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# Effect of Pectin on the Activity of Pancreatic α-Amylase in Growing Rats with Lead Intoxication

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**Abstract:** This study was conducted to know the effect of pectin on the α-amylase activity in the pancreas, intestine content and blood in growing rats with lead intoxication. After a single oral administration of lead acetate (80 mg/kg) to growing rats pectin was administered (400 mg/kg/12h) for 4 days. It is revealed that the administration of lead acetate causes an increase in pancreas weight, protein content in the pancreas and α-amylase activity in the pancreas and blood and reduced activity of the enzyme in the intestinal cavity in rats. Regular oral administration of pectin has a normalizing effect on pancreatic weight, protein content and on the level of the activity α-amylase in the pancreas, intestinal contents and blood in the 48-72 hours of observation in rats. These results support our suggestion about the possibility of the use of pectin for correcting of pancreatitis similar shifts at lead intoxication.

**Keywords:** Rat, Lead Acetate, Pectin, α-Amylase, Pancreas

## 1. Introduction

Pectin is heteropolysaccharide consisting of mainly residues of galacturonic acid. This soluble fiber is present in all higher plants, especially in fruits. Due to its properties pectin swells in the gastrointestinal tract, forming a gelatinous mass that adsorbs undigested food residues, binds and removes cholesterol and other toxic substances and waste products from the body, improves blood circulation and intestinal peristalsis [1-4].

Pectin has a wide range of positive effects on the regulation of metabolism, cleansing the body of toxins and slags, normalizing intestinal microflora [1, 3]. Pectin, unlike other food additives is resistant to digestion and absorption in the small intestine and completely or partially fermented in the colon [5]. Pectin is able to absorb and excrete toxins, anabolic steroids, xenobiotics, cholesterol, bile acids, urea, bilirubin, serotonin, histamine, mast cell products and other biologically harmful substances: that accumulate in the body [1, 2]. The presence reactive alcoholic and carboxyl groups in pectin promotes the formation of stable complexes with metals [5].

In the digestive tract as a result of pectin slitting polygalacturonic acid is formed. The acid connects with ions of lead, cadmium, mercury, forming insoluble salts. These salts are not absorbed from the cavity of the gastrointestinal tract and excreted with feces [2, 3].

In the last decades among the most dangerous heavy metals, lead is known to have toxic effects on various organs [6-12]. Prolonged toxicity of this metal is associated with the fact that the lead is able to accumulate in the body. The half-life of lead is more than 20 years in the human bones [13]. In addition to the negative effects on the metabolic processes lead has carcinogenic and mutagenic effects [6, 8]. The effects of lead exposure can occur even in the next generations [9, 14-16]. Lead is toxic for almost all organs. It has a significant negative impact on the nervous system [11, 17], bone [14, 18-19], kidney [20] heart [21] and blood [11, 12]. Hypothalamic-pituitary system [9, 10], adrenal glands [22], thyroid gland [22, 23], gonads [24] also suffer from the lead in the body

Effect of lead also extends to the digestive organs: intestine [25], liver [26, 27, 28], submandibular gland and

pancreas [29, 30]. Activity of digestive enzymes particularly pancreatic enzymes is very exposed to the effects of various endogenous and exogenous factors [16, 30]. It is noted that among the pancreatic enzymes α-amylase activity are extremely reactive to exposure of environmental factors [30-33].

# 2. Objective

The study of the effect of pectin on the activity of α-amylase in the pancreas, intestine content and blood in growing rats with lead intoxication.

# 3. Materials and Methods

### *3.1. Animals and Treatment*

The experiments were performed on white rats weighing about 60 ± 5 g bred in vivarium of Biological faculty of the National University of Uzbekistan. Growing rats were kept in similar plastic cages (8-10 rats/cage). The rats were kept on a standard vivarium diet consisting of cereals and vegetables under the natural light-dark regime at room temperature with free access to food and water.

