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ADENOSINE DEAMINASE ACTIVITY IN THE HUMAN DUODENAL MUCOSA IN RELATION TO GASTRIC ACID SECRETION

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Adenosine deaminase (ADA) activity was estimated in mucosal specimens obtained endoscopically from the duodenal bulb. Three groups of subjects were studied: 1. 9 patients with achlorhydria, 2. 12 subjects with normal gastric acid secretion, 3. 5 patients with hypersecretion. Enzyme activity was measured by determination of ammonia liberated from the substrate according to the Chaney and Marbach method. In patients with hypersecretion the ADA activity was lower than in those with achlorhydria ($p < 0.001$) and normal acid secretion ($p < 0.02$). A significant negative correlation between ADA activity in the duodenal bulb mucosa and basal and maximal gastric acid outputs was found. The present study seems to indicate a possible relationship between gastric acid secretion and duodenal ADA activity.

Key words: *adenosine deaminase, gastric acid secretion, duodenal mucosa.*

INTRODUCTION

The physiological role of adenosine in the duodenum is not fully understood. The adenosine content in the duodenal mucosa does not depend on gastric acid secretion and it remains unchanged despite the ulcer generation (1). Adenosine deaminase (ADA) activity, the main enzyme of adenosine inactivation, is relatively high in the duodenum compared to other segments of the gastrointestinal tract (2, 3). Its role in adenosine-mediated relaxation of duodenal smooth muscles was documented (4).

Previously, a positive correlation between ADA activity in the gastric mucosa (fundic region) and acid outputs was found (5). The purpose of this study was to determine ADA activity in the duodenal bulb mucosa in relation to basal and pentagastrin-stimulated gastric acid secretion.

MATERIAL AND METHODS

26 subjects (17 male and 9 female), aged 18—74 years, were included in the study. They were divided into 3 groups according to the results of the gastric acid secretion tests. Group 1 consisted of 9 patients with achlorhydria and histologically confirmed atrophic gastritis. Group 2 involved 12 patients with a mean basal acid output (BAO) — 1.25 mM/h and a mean maximal acid output (MAO) — 11.28 mM/h. They had histologically verified superficial chronic gastritis. In 5 patients of group 3, the mean value of BAO was 7.26 mM/h and MAO — 26.32 mM/h; the patients of this group had chronic duodenal ulcers. None of the patients received any drugs for 48 hours before endoscopic examination.

Gastric acid secretion was measured according to Kay's method using pentagastrin (6 μg/kg body weight subcutaneously) as a stimulus. Gastroduodenal endoscopy was performed with a GIF K10 (Olympus) panendoscope. 3—6 samples of duodenal bulb mucosa, each weighing 4—6 mg, were taken for ADA activity and protein content examination.

The ADA activity in the mucosal homogenates was measured by determination of ammonia liberated from the substrate according to the method of Chaney and Marbach (6). The protein was assayed by the method of Lowry et al. (7). The results were expressed as nM NH_3/mg of protein/min.

The results were evaluated statistically using Student's t-test for unpaired data accepting statistical significance at 5% level.

The study was approved by the Institutional Ethical Committee and informed consent was obtained from all subjects.

RESULTS

No significant difference in duodenal ADA activity was found between the group of patients with achlorhydria and the group with normal acid secretion *(Table 1)*. The lowest ADA activity was observed in the group of patients with gastric hypersecretion; the differences in relation to the other groups studied were statistically significant. Moreover, we found a significant negative correlation between the ADA activities in the duodenal bulb mucosa and BAO ($r = 0.709$, $p < 0.001$) and MAO ($r = 0.708$, $p < 0.001$) values *(Fig. 1)*.

Table 1. Basal and stimulated gastric acid outputs and adenosine deaminase activity in the duodenal mucosa

Groups of patients	Acid secretion (mM/h)		ADA activity (nM NH_3/mg protein/min)
	BAO	MAO	
1	0	0	340.20 ± 63.79
2	1.25 (0.02—3.20)	11.28 (1.22—25.67)	260.26 ± 96.66
3	7.26 (5.20—9.88)	26.32 (15.94—39.74)	118.57 ± 49.71[ab]

