

***Utilization of wheat bran as by- product to produce pectin to be effective on lead acetate toxicity in rats***

***Amira M. El-Moslemany***

*Nutrition and Food Science Department,  
Faculty of Home Economics, Al-Azhar University, Egypt*

***Abstract***

This study was carried out to be use wheat bran (rough and soft) as aby-product to produce pectin to be effective on lead acetate toxicity in rats. The biological experimental was design using male rats (36 rats) divided into six groups and it was fed on basal diet for four weeks. The first group (n= 6 rats) was administered plain water considered as negative control. The second group (n= 6 rats) was taken 0.13% lead acetate in drinking water considered as positive control. The third and fourth groups (n= 6 rats for each) was taken orally pectin 200 and 400 mg/kg/day prepared from rough wheat and it was taken 0.13% lead acetate in drinking water. The fifth and sixth groups (n= 6 rats for each) was taken orally pectin 200 and 400 mg/kg/day prepared from soft wheat) and it was taken 0.13% lead acetate in drinking water. At the end of experimental period (30 day) complete blood picture, Red blood cells (RBCs) and white blood cells (WBCs) were measured. The lipids profile, serum liver enzymes activity, kidney functions and pancreatic function were determined and also histopathological was examined in the liver. The results showed that the lead acetate was significantly induced decreased in RBCs, WBCs counts, blood hemoglobin, hematocrit and platelets. In the

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contrary, liver enzymes, kidney function and also pancreatic enzymes were significantly increased in positive control rat groups compared to negative control. Meanwhile, all lipid profile decreased in the positive group as compared to the negative control group, except LDL-c. Pectin from rough and soft wheat bran were significantly decreased the effect of lead acetate on the tested parameters. Histopathological examination clearly indicated that pectin from rough and soft wheat bran were eliminated from the harmful effect of lead acetate on liver tissues. From the obviously results it could be recommended that the pectin from rough and soft wheat bran have advantageous effects and it could may be able to become hostile for lead toxicity. Moreover, it was contributed to fast elimination on the toxicity and removed it from the blood. It could be recommended that pectin from rough and soft wheat bran has highly effect to binding the lead and should improvement of human nutrition to lowering the lead contamination in food and water.

### ***Introduction***

In the last decades, the amount of contamination has increased by lead ions ( $Pb^{2+}$ ) and it was becoming an important problem in public human health. This contaminant can be present in the atmosphere, rivers, soil, etc., and represents a dangerous warning to public human health, living materials, and environmental systems, not only but also due to its true high toxicity and accumulation effects (***Cao et al., 2015***). In addition, there are cases where people are in direct contact with lead through the skin or by ingestion of lead-based objects such as batteries (***Maikoe and Van Rijn, 2010***). A large amount of devices commonly used these days (including gadgets) are powered by batteries, and most of these

batteries are based on heavy-metal ions (cadmium, mercury, chromium, etc.) including lead.

**Metwally et al., (2015)** found that the lead is toxic for virtually all organs of the body and the liver is considered as one of the target organs affected by lead toxicity due to founds of site the storage in the liver after exposure to lead toxicity. Moreover, the liver is one of the major organs in detoxification of toxic materials. The lead is stored in soft tissues at most in the liver through the portal vein which that it can be used to examine the histological analysis and the morphological changes that reflect possible lead effects on somatic cells.

**Silbergeld et al., (2000)** observed that the lead acetate can begin in an action to inhibitions the activities of antioxidant enzymes. Furthermore, found of oxygen free radical increased the level of lipid oxidation and reduction of phytochemical. All of them which were the major contributors to lead-exposure related diseases (**Patrick, 2006**). Lead is known to cause histological liver damage, resulting in increased liver enzyme levels which results in DNA damage (**Zhang et al., 2004**).

Pectin is heteropolysaccharide consisting of mainly residues of galacturonic acid as a soluble fiber is present in all higher plants. Pectin can be swells in the gastrointestinal tract, forming a gelatinous mass that adsorbs undigested food residues and removes cholesterol and other toxic substances and waste products from the body, improves blood circulation (**Thakuret et al., 1997**).

Pectin has a wide range of positive effects on the regulation of metabolism, cleansing the body of toxins and slag, normalizing

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intestinal microflora (*Hotimchenko et al., 2005*). Pectin is able to absorb 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 (*Ovodov, 2009*).