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

The animals were divided into three groups: two experimental and one control groups. In the first experimental group (experiment 1) rats were treated once by lead acetate at a dose of 80 mg/kg orally and then were given every 12 hours saline by the same way. In the second experimental group (experiment 2) rats received a single oral administration of lead acetate (80 mg/kg body weight), then were orally administered pectin (Cargill pectin PG DS, (Germany) in a dose of 400 mg/kg/12 h to end of the experiment. Such dose of pectin was effective in the reduction of red blood indices at lead intoxication in rats [34]. The control animals were treated with saline every 12 hours orally. Rats were sacrificed at 24, 48, 72 and 96 hours after intoxication at the same time in the morning between 9.00-10.00 a.m.

### *3.2. Sampling Procedure*

After opening the abdominal cavity the pancreas was rapidly removed and freed of adhering fatty and connective tissue. It was then 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 ileoecal region) was removed and trimmed of fat and mesentery. Then intestine were washed with 5 ml of cold Ringer's solution to the centrifuge tubes with known weight. Intestinal samples in tubes were weighed to determine the chyme weight. Samples were carefully mixed and centrifuged at 3000 g/ min for 15 min.

Blood was obtained at rat decapitation and immediately centrifuged for 15 min at 3000 g /min.

### *3.3. Biochemical Analyzes*

α-Amylase activity was determined by Ugolev [35] in the supernatant of the pancreas homogenate, small intestine content and blood. The protein content in the supernatant of pancreatic tissue was determined by Lowry et al. [36].

### *3.4. Statistics*

All results are presented as mean ±S.E. Difference between mean of experimental and control groups were evaluated by unpaired Student's test with $P < 0.05$ considered as significant.

# 4. Results

The data on body and pancreas weight and pancreas protein content in the control and both experimental groups are given in Table 1. It is seen that body weight in animals treated with lead acetate (experiment I) tended to decrease in comparison with control group animals. This decreasing was statistically significant at the 96 hour of experiment. Body weight of rat treated with pectin after lead acetate intoxication (experiment II) was recorded at the control level throughout the four-day study (Table 1).

***Table 1.*** *Effect of lead acetate (experiment I) and pectin after lead acetate intoxication (experiment II) on the body weight, pancreatic weight and pancreas protein content in rats (M±m; n=10).*

| Experiment hours | Control | Experiment I | Experiment II |
|---|---|---|---|
| | **Body weight (g)** | | |
| 24P | 58.2±3.3 | 60.1±3.3<br>>0.5 | 57.9±3.6<br>>0.5 |
| 48P | 62.4±3.9 | 59.8±2.9<br>>0.5 | 58.2±3.9<br>>0.5 |
| 72P | 64.2±2.9 | 57.3±3.5<br>>0.1 | 63.2±3.5<br>>0.5 |
| 96P | 66.2±3.3 | 57.4±3.0<br><0.05 | 63.4±3.9<br>>0.5 |
| | Pancreas weight (mg) | | |
| 24<br>P | 58.1±3.4 | 65.3±4.1<br>>0.2 | 62.2±3.0<br>>0.4 |
| 48<br>P | 61.4±3.2 | 72.9±3.7<br><0.02 | 69.9±3.0<br><0.05 |
| 72<br>P | 65.2±3.3 | 82.3±3.5<br><0.001 | 63.2±3.5<br>>0.5 |
| 96<br>P | 68.7±3.3 | 86.4±3.0<br><0.001 | 65.4±3.9<br>>0.2 |
| | Protein content in pancreas (mg/g) | | |
| 24<br>P | 123.2±3.4 | 135.1±3.7<br><0.02 | 130.2±3.6<br>>0.2 |
| 48<br>P | 121.4±2.9 | 137.8±3.9<br><0.002 | 129.0±2.7<br><0.05 |
| 72<br>P | 120.7±4.1 | 140.4±3.2<br><0.002 | 126.1±3.2<br>>0.3 |
| 96<br>P | 122.7±3.7 | 149.0±3.1<br><0.0005 | 119.4±4.1<br>>0.25 |