[a] $p < 0.001$ — group 3 versus group 1
[b] $p < 0.02$ — group 3 versus group 2

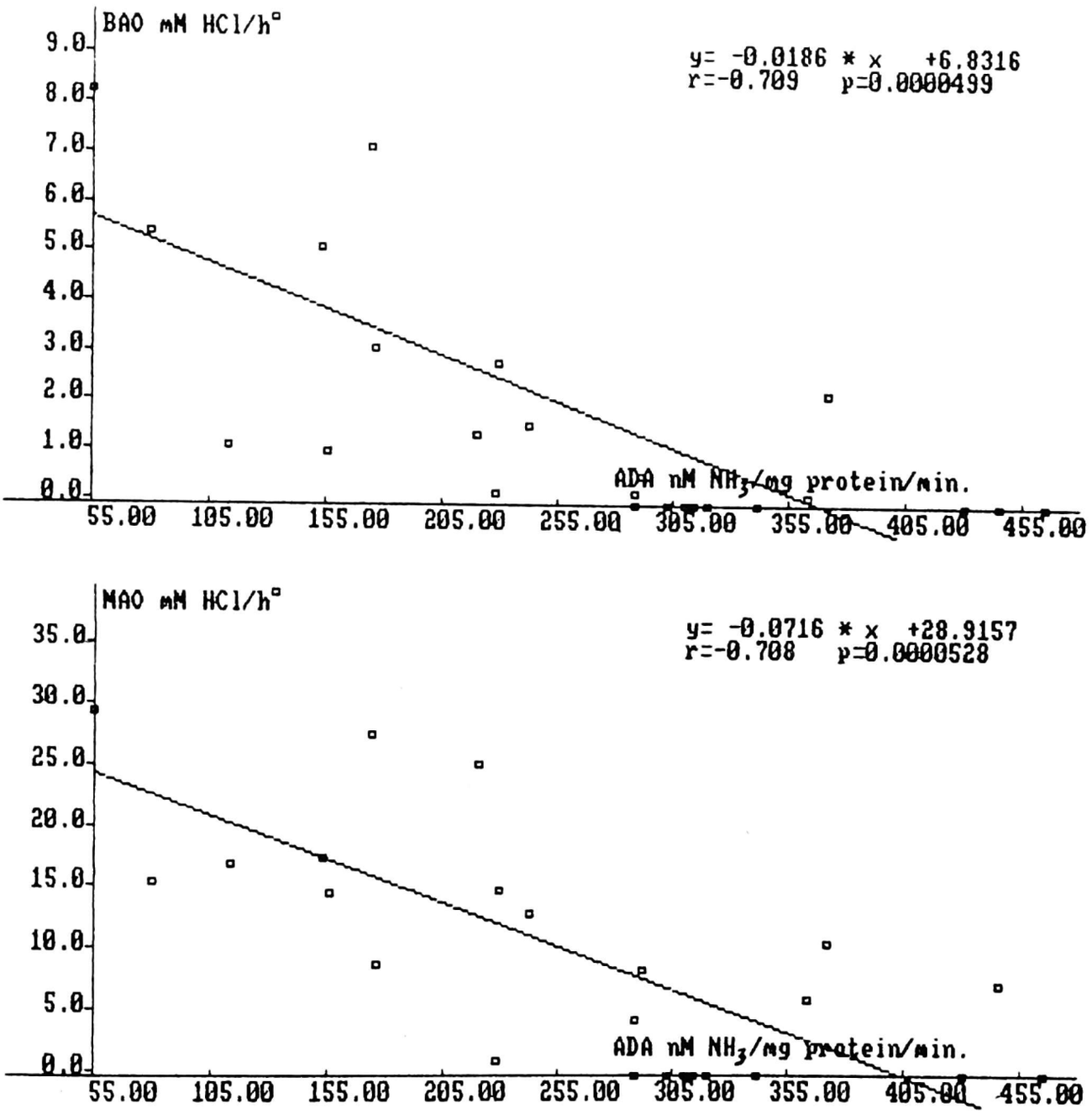

Fig. 1. Relationship between ADA activity in the duodenal bulb mucosa and the BAO and MAO values.

DISCUSSION

Vetvik et al. (8) found significantly decreased activities of some membrane (lactase and neutral-alpha-glucosidase) and mitochondrial (monoamine oxidase) enzymes in the duodenal bulb in patients with duodenal ulcers. The enzyme activities were unrelated of the ingestion of antacids before the investigation (8). Unfortunately, the acid secretion was not measured in these patients. In dogs (9) and rats (10), gastric hypersecretion led to decreased activities of the various enzymes in the duodenum; the mechanism of these observations remains unknown.

We observed a negative correlation between basal and stimulated gastric acid secretion and ADA activity in the duodenal bulb mucosa; the lowest activity of the enzyme was noted in the patients with duodenal ulcer and gastric acid hypersecretion. It should be stressed that ADA activity in the gastric fundic mucosa was highest in the patients with hypersecretion and a positive correlation between BAO and MAO values and the enzyme activities was observed (5). Such a relationship was not found in the mucosal samples coming from the antrum (5). It would seem that ADA could possible be involved in the regulatory mechanism of gastric acid secretion. Hypothetically, ADA can also be important for the secretory systems in the duodenal mucosa, especially since its activity is particularly high in this segment of the gastrointestinal tract (2, 3).

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