Cholesterol level and blood pressure were reduced by soluble fiber as pentosans, pectin, gums, and mucilage. Moreover, soluble fiber performs important physiological functions, prevents gastrointestinal problems, protects against the onset of several cancers, and increases mineral bioavailability (*Chawla and Patil, 2010*).

The present study was carried out to investigate the effect of two levels of pectin isolated from wheat bran on rats suffering from toxicity by lead acetate.

### ***Material and Method***

#### **Materials:**

Pure lead acetate  $[(\text{CH}_3 \text{COO})_2 \text{Pb} \cdot 3\text{H}_2\text{O}]$  was purchased from Sigma Chemical (St. Louis, Mo). Lead acetate was dissolved in distilled water.

Casein, corn oil, corn starch, sucrose, cellulose, salt mixture and vitamin mixture were obtained from the Cairo Company for Chemical Trading, Cairo, Egypt.

Male Wistar adult rats (36 rats) weight ranging 170-180 g were purchased from the animal colony, Helwan farm, Vaccine and Immunity Organization, Ministry of Health, Cairo- Egypt.

BIOMERIEUX kits were obtained from Randox Laboratories Ltd., Diamond Road, Crumlin Co., Antrim, United Kingdom, BT294QY.

Wheat seeds were purchased from local market. Wheat seeds were milled in Laboratory Quadramate Mill Junior at different mesh to give soft wheat bran (white and red wheat bran) and rough wheat bran.

#### **Methods:**

##### **Extraction of pectin from wheat bran**

The soft and rough wheat bran was washed with water (1:1) for five minutes at 22°C, centrifuged at 860 g until total liquid drainage and dried overnight in an air circulated oven at 70°C. The dried bran was ground through 60 mesh size and stored in polyethylene bags at room temperature. Pectin from wheat bran was extracted according to the experimental conditions reported by *Fertonaniet al., (2006)*, with a nitric acid (HNO<sub>3</sub>) solution (solid-liquid ratio 1:40) adjusted to 100 mM, at the boiling point for 10 min. Pectin was isolated after cooling to 4°C by ethanol 66%(v/v) precipitation air-dried in a ventilated oven at 50°C and stored until use.

##### **Chemical analysis of pectin**

Moisture and ash content of pectin prepared from soft and rough wheat bran were determined according to *AOAC (2005)*. Meanwhile, pH value of 1% from different pectin was measure using pH meter Model CG710 West Germany.

##### **Physical characteristics of pectin**

##### **Determination of equivalent weight**

Equivalent weight was determined by *Ranganna's method (1995)*. Pectin sample 0.5 g was taken in a 250 ml conical flask and 5 ml ethanol was added. Sodium chloride (1.0g) and 100 ml of distilled water were added. Finally 6 drops of phenol red was added and

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titrated against 0.1 N NaOH. Titration point was indicated by purple color. This neutralized solution was stored for determination of methoxyl content. Equivalent weight was calculated by following equation:

$$\text{Equivalent weight} = \frac{\text{Weight of sample}}{1000 / \text{ml alkali} \times \text{Normality alkali}}$$

### Determination of Methoxyl content (MeO)

Determination of MeO was done by using the *Ranganna's method (1995)*. The neutral solution was collected from determination of equivalent weight, and 25 ml of sodium hydroxide (0.25 N) was added. After 30 min 25 ml of 0.25 N hydrochloric acid was added and titrated against 0.1 N NaOH. Methoxyl content was calculated by following equation:

$$\text{MeO} = \frac{\text{ml alkali} \times \text{Normality alkali} \times 3.1}{\text{Weight of sample}}$$

### Determination of Total Anhydrouronic Acid content (AUA)

Total AUA of pectin was obtained by the following equation (*Mohamed and Hasan, 1995*).

$$\text{AUA \%} = \frac{176 \times 0.1 z \times 100}{w \times 1000} + \frac{176 \times 0.1 y \times 1000}{w \times 1000}$$

When molecular unit of AUA (1 unit) = 176 g

Where, z = ml (titration) of NaOH from equivalent weight determination.

y = ml (titration) of NaOH from methoxyl content determination.

