Absorption of pancreatic lipase from the duodenum into lymphatics

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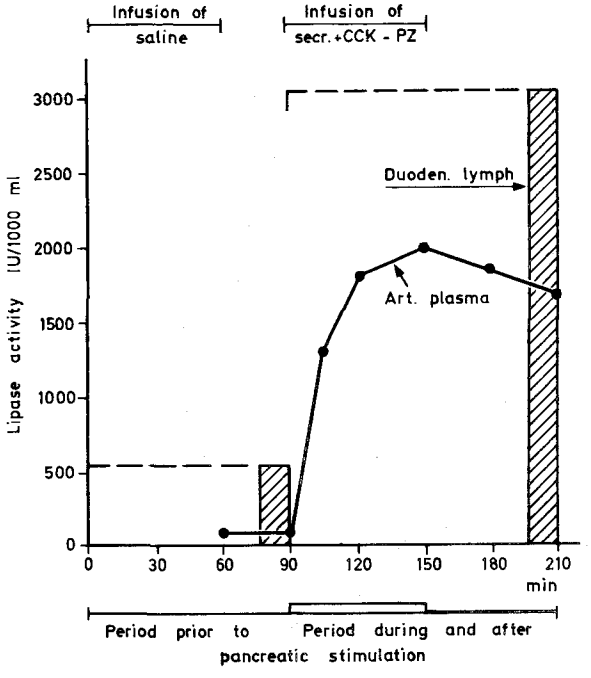
Summary. A significantly higher lipase activity was measured in the duodenal lymph samples of 15 dogs than in each of corresponding arterial blood plasma samples collected prior to, during and after maximal hormonal stimulation of pancreatic secretion. The result may be evaluated as a sign of pancreatic lipase absorption by the duodenum into lymphatics.

Some of the digestive enzymes administered or secreted into the intestinal lumen are absorbed as intact enzyme molecules[1–7]. They are transported by the blood stream and lymph[5] and the amount carried back into pancreatic acinar cells is again secreted into the intestinal lumen[6,7]. To study the absorption of pancreatic enzymes, lipase activity was determined in the duodenal lymph and in the blood plasma of the femoral artery of dogs prior to and during maximal hormonal stimulation of exocrine pancreatic function.

Material and methods. 15 male dogs of 18 kg mean weight were fasted after a copious meal for 24 h. Under chloralose anaesthesia (0.1 g/kg b.wt), laparotomy was performed and the lymphatics originating from the main parts of the duodenum were cannulated by polyethylene cannulas close to the organ, before the duodenal lymph could have been contaminated by pancreatic lymph. Duodenal lymph was collected first for 90 min during saline infusion (1st lymph collection period) then for 120 min during and after stimulation of exocrine pancreatic secretion by 4 U/kg b.wt of secretin and pancreozymin (CCK-PZ) (GIH Laboratory) dissolved in saline (2nd lymph collection period). Saline, as well as the hormone solution, was infused into the femoral vein at a rate of 1.0 ml/min for 60 min. Blood samples from the femoral artery were collected in the 60th min of the 1st lymph collection period (initial values) then during hormone infusion in the 20th, 40th and 60th min and, after stopping it, in the 90th and 120th min of the 2nd lymph collection period. The samples were collected into heparinized tubes kept in ice. Pancreatic lipase activity was measured[8] in each lymph and plasma sample after centrifugation as well as in pancreatic juice collected from the Santorini's duct, which was cannulated when the experiment was finished. The principle of the method is to measure acid release in 2 mixtures incubated for 120 min. Both mixtures consisted of the sample and sunflower oil, but to one of them ethanol was added immediately to inactivate the lipase (control). The difference in acid release between the 2 mixtures was expressed as lipase activity in IU/1000 ml[8]. Lipase activity in the lymph and plasma samples was compared statistically after analysis of variance by Dunnet contrast and Student's t-test.

Results. Under basal conditions, mean pancreatic lipase activity was significantly higher in the duodenal lymph than in arterial blood plasma ($p < 0.01$) (table). When hormonally stimulated pancreatic juice was secreted into the duodenum, lipase activity was significantly elevated both in the duodenal lymph ($p < 0.01$) and in each of the plasma samples ($p < 0.01$) as compared to the initial values (table). Under such conditions, mean pancreatic lipase activity in the duodenal lymph samples was significantly higher ($p < 0.01$) than in the plasma samples (table). A typical experiment is shown in the figure. Under hormonal stimulation, pancreatic juice of very high lipase activity was secreted into the duodenal lumen (table).