Pancreas weight of rats treated with lead acetate was unchanged at 24-hour observation and increased to 18.7%; 26.2% and 25.8% in the 48-, 72- and 96-hours of experiment

respectively. Treatment with pectin of rat with intoxication significantly prevented the increase in weight of the pancreas caused by lead acetate. In intoxicated with lead acetate and treated pectin rats increase in the pancreas weight was observed only at the 48-hour observation

In the growing rats treated with lead acetate protein content in pancreatic tissue was increased by 9.6%; 11.8%; 16.3% and 21.4% at the 24-, 48-, 72- and 96 hours of experiment. However, in rats which received after lead acetate intoxication pectin, increase of the protein content in pancreas compared to control value was observed only at the 48 hour of observation.

Data on shift of α-amylase activity in the pancreas, intestinal content and blood are shown in Table 2.

***Table 2.*** *Effect of lead acetate (experiment I) and pectin after lead acetate intoxication (experiment II) on α-amylase activity in the rat pancreas, intestine content and blood (M±m; n=10).*

| Experiment hours | Control | Experiment I | Experiment II |
|---|---|---|---|
| | **Pancreas (g/min/g protein)** | | |
| 24 | 107.9±7.4 | 129.9±7.1 | 131.1±8.4 |
| P | | <0.05 | <0.05 |
| 48 | 107.9±.4 | 178.3±12.1 | 139.1±10.2 |
| P | | <0.001 | <0.05 |
| 72 | 99.4±8.3 | 159.1±8.1 | 119.2±6.9 |
| P | | <0. 001 | >0.1 |
| 96 | 112.4±7.2 | 143.3±8.1 | 118.4±6.3 |
| P | | <0.01 | >0.5 |
| | Intestinal content (mg/min/ml) | | |
| 24 | 10.2±0.8 | 5.6±1.5 | 12.1±0.7 |
| P | | <0.02 | >0.1 |
| 48 | 14.1±0.7 | 6.2±0.4 | 16.3±1.0 |
| P | | <0.01 | >0.1 |
| 72 | 12.4±1.0 | 7.6±1.2 | 14.6±0.9 |
| P | | <0.01 | >0.1 |
| 96 | 15.4±0.8 | 12.6±1.1 | 14.1±0.5 |
| P | | <0.05 | >0.2 |
| | Blood ((mg/min/ml) | | |
| 24 | 4.9±0.3 | 7.8±0.5 | 6.8±0.4 |
| P | | <0.001 | <0.001 |
| 48 | 4.8±0.4 | 7.1±0.5 | 5.3±0.5 |
| P | | <0.002 | >0.4 |
| 72 | 4.9±0.3 | 5.3±0.4 | 4.8±0.3 |
| P | | >0.4 | >0.5 |
| 96 | 5.1±0.4 | 5.4±0.3 | 4.4±0.4 |
| P | | >0.5 | >0.2 |

It is shown that pancreatic α-amylase activity was increased after a single injection of lead acetate to rats. Increasing of the enzyme activity in this group was 20.4%, 65.2%, 60.1% and 27.4% compared with the control at 24, 48, 72 and 96 experiment hours respectively. After the treatment with pectin of intoxicated rats the level of α-amylase activity was detected at the control level on the third day of observation.

The enzyme activity in the small intestine content, in contrast, was not increased, but decreased. Reduction of α-amylase activity in the intestinal content as compared to animals treated with saline was 45.1%; 56.0%, 38.7% and 18.2% by the end of the first, second, third and fourth days of observation, respectively, compared with the control. Oral up taking of pectin by rats treated with lead acetate solution resulted in a normalization of enzyme activity in the intestinal contents at 48 hour of observation.

