

Effect of inulin on α -amylase activity of the pancreas in rats with lead intoxication

Kuchkarova L.S, Kudeshova G.T.

National University of Uzbekistan, Uzbekistan

Resume

A weekly treatment of growing rats with inulin (200 mg/kg body weigh/24 h) at weekly lead acetate (5 mg/kg/body weigh/24 h) intoxication provided corrective and/or preventive effect on shifts of α -amylase activity in pancreas and intestinal content. Effect of inulin on α -amylase secretion in intoxicated rats depended on its treatment regime. It was most expressed when rats were treated with lead acetate and inulin simultaneously. Administration of inulin to rats before intoxication slightly prevented changes in α -amylase activity in pancreas and in intestinal content caused by lead acetate intoxication. However treatment of rats with inulin after intoxication only partly eliminated negative effects of lead in the activity of α -amylase in pancreas and intestinal content

Keywords

Growing rats, α -amylase, pancreas, intestinal contents. lead acetate, inulin.

It is known that prebiotic inulin is not split in the upper gastrointestinal tract but is fermented in the large intestine by symbiotic microflora. Inulin is prescribed to normalize enteral microflora and gastrointestinal tract function at immune and allergic deviations and intoxications to activate metabolism [4, 9]. However, data on the effect of inulin on the pancreas enzyme secretion at heavy metal intoxication is absent. In the current work the effect of inulin on pancreatic secretion has been studied on the base of α -amylase, one of the most reactive of pancreatic enzymes [8].

The research purpose is study the influence of inulin on the activity of α -amylase in the pancreas and intestinal contents of rats with lead intoxication

Material and methods

The experiment were performed on growing Wistar rats weighing 60 ± 5 g

The animals were divided into 5 groups - one control and four experimental. Control group rats orally received saline for two weeks every morning The first experimental rat group was given saline for a week, then it was treated with lead acetate also for a week (experiment 1). The second experimental group of rats was administered inulin for a week, followed by administration of lead acetate for a week too (experiment 2). Rats of the third group received saline for a week and then the following week rats received lead acetate first and in 30 minutes inulin (experiment 3). Rats of the fourth group during the first observation week were treated with lead acetate, and during the

second observation week were treated with inulin (experiment 4). In all groups, the same dose of lead acetate (5 mg/kg/24 h) and inulin (200 mg/kg body weight/24 h) was used. All drugs were administered orally. Rats were sacrificed after 2 weeks of observation in the morning hours between 9.00-10.00 a.m

The animal procedures were performed in accordance with International Guidelines of using Animals in Scientific Procedures

After decapitation and opening the abdominal cavity the pancreas was rapidly removed and freed of adhering fatty and connective tissue. Then it was weighed, homogenized for 1 min at 0°C in 9 times their weight of cold Ringer solution (pH – 7.3). The homogenate was centrifuged at 3000 g/min for 15 min at 0 °C.

The small intestine (from the pyloric end to the ileal-caecal region) was removed and trimmed of fat and mesentery. Then intestines were washed with 5 ml of cold Ringer's solution to the centrifuge tubes with known mass. Intestinal flushing with tubes was weighed to determine the chyme weight. Samples were carefully mixed and centrifuged at 3000 g/min for 15 min.

α -Amylase (α -1,4-glucan glucanohydrolase; EC 3.2.1.2) activity was determined by Ugolev [10] in the supernatant of pancreas and content of small intestine.

All results presented as mean \pm S.E. Difference between mean of experimental and control groups were evaluated by unpaired Student's t test where $P < 0.05$ considered as significant.

Results and discussion

Data on α -amylase activity level in the pancreas and intestinal content in control and experimental groups of rats are shown in Table 1.

Table 1

Effect of inulin on pancreatic α - amylase activity in growing rats with lead acetate intoxication
(M \pm m; n = 6)

Animal groups	Pancreas		Intestinal content	
	(g/min/g issue)	%*	(mg/min/ml)	%
Control	8,9 \pm 0,4	100.0	12,5 \pm 0,7	100.0
Experiment1 P	17,6 \pm 1,2 <0,02	197.8	5,6 \pm 0,2 <0,001	44.8
Experiment 2 P	12.9 \pm 1,2 <0,002	144.9	10.8 \pm 0,7 >0.1	86.4
Experiment 3 P	9,9 \pm 0,4 >0.5	111.2	10,0 \pm 0,5 >0.1	80.0
Experiment 4	13,2 \pm 1,0	148.3	8,4 \pm 0,6	67.2

P	<0,001		<0,001	
---	--------	--	--------	--

*% in relation to the control taken as 100

It is seen that after daily administration of lead acetate during a week (experiment 1) α -amylase activity was increased by 97.8% in the pancreas and decreased by 55.1% in the intestinal content. Intoxication of rats with lead acetate after a weekly treatment with inulin (experiment 2) resulted in increasing of pancreas α -amylase activity by 44.9%. It should be noted that the enzyme activity in rats treated with lead acetate after receiving of inulin, although was increased, but it was significantly lower than in rats treated only with lead acetate ($P < 0.01$). Enzyme activity in intestinal content of this group animals was at the control level.

