

# BIOCHEMICAL CHANGES IN THE BLOOD IN VARIOUS FUNCTIONAL STATES OF THE PANCREAS

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When pancreatic function is disturbed in dogs, reciprocal changes take place in the blood levels of the pancreatic enzymes (the trypsin concentration falls while the amylase concentration rises). The serum trypsin inhibitor level remains unchanged under these circumstances or falls.

The diagnosis of diseases of the pancreas is nowadays largely based on the results of biochemical tests, including investigation of the blood levels of amylase, trypsin, trypsin inhibitor, lipase, deoxyribonuclease, methemalbumin, and so on [9, 10, 17-19]. Some investigators consider that the most sensitive and reliable biochemical test is the ratio between the concentration of trypsin inhibitor and the trypsin level in the blood serum, which falls appreciably in pancreatitis [3, 12]. However, other workers [15, 16, 20] found an increase in the concentration of trypsin inhibitor during an exacerbation of this disease. Lukanev [5] considers that a decrease in the serum trypsin level is an important indicator of pancreatic exocrine function.

A parallel investigation of the concentrations of trypsin and its inhibitor in the blood serum during experimental disturbance of the pancreatic exocrine function was carried out, and in some cases the blood amylase level was determined at the same time.

## EXPERIMENTAL METHOD

Experiments were carried out on seven dogs with a chronic Pavlov pancreatic fistula. In two dogs the discharge of juice through the fistula was stopped at various times after the operation by healing over of the external orifice of the fistula. In these animals, attempts were made subsequently to produce experimental pancreatitis. For this purpose crystalline trypsin (Spofa, Czechoslovakia) in a dose of 0.5 mg/kg body weight, in 0.9% NaCl solution, was injected under pressure into the pancreatic duct of the dog Dzhaz through a puncture wound in its wall. After laparotomy the same dose of trypsin was injected from a syringe into various parts of the pancreas of the dog Zhuchok. Two dogs undergoing laparotomy alone served as the control.

The concentrations of trypsin and its inhibitor in the blood serum [12, 14] and of amylase in the blood [6] were determined.

## EXPERIMENTAL RESULTS

In dogs with an external fistula of the pancreatic duct, a specific disease resembling pancreatitis in character develops at various times after the operation [2, 8]. The amylase concentration in the blood of these animals was increased (Table 1). This confirmed previous findings [1, 7]. Meanwhile, not only was the serum trypsin level not increased in any of the seven dogs which developed the disease, but on the contrary, it was considerably reduced. In some of these dogs the tryptic activity of the blood serum fell on

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TABLE 1. Concentrations of Trypsin and Its Inhibitor in Blood Serum and of Amylase in Blood of Dogs with Experimental Disturbance of Pancreatic Function ( $M \pm m$ )

Procedure	Dog's name	Satisfactory state			Period of illness			Significance of differences (P)		
		trypsin (in milliunits/ml) 1	trypsin inhibitor (in milliunits/ml) 2	amylase (in mg %) 3	1	2	3	1	2	3
Exteriorization of main pancreatic duct with a subsequent loss of juice	Zhuchok	1,3+0,2	551+21	—	0,4+0,06	475+11	—	0,001	0,01	—
	Kashtan	1,5+0,2	573+25	—	0,2+0,14	355+39	—	0,001	0,01	—
	Akbar	1,2+0,25	524+41	69+8,5	0,2+0,05	370+20	129+18	0,01	0,01	0,02
	Bobik	0,8+0,06	510+8	71+10,5	0,06+0,06	361+27	128+22,4	0,001	0,001	0,05
	Dzhaz	2,5+0,1	476+36	—	1,8+0,6	387+18	—	0,2	0,05	—
	Ashkar	1,1+0,0	578+10	39+4,3	0,5+0,1	469+21	73+5,6	0,001	0,05	0,01
	Feya	3,3+0,65	298+24	—	3,9+1,04	409+23	—	0,5	0,01	—
Before injection					After injection					
Injection of trypsin:										
into gland . . . . .	Zhuchok	2,2+0,17	614+6	52	0,2+0,11	603+34	238+83,5	0,001	>0,5	—
into duct . . . . .	Dzhaz	2,4+0,14	602+23	55	1,3+0,13	604+29	129+38,7	0,001	>0,5	—

certain days to zero. The decrease in trypsin concentration was accompanied as a rule by a decrease in the blood level of its inhibitor.