In serum increasing of the enzyme activity was detected only in the first and second day of observation and was 59.2% and 47.9% respectively. Significant increase in α-amylase activity after oral administration to intoxicated rat pectin was observed only at 24 hour of observation.

Thus, the acute toxicity of lead acetate in rats causes a reduction in body weight, increase in the pancreas weight and protein content in the pancreas tissue. Further in intoxicated rats there are significant changes in the α-amylase activity in the pancreas, intestinal content and blood. Treatment rat with pectin after lead acetate consumption, quite soon results in to restoration of α-amylase activity to the level of control values in all biological samples.

# 5. Discussion

The obtained data completely confirmed possibility of using pectin as a food additive for correcting shifts occurring in the pancreas at an acute lead poisoning. It was revealed that after a single administration of lead acetate (80 mg/kg body weight) to growing rats there is a decrease in body weight, increase in the pancreas weight and pancreas protein content. In addition activity of α-amylase in pancreatic tissue and blood is increased but enzyme activity in intestinal content is decreased in intoxicated rats. Pectin treatment (400 mg/kg body weight/12 h) after intoxication completely restored the body and pancreas weight and pancreas protein content shifts as well as enzyme activity in pancreatic tissue, intestinal content and blood on the third day of observation.

Decrease of the body weight in rats exposed to lead intoxication have been also observed in other researches [27, 37, 38]. If it is considered that lead ions do not effect on food intake [27], the decrease in body weight gain in rats may be related to metabolic disorders associated with shift of zinc dependent enzyme activity [39]. Furthermore, exogenous lead results in reduction of levels of erythropoietin [40] and sex hormones [9] which have anabolic activity. An important role in reducing body weight in lead treated animals can play a decrease in the activity of brush border carbohydrases of the small intestine [38]. Decreasing of α-amylase activity in the intestinal contents (table 2) descreases polysaccharides hydrolysis in intoxicated animals. Lower α-amylase and possibly other pancreatic enzymes activity in the small intestine cavity in intoxicated rats decrease the hydrolysis of food polymers. Due to this decreasing formation and hydrolysis of oligomers by brush bolder enzymes is also limited [41]. In general, these changes contribute to decrease the common hydrolytic ability of the small intestine, which ultimately manifest in reducing body weight of animals treated with lead acetate.

In intoxicated rats treated with lead acetate was noted an increase of pancreas weight and specific protein content (Table 2). Caused by the treatment of lead acetate, these changes probably are signs of pancreatitis, accompanied by an increase in the volume and weight of the pancreas [42].

Increasing of α-amylase activity in the pancreas and blood also suggests the presence of pancreatitis similar shifts in rats received orally lead acetate. Probably along with an increase of α-amylase activity is an increase of other enzymes activity in the pancreas and blood in rats intoxicated by lead acetate. This phenomenon has been observed in models of experimental pancreatitis in animals [42]. Increasing of pancreas weight and volume contribute to the spasm of excretory tract, hypertrophied nuclei, expanding tubular of rough endoplasmic reticulum, swelling of mitochondria in the pancreas cells in acute lead intoxication [42, 43].

A significant reason for the increase of weight of the pancreas in acute lead intoxication can be reduced secretion of pancreatic juice into the lumen of the small intestine. This shows a decrease of enzyme activity in the intestinal contents with increased α-amylase activity and protein content in the pancreatic tissue (Table 2). In addition, increasing the protein content in the pancreas possibly linked with the accumulation of lead-protein complexes that are identified in lead poisoned rats [43]. Furthermore, such protein accumulation may be due to concentration of enzyme macromolecules in the tissue, which can be seen in α-amylase activity increasing in pancreas.

It is found that the pancreatic secretory cells not only secrete enzymes into the cavity of the small intestine, but also in blood. Thus, the pancreas acinar cells secrete major amount exocrine enzymes through apical membrane into the pancreatic duct and duodenal cavity. Some enzymes through the basolateral plasma membrane is transferred into the extracellular space, and thence into the lymph and blood capillaries [44]. This enzyme pathway into the blood usually is transcellular as intercellular contacts not permeable to proteins including the enzyme [45]. Intercellular transport of hydrolases occurs at disturbance of these contacts by intraductal high pressure, high enzyme activity or other damage to the gland tissue [30, 46].