Lipase activity in duodenal lymph and arterial blood plasma in a typical experiment.

Discussion. Pancreatic lipase activity in arterial blood plasma originates from the pancreas due to the escape of enzyme from the excretory duct system into the interstitium of the gland and drained from there by venous and lymphatics routes[9–10]. It has been shown that, after stimulation of pancreatic excretion, lipase activity was considerably elevated in thoracic duct lymph and blood plasma of the femoral artery[11] as well as in pancreatico-

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Pancreatic lipase activity (IU/1000 ml) in duodenal lymph and arterial blood plasma of dogs prior to and during stimulated exocrine pancreatic secretion

Initial values (Saline$^+$: 0–60 min)		Stimulation of pancreatic secretion (4 U/kg b.wt secretin + pancreozymin$^+$: 0–60 min)					
Duodenal lymph	Arterial blood plasma	Duodenal lymph	Arterial blood plasma				Pancreatic juice
0–90 min	60 min	0–120 min	20 min	40 min	60 min	120 min	120 min
671 ± 103 (15)	279 ± 31 (15)	2391 ± 394 (15)	1141 ± 241 (15)	1460 ± 248 (15)	1569 ± 288 (15)	1578 ± 325 (15)	$433 \times 10^3 \pm 61 \times 10^3$ (15)

$p < 0.01$ $p < 0.01$

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The mean $\pm$ SE lipase activity in arterial blood plasma of 5 dogs collected in the 90th min of the pancreatic stimulation period was 1405 ± 184 IU/1000 ml ($p < 0.01$ compared to the second lymph sample).

duodenal lymph and venous plasma[12]. The results presented do not exclude that part of the pancreatic lipase in arterial blood plasma had originated from enzyme absorbed from the intestinal lumen into their blood and lymph vessels[11,12]. Maintenance of the serum lipase level by the intestines for a short period after pancreatectomy[13] might be due to the same mechanism.

Pancreatic lipase activity in duodenal lymph can originate from arterial blood plasma due to diffusion of the enzyme from blood capillaries into the duodenal interstitium and from there into the lymphatics, or also by direct absorption from the duodenal lumen into the lymphatics. A fact pointing to lipase absorption from duodenum into lymphatics was its significantly higher activity in duodenal lymph than in the corresponding arterial plasma samples, both under basal conditions and stimulated pancreatic secretion. It cannot be excluded that the lipase originating from the duodenum, as well as from the pancreas, might take part in the intralymphatic breakdown of triglycerides absorbed from the intestines.

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Reduction of bovine pulmonary hypertension by normoxia, verapamil and hexaprenaline[1]

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Summary. In calves with hypoxic pulmonary hypertension, resistance was reduced by $40 \pm 3\%$ with normoxia, $19 \pm 4\%$ with verapamil, and $60 \pm 1\%$ with hexoprenaline. It is possible that the increased resistance during normoxia is due partly to vasoconstriction rather than solely to vascular thickening, and that the vasoconstriction is due to an abnormality in calcium metabolism by the hypertensive vasculature.

Acute administration of oxygen to man or cattle with chronic, hypoxia-induced pulmonary hypertension results in only partial reduction of pulmonary arterial pressure[2–4]. Complete reversal of the hypertension requires prolonged exposure to alveolar normoxia[3,5]. The persistent elevation of pulmonary vascular resistance is generally considered to reflect luminal encroachment by vascular hypertrophy[6,7]. However, it has been suggested that a contractile abnormality of the vascular smooth muscle might also be involved[8,9]. If augmented smooth muscle tone accounts for part of the persistent hypertension, then potent pharmacologic vasodilators might elicit more smooth muscle relaxation and pulmonary vasodilation than does oxygen. The purpose of this study was to determine in cattle if either of the spasmolytics, verapamil, a calcium antagonist[10–13], or hexoprenaline, a β-adrenergic agonist[14,15], reduced chronic, hypoxia-induced pulmonary hypertension to a greater extent than did acute alveolar normoxia.

Methods. Cardiopulmonary variables were measured in 8, 4-month-old, unanesthetized, Hereford calves following catheterization as described previously[16]. Baseline measurements were made at the resident altitude of 1520 m ($P_B = 630$ mm Hg). The calves were then exposed in a hypobaric chamber to a simulated altitude of 4270 m ($P_B = 440$ mm Hg). After 2 weeks, the calves were re-