w = weight of sample

**Determination of Degree of Estrification (DE)**

The DE of pectin was measured on the basis methoxyl and AUA content (*Owens et al., 1952*) and calculated by following equation:

$$\text{DE \%} = \frac{176 \times \% \text{ MeO}}{31 \times \% \text{ AUA}} \times 100$$

**Determination of neutral sugars**

Neutral sugars were determined according to *Miller (1959)*; pectin powder (0.5 g) was taken into a volumetric flask and made up 10 ml by adding distilled water and filtrate. One ml filtrate was transferred into a test tube and added 3 ml of dinitrosalicylic acid solution was added and boiling for 15 min and it was cooled with tap water. Then the absorbance was taken at 480 nm in the Unicam spectrophotometer (UV-120-OI- Shimadzu). Calibration of standard curve was drawn using known different concentrations glucose.

**Biological investigation:**

Male Wistar adult rats (36 rats) weight ranging 170-180 g were purchased from the animal colony, Helwan farm, Vaccine and Immunity Organization, Ministry of Health, Cairo- Egypt. Rats were housed in individual cages with screen bottoms and fed *ad libitum* on a basal powdered diet appropriate for growing the rats which containing casein (20 %  $\geq$  85% protein), corn oil (8%), corn starch (31%), sucrose (31%), cellulose (4%), salt mixture (4%) and vitamin mixture (1%) according to *Pell et al. (1992)*.

After feeding on basal diet for eight days, rats were divided into six groups and it was fed on basal diet for four weeks. The first group (n= 6 rats) was administered plain water considered as negative control. The second group (n= 6 rats) was taken 0.13% lead

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acetate in drinking water considered as positive control. The third and fourth groups (n= 6 rats for each) was taken orally pectin 200 and 400 mg/kg/day prepared from rough wheat and it was taken 0.13% lead acetate in drinking water. The fifth and sixth groups (n= 6 rats for each) was taken orally pectin 200 and 400 mg/kg/day prepared from soft wheat) and it was taken 0.13% lead acetate in drinking water. Each rat was weighted every two days and the food consumption was calculated. Biological evaluation of the different diets was carried out by determination of body weight gain % (BWG %) feed efficiency ratio (FER) (*Chapman et al., 1959*).

At the end of experimental period (30 day)the blood samples were taken with drawn from the orbital plexus and centrifuged at 3000 rpm to obtain the sera. After that, the serum was kept on a deep-freezer at -20°C until their analyses. Complete blood picture as hemoglobin (Hb), hematocrite (Ht) and platelets were determined using a whole blood sample and the method described by *Dacie and Lewis (1984)* respectively. Red blood cells (RBCs) and white blood cells (WBCs) were measured as recommended by *Riley (1960)*.

The levels of serum total lipids, total cholesterol and triglycerides were determined according to *knight et al. (1972)*, *Allain et al. (1974)* and *Fossati and Prencipe (1982)*. High, low and very low density lipoprotein- cholesterol in serum was determined according to *Lopes-Virella et al. (1977)* and *Steinberg (1981)*.

Serum liver enzymes activity, aspartate amino transferase (AST) and alanineamino transferase (ALT) were determined according to *Bergmeyer et al. (1986)*. Alkaline phosphatase was determined according to *Moss and Henderson (1999)*. Kidney functions as uric acid, creatinine and urea were determined according

*to Barham and Trider (1972), Bartleset al. (1972) and Fawcett and Soctt (1960).*

Pancreatic function as blood sugar,  $\alpha$ -amylase enzyme and lipase enzyme were determined according to *Trinder (1969)*, *Lorentz (2005)* and *Lorentz (1998)*, respectively.

#### **Histopathological examination**

Liver pieces preserved in 10% formaldehyde solution were used for histopathological study. The liver tissues were placed in plastic cassettes and immersed in neutral buffered formalin for 24 h. The fixed tissues were processed routinely, embedded in paraffin, cut into 4 mm-thick sections and stained with hematoxylin and eosin (H&E) according to *Bancroft et al. (1996)*. The extent of lead acetate-induced hepatic damage was evaluated by assessing the morphological changes in the liver sections.

#### **Statistical analysis:**

The obtained data were exposed to analysis of variance. Duncan's multiple range tests at ( $P \leq 0.05$ ) level was used to compare between means. The analysis was carried out using the PRO ANOVA procedure of Statistical Analysis System (*SAS, 2004*).