Rats treated with inulin during toxicity of lead acetate (Experiment 3) did not show any significant changes in the activity of the enzyme in both biological samples. Treatment with inulin after intoxication of rats with lead acetate (experiment 4) resulted in increasing of α -amylase activity by 48.3% in the pancreas and decreasing enzyme activity by 32.8% in intestinal content compared to control group. It should be noted that although the level of activity of α -amylase in the pancreas of rats in the fourth experimental group was higher than in the control group, the enzyme activity was significantly lower than in rats treated only with lead acetate ($P < 0.001$). Similarly, the enzyme activity in the intestinal contents were lower than in control, but it was higher than in rats receiving lead acetate without additional inulin treatment ($P < 0.001$).

Consequently, inulin effect on pancreas secretion is depended on the treatment regime. In all regimes, the use of inulin warns and/or decreases shifts of α -amylase activity in the pancreas and intestinal content caused by lead acetate intoxication.

It is known, increased activity of digestive hydrolases in pancreas acini with parallel decreased activity of digestive hydrolases in the intestinal content is pancreas disorder symptom [8]. So, the redistribution of the activity of enzymes in rats with lead intoxication is associated with disturbance of pancreatic juice release into the duodenum.

It is believed that effect of lead on pancreas membrane cells is accompanied by metabolic disorders, damaging of cell membranes with the subsequent tissue destruction and forming of lipid peroxidation products [2]. In addition lead ions affect on intestinal microecology [1], which takes place in the etiology of pancreatitis [6].

Inulin corrective effect on pancreatic secretion, probably, occurs due to recuperation both the key points of metabolism and intestinal microbiota. It is recognized that inulin stimulates the growth and activity of colon bacteria, which have a wide influence range almost on all processes in the body, including secretion of digestive glands [4, 7, 9]. Inhibition by inulin the processes of forming reactive oxygen forms and inhibition of lipid peroxidation products [3] shows that inulin

has antioxidant properties.

Hence, being a prebiotic and antioxidant, inulin has a corrective effect on pancreas secretion at lead intoxication. The effectiveness of anti-toxic effects of inulin depends on its use regime. The most corrective inulin effect is seen during a weekly treatment of animals with both lead acetate and inulin at the same time. Inulin, consumed before lead acetate intoxication, slightly prevents shifts in α -amylase activity in biological samples. Effect of inulin consumption after lead intoxication on pancreatic secretion of rats is weaker than its use before the intoxication.

Conclusion

1. Inulin has a corrective effect on damaged α -amylase secretion caused by lead intoxication in growing rats
2. Effect of inulin on the activity of α -amylase in the pancreas and intestinal content in rats with lead intoxication depends on the regime of its application.

Reference

1. Breton J.1., Le Clère K., Daniel C., Sauty M., Nakab L, Chassat T., Dewulf J., Penet S., Carnoy C., Thomas P., Pot B., Nesslany F., Foligné B. Gut microbiota limits heavy metals burden caused by chronic oral exposure. //Toxicol. Lett. – 2013. – V. 222. – P. 132–138.
2. Chen Y.W., Yang C.Y., Huang C.F., Hung D.Z., Leung Y.M., Liu S.H. Heavy metals, islet function and diabetes development. // Islets.- 2009. - N1. – P.169–176.
3. Danilenko A.L., Mamcev A.N., Maksyutov R.R. Solov'eva E.A., Kozlov V.N.. Izuchenie antiokislitel'noj aktivnosti inulina v regimel'nyh test-sistemah // Tekhnologii 21 veka v pishchevoj, pererabatyvayushchej i legkoj promyshlennosti. – 2013. – № 7. – S. 142-149.
4. Gibson GR, Berry-Ottaway P, Rastall RA. Prebiotics: new developments in functional food. Oxford, UK: Chandos Publishing Limited, 2000.-145 p.
5. Grigor'eva YU. V. , YAkovenko EH. P. , Voloshejnikova, Ovsyannikova I. A. , Lavrent'eva S. A. Rol' izbytochnogo bakterial'nogo rosta v formirovanii klinicheskikh proyavlenij i razvitii strukturnyh izmenenij slizistoj obolochki dvenadcatiperstnoj kishki u bol'nyh hronicheskim pankreatitom. // Eksperimental'naya i klinicheskaya gastroehnterologiya. – 2010. - №11. – S. 29-34.
6. Leal-Lopes C, Velloso F.J., Campopiano J.C., Mari C., Sogayar M.C., Correa R.G. Roles of Commensal Microbiota in Pancreas Homeostasis and Pancreatic Pathologies // J. Diabetes Res. - 2015. – V. 6. -2015:284680.
7. Othman M., Agüero R., Lin H.C. Alterations in Intestinal Microbial Flora and Human Disease. // Curr Opin Gastroenterol. (Current Opinion in Gastroenterology. - 2008. – V.24, N.1. – P. 11-16.
8. Serebrova S.Yu. Hronicheskij pankreatit: sovremennyj podhod k diagnostike i lecheniyu // Russkij Medicinskij Zhurnal. Bolezni organov pishchevareniya. – 2008. – T. 10. – № 1. – S. 30.
9. Shenderov B.A. Sovremennoe sostoyanie i perspektivy issledovanij v oblasti mikrobnnoj ehkologii cheloveka. // Klinicheskoe pitanie. — 2007. — №1-2. — S. A75-A76.

10. Ugolev A.M. Opredelenie amiloliticheskoj aktivnosti // Issledovanie pishchevaritel'nogo apparata u cheloveka - L.: Nauka, 1969: 187–192.