observed in diabetes[17,18], and is not due to the lack of insulin in this derangement, since insulin administration alone to control animals also augments the activity of sucrase and lactase. Increase in the concentration of these insulin antagonists also occurs in insulin induced hypoglycemia[19], which may again be responsible for the increased enzyme activities in insulin-treated animals. Recently Clenano et al.[20] showed that cortisone or tri-iodothyronine injection to pregnant female rats can elicit precocious appearance of jejunal sucrase in their foetuses.

There is no change in the activities of AP and Mg-ATPase in diabetic rats compared to controls, as revealed by the results shown in table 2; however, insulin treatment of the animals led to a marked increase of AP and an appreciable stimulation of Mg-ATPase activities. Increase in the uptake of sugars and amino acids in the intestine of insulin-treated animals has also been demonstrated[21,22]. Such a facilitative action of insulin on the enzyme systems could be due to the general anabolic action of this hormone on protein biosynthesis[23]. In view of the close functional link between sugar absorption process and the disaccharidases in intestine[24,25], it would appear that increased sugar uptake and the disaccharidase activities in diabetes and in hyperinsulinism is due to similar or identical mechanism(s).

In order to elucidate whether increase in the activity of disaccharidases in diabetes is the result of a new enzyme formation with high substrate affinity or increased maximum velocity, we studied the kinetics of sucrase in control and in diabetic animals. Kinetic parameters calculated from the double reciprocal plot (figures not shown) indicate that there is no change in K_m of the enzyme in diabetic and control animals ($K_m = 24.4$ mM and 26.3 mM in diabetic and control groups respectively). But V_{max} (μmoles glucose/min mg protein) increases from 1.79 in control to 3.33 in diabetic animals. This clearly suggests a net increase in the enzyme content.

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Sex difference in polyethylenglycol-induced thirst

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Summary. The polyethylenglycol-induced thirst in male and female castrated rats has been studied. The polyethylenglycol (PG) increases the water intake more in females than in males. Estradiol benzoate and testosterone P. diminishes the amount of water drunk after PG treatment in the females, but not in the males.

Extracellular thirst can be stimulated by reducing the volume of extracellular fluid, without changing the general osmolarity or the volume of the intracellular compartment. This can be done by injecting i.p. or s.c. a hyperoncotic colloidal solution of polyethylenglycol (PG)[1,2]. It was proved that the injection of PG produces an acute edema in the area of the injection and an increase in the activity of the plasmatic renin (PRA)[2]. This increases of the PRA provokes the formation of greater quantities of angiotensin-II, which, as is know, is a powerful dipsogen when administered in different ways[2-5].

Because the food intake seems to be related to sexual factors[6-8], this would be possible also for water intake. We have seen[9] that the administration of angiotensin-II produces different effects on the water intake in male and female rats. The females ingest more water than the males when stimulated with angiotensin-II. A progressive pattern of differentiation was observed between the 2 sexes when the animals were castrated at different levels of their development. The males and females castrated at birth drunk exactly the same volumes of water when, as adults, they were injected with angiotensin-II. The differences started when the animals were castrated before puberty or as adults. The object of the present study is to clarify the real significance of sex, and the role of the sexual hormones in the extracellular thirst.