

**COMPARATIVE EFFECTS OF AMYLASE ADMINISTRATION INTO THE
DUODENUM AND THE ILEUM**

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ABSTRACT

This urgent adaptation is also manifested in the characteristic set of enzymes in the secretion released in response to each type of food. In ensuring rapid adaptations of the secretory activity of the pancreas to the type of food consumed, corrective influences on the gland originating from the duodenum are of great importance, depending on the composition and properties of its chyme.

Receptors, and especially endocrine cells of the mucous membrane of the duodenum, respond to many components of the duodenal contents, including the presence of pancreatic secretions. The inhibition of pancreatic secretion is associated with the action of pancreatic proteinases and products of proteolysis on the mucous membrane of the duodenum and its endocrine apparatus.

The role of other pancreatic juice hydrolases has been insufficiently studied, and only a few studies have addressed this issue. The presence of such influences would allow a more complete understanding of the subtle adaptations of pancreatic secretion based on the corrective effects of the duodenum on the pancreas, not only through positive stimulating influences but also through negative inhibitory ones.

Keywords

Pancreas, duodenum, ileum, pancreatic juice, secretion, digestive enzymes.

The data from our series of experiments indicate that selective inhibition of amylase secretion occurs through the duodenum but not through the ileum. Taking into account data from the literature and the fundamental differences in the endocrine apparatus of the proximal and distal sections of the small intestine, it can be concluded that the selectivity of the duodenal inhibitory effects of amylase is mediated through the endocrine apparatus of the duodenum.

Two acute series of experiments were conducted and their results were analyzed. Pancreatic secretion was stimulated by introducing acidified hydrolysine into the duodenum. However, in one series of experiments pancreatic amylase was introduced into the duodenum, while in the other it was introduced into the ileum.

The dose of amylase administered into the intestine in both series of experiments was the same: **0.15 mg/kg/h**. This dose of amylase, when administered into the duodenum, produced selective inhibition of pancreatic amylase secretion due to a decrease in the amylolytic activity of the juice. Due to the action of amylase, the concentration of protein in the pancreatic juice and the rate of its secretion decreased (Table 1). Other indicators of pancreatic secretion did not change.

During the hour of amylase administration into the ileum, inhibition of pancreatic secretion was not observed for any component of the pancreatic juice. Moreover, in several experiments there was a tendency toward an increase in the amylolytic and lipolytic activity of the juice, but the wide variation of the average values made these changes statistically insignificant.

During the subsequent two hours, secretion according to most of its parameters was inhibited non-selectively: the volume of secretion decreased, the bicarbonate content in the juice decreased, and the amylase content decreased. Due to the reduction in juice volume, the secretion rates of bicarbonates, total protein, amylase, lipase, and proteases also decreased sharply.

These data allow the conclusion that the selective inhibitory effect of amylase on its own secretion by the pancreas occurs only when amylase acts through the duodenum.

Previously, we noted that general inhibitory effects on pancreatic secretion are observed when there is a shift in the homeostasis of pancreatic hydrolases in peripheral blood. This is evidenced not only by changes in enzymatic activity but also by changes in urine indicators. A comparison of these parameters in experiments involving the administration of **0.15 mg/kg of amylase** into the duodenum and into the ileum revealed that, during the hour of amylase administration from the distal section of the small intestine, the indicators of its activity did not show significant changes.

Table 1.

Pancreatic secretion during (A) and after (B) the administration of amylase into the **duodenum (I)** and the **ileum (II)** (as a percentage of the values before amylase administration, $M \pm m$).