When pancreatic function was disturbed by injection of a solution of crystalline trypsin, just as during the illness accompanied by loss of juice, the trypsin level was lowered, and the amylase concentration increased in the blood. However, no significant changes in trypsin inhibitor were observed in these cases.

In dogs with pancreatitis accompanied by loss of pancreatic juice, as also following injection of crystalline trypsin into the pancreas (i.e., with no loss of juice), the changes in the indices studied were thus similar.

The increase in the blood amylase level in pancreatitis is associated with many factors, including increased pressure in the duct system and disturbance of the integrity or altered permeability of the membranes of the acinar cells [13]. However, it is difficult to explain the decrease in trypsin concentration during pancreatitis by a change in membrane permeability of the acinar cells, for the trypsin molecule is small. The decrease in serum trypsin concentration likewise cannot be explained by inactivation of the enzyme [11]. It is perhaps due to weakening of the trypsin-synthesizing ability of the pancreatic acinar cells, although under these conditions precursors of trypsin are found in the juice [4, 11].

#### LITERATURE CITED

1. A. A. Anoshina, in: The Physiology of Digestion [in Russian], Part 1, Odessa (1967), p. 9.
2. G. N. Voronin and Z. S. Lavrova, Transactions of the Institute of Experimental Medicine, Academy of Medical Sciences of the USSR [in Russian], Vol. 9, No. 2, Leningrad (1966), p. 31.
3. I. I. Knyazev, in: Proceedings of a Conference on the Pathogenesis, Clinical Picture, and Treatment of Diseases of the Pancreas [in Russian], Moscow (1965), p. 48.
4. I. I. Lintvarev, Bol'nich. Gazeta Botkina, No. 19, 817 (1901); *ibid.*, No. 20, 878 (1901).
5. G. D. Lukanev, Lab. Delo, No. 10, 30 (1968).
6. G. F. Milyushkevich, Byull. Éksperim. Biol. i Med., No. 12, 3 (1956).
7. G. F. Milyushkevich, Proceedings of a Conference on Problems in the Physiology and Pathology of Digestion [in Russian], Tartu (1959), p. 278.
8. A. V. Rikkl', in: Interaction between Organs of the Digestive System [in Russian], Leningrad (1968), p. 110.
9. S. I. Rybakov, in: The Physiology and Pathology of the Digestive Organs [in Russian], Kiev (1968), p. 134.
10. T. I. Tikhonova and B. D. Amelichev, in: The Physiology and Pathology of the Digestive Organs [in Russian], Kiev (1968), p. 162.
11. E. P. Fenina, Fiziol. Zh. SSSR, No. 4, 671 (1970).
12. V. A. Shaternikov, Vopr. Med. Khimii, No. 1, 103 (1966).
13. H. L. Bockus, Gastroenterology (Philadelphia), 3 (1965).

14. B. F. Erlanger, N. Kokowsky, and W. Cohen, *Arch. Biochem.*, 95, 271 (1961).
15. J. Hardy, L. Balart, and C. C. Graighead, *Ann. New York Acad. Sci.*, 146, 548 (1968).
16. G. M. Homer, R. E. Lipf, and T. E. Hieber, *Am. J. Clin. Path.*, 34, 99 (1960).
17. O. D. Kowlessar and R. K. McEvoy, *J. Clin. Invest.*, 35, 1325 (1956).
18. B. E. Northam, D. S. Rowe, and N. E. Winstone, *Lancet*, 1, 348 (1963).
19. O. Paganoni, *Schweiz. Med. Wschr.*, 96, 50 (1966).
20. A. G. Thompson, *Ann. New York Acad. Sci.*, 146, 540 (1968).