It is natural to suggest that the repression of pancreatic juice secretion under the effect of lead acetate results in accumulation of pancreatic juice in the acinus cavity and pancreatic ducts, causing an increase of volume and pressure in the pancreas. As a result of high pressure increase secretion of pancreatic enzymes in the blood occurs. Increased of α-amylase activity in the blood in rats treated with lead acetate (Table 2), can be also the result of hypersecretion of pancreatic juice into the blood stream, due to spasms of pancreatic duct [47]. Thus, increasing of α-amylase activity in blood and pancreas tissues on the background of decreasing enzyme activity in the intestinal contents after lead intoxication of rats shows pancreatitis similar shifts in the pancreas.

Currently, for correction of lead intoxication widely used herbal chelators. Among them a special place belongs to the pectin [4, 5]. It is shown that pectin stimulates the production of short-chain fatty acids and has a trophic effect on mucosa of small intestine, colon and rectum, promoting stimulation of protein synthesis in these tissues [3, 27]. One important property of pectin is its ability to form complexes with heavy metal ions especially with lead ions [48]. The compounds of heavy metals with pectin due to insolubility is not absorbed and excreted from organism. [1, 2, 48].

The results showed that the introduction of pectin to intoxicated rats prevents body weight loss caused by administration of a toxic dose of lead acetate. Besides the regular treatment rat with pectin treats pancreatitis similar shifts in the pancreas. This is manifested in the normalization of the pancreas weight, pancreas protein content, α-amylase activity in the pancreas, intestinal content and blood after treatment with pectin of intoxicated rats.

Pectin binds and eliminates lead from the liver, cavity of the stomach and intestine [48]. Besides pectin cleanses the blood of the products of lipid peroxidation, cholesterol and other toxins and slags [34, 49]. It is known its beneficial effect on blood creation and anemia correction [34, 50]. It is shown that low-esterified pectin exhibits antioxidant activity [49], causing normalization level of malonic dialdehyde and the activity of glutathione reductase and glutathione peroxidase in the rat liver at lead poisoning [49]. The amount of lipid peroxidation products is increased under the action of lead [51], and as a result of experimental pancreatitis [52, 53]. In intoxicated with lead acetate and treated with pectin rats normalization of the secretory pancreas function takes place probably due to excretion of lead and products of lipid peroxidation by pectin.

It has been shown that treatment of rats with pectin leads to intestinal hyperplasia and a substantial increase in the ileum brush border enzyme activity [54, 55] Thus the trophic effect of pectin on the ileum mucosa increases the ability of the small intestine to absorb various nutrients. It is possible that in the restoration of body weight in intoxicated rats treated with pectin occurs pectin trophic effect on the mucosa of the small intestine.

Thus summarizing the results and literature data, it can be concluded that the use of food pectin has a correcting effect on pancreatitis similar shifts caused by lead intoxication. Pectin correcting effect is depends on both by the delay of lead absorption into blood circulation, and by correcting key metabolic disorders [25, 49]. The appointment of pectin in acute lead intoxication can be used for the correction of pancreatitis similar shifts of growing organism.

# 6. Conclusion

Oral administration of lead acetate to growing rats (80 mg/kg body weight) results in pancreatitis similar shifts in the pancreas. It is revealed in increasing of the pancreas weight, protein content in the pancreas and α-amylase activity in the pancreas and blood, as well as in decreasing the enzyme activity in the small intestine cavity. Oral administration pectin (400 mg/kg body weight/12 h) to intoxicated rats has a normalizing effect on the pancreas weight, protein content and level of α-amylase activity in the pancreas, intestinal contents and blood.

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