Site of administration	Secretion time	Volume	Bicarbonates	Protein	Amylase	Lipase	Protease
I	A	83,6 ±14,5	93,1 ±10,7 86,4 ±19,9	83,0 ±11* 53,5 ±14,2	58,6 ±12,6* 88,2 ±17,0	87,9 ±6,7 149,2 ±17,8	112,2 ±23,1 191,1 ±39,9
	Б	82,2 ±8,1	104,1 ±21,8 82,8 ±15,7	74,6 ±13,5 63,9 ±13,8	78,4 ±14,8 48,9 ±15,1*	110,5 ±25,1 94,9 ±23,7	118,7 ±30,7 152,2 ±48,7
II	A	100,0 ±10,1	97,0 ±11,8 98,4 ±17,8	78,9 ±11,3 86,6 ±14,5	122,2 ±29,6 120,9 ±29,8	120,7 ±33,4 124,1 ±44,2	98,4 ±10,7 76,8 ±13,7
	Б	52,9 ±6,2*	74,8 ±10,6* 45,3 ±11,0	83,4 ±11,5 45,1 ±6,2*	70,6 ±7,3 39,4 ±3,7*	66,8 ±21,9 39,7 ±5,7*	99,4 ±16,3 29,2 ±3,4*

Note: * – significant differences; numerator – content and activity, denominator – secretion rate.

Changes in homeostasis did not show significant effects initially, but during the following two hours (Table 2, II B) there was a marked increase in urinary amylolytic activity and the excretion rate of amylase in urine. Amylolytic activity in blood during the hour of amylase administration into the duodenum did not change, but diuresis increased, and urinary amylolytic activity was highly unstable with generally elevated values (Table 2, I A), as was the renal excretion rate of amylase. In the subsequent hour, amylase indicators increased further and became even more variable.

Table 2.

Blood and urine amylase during (A) and after (B) administration of amylase into the **duodenum (I)** and the **ileum (II)** (as a percentage of the values before amylase administration, $M \pm m$)

Site of administration	Secretion time	Blood amylase	Urine		
			Diuresis	Activity	Secretion rate
I	A	97,3 ±31,6	127,9 ±9,3*	1160 ±871	1406 ±1090
	B	77,1 ±25,0	126,4 ±22,2	2590 ±2285	2202 ±1776
II	A	99,6 ±8,4	103,3 ±8,7	176,7 ±46,5	168,0 ±37,0
	B	96,1 ±4,6	120,7 ±15,9	1347 ±466*	1433 ±142*

References

1. Korotko G.F. *Introduction to Physiology of the Gastrointestinal Tract*. “Meditsina UzSSR”, Tashkent, 1987. – 219 p.
2. Korotko G.F., Baybekova G.D., Nishanova A.A. Mechanisms of Pancreatic Secretion Stimulation upon Drainage of Its Juice from the Duodenum. *Physiological Journal of the USSR*, 1991, No. 10, pp. 94–101.
3. Araya S., Kuster E., Gluch D., Mariotta L., Lutz C., Reding T.V., Graf R., Verrey F., Camargo S.M.R. Exocrine pancreas glutamate secretion helps to sustain enterocyte nutritional needs under protein restriction. *Am J Physiol Gastrointest Liver Physiol*. 2018 Apr 1;314(4):G517–G536. doi: 10.1152/ajpgi.00135.2017. Epub 2017 Nov 22. PMID: 29167114.
4. Chaudhary A., Domínguez-Muñoz J.E., Layer P., Lerch M.M. Pancreatic Exocrine Insufficiency as a Complication of Gastrointestinal Surgery and the Impact of Pancreatic Enzyme Replacement Therapy. *Dig Dis*. 2020;38(1):53–68. doi: 10.1159/000501675. Epub 2019 Aug 16. PMID: 31422398; PMCID: PMC6979421.
5. Diéguez-Castillo C., Jiménez-Luna C., Prados J., Martín-Ruiz J.L., Caba O. State of the Art in Exocrine Pancreatic Insufficiency. *Medicina (Kaunas)*. 2020 Oct 7;56(10):523. doi: 10.3390/medicina56100523. PMID: 33036352; PMCID: PMC7599987.
6. Lan X., Robin G., Kasnik J., Wong G., Abdel-Rahman O. Challenges in Diagnosis and Treatment of Pancreatic Exocrine Insufficiency among Patients with Pancreatic Ductal Adenocarcinoma. *Cancers (Basel)*. 2023 Feb 20;15(4):1331. doi: 10.3390/cancers15041331. PMID: 36831673; PMCID: PMC9953920.