## **Results and Discussion**

### **Physic-chemical characteristics of pectin isolated from wheat bran.**

The Physic-chemical characteristics of pectin isolated from soft and rough wheat bran were determined and the results are reported in Table (1). Premature sample showed the lower moisture content of isolated pectin from soft and rough wheat bran (10.38 and

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10.33%) than *Ismail et al., (2012)* who reported that the growth of microorganisms and production of pectinase enzymes which can affect the pectin quality may be due to high moisture content in the pectin (*Muhamadzadeh et al., 2010*).

Moreover, ash content was lower for isolated pectin 1.89% from soft than rough wheat bran was 1.97%. The good criteria for purity of the pectin showed by *Ismail et al., (2012)* who found that the ash content less than 10% and the maximum ash content was 10%. Therefore, in the present study the ash content is recorded the purity of the pectin due to the pectin had contained low ash content. These results are agreement with *Mansoor and Proctor (2001)* who found that the pectin at pH 3.5 is contained of powerful gel net work, can may be caused to a balance between hydrophilic and hydrophobic character. From our experimental study the pH value is found  $3.0 \pm 2$  this indicates that the pectin is purity and good desirable balance.

The physical characteristics of isolated pectin from soft and rough wheat bran, the results showed that the Methoxyl content was more than 7%. These results are agreement with *Food Chemical Codex (1996)* who reported that theMethoxyl content of standard pectin used in the industry is not less than 7%. Also, degree of Estrification referring to ratio of carboxyl group esterifies with methanol to the free carboxyl groups. Degree of Estrification for soft and rough wheat bran pectin were 57.24 and 59.73%, which indicated that the highest solubility of wheat bran pectin may be due to the presence of low levels of ester groups.*Sundar Raj et al., (2012)* Degree of Estrification in fact depends on variety and stages of maturity.

From the results in this study showed that the highest AUA content was found in soft and rough wheat bran pectin (69.34 and 72.45%, respectively). This resultant is agreement with **Food Chemical Codex (1996)** reported that the extract of anhydrogalactouronic acid (AUA) from the pectin less than 65%, the pectin is highly purity. Natural sugars for soft and rough wheat bran pectin were 8.12 and 7.38%, respectively. Pectin had contained less sugar and high molecular weight to give a greater gelling ability than that pectin has consists of high sugar and low molecular weight (*Phatak et al., 1988*).

**Effects of pectin isolation from wheat bran on body weight gain and feed efficiency ratio in rats suffering from toxicity with lead acetate.**

The results from Table(2) indicated that the effect of pectin isolation from wheat bran on initial, final body weight and feed efficiency ratio in rats suffering toxicity from lead acetate. From our results it could be observed that the normal negative control group was fed on basal diet had the highest in final body weight (194.5 g, increased in gain body weight 27.0 g) and feed efficiency ratio (5.84) at the end experimental period (four weeks). While, the positive control group was fed on basal diet and 0.13% lead acetate in drinking water the lowest significantly increased in final body weight (177.0 g increased 6.7g about initial body weight) and feed efficiency ratio was 1.92 than control negative and other groups. Meanwhile, the rat groups fed on basal diet and 0.13% lead acetate in drinking water and pectin isolation from wheat bran (rough and soft) was taken separately orally per day at levels 200 and 400 mg/ kg was observed that slightly significantly changes in final body weight and total feed intake between them. The results revealed that lead induced a higher significant decrease in the body weight gain and

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food efficiency ratio. This is in accordance to *El-Nekeety et al., (2009)* who reported that mean body weight of the animals treated with lead acetate was significantly lower than that of the other groups, many others studies have suggested that the decrease of weight according to dose administrated and the time of exposition and appeared for feed intake in rat (*Duane et al., 2008*).

### **Effects of pectin isolation from wheat bran on complete blood picture in rats suffering from toxicity with lead acetate.**

Effects of pectin isolation from wheat bran on complete blood picture as hemoglobin (Hb), hematocrite (Ht) red blood cells (RBCs), white blood cells (WBCs) and platelets were determined in rats suffering from toxicity with lead acetate and the results are reported in Table (3). From the results it could be noticed that all parameters increased significantly in the blood negative control that of positive control group ( $p < 0.05$ ). *Harbison (1998)* found that complete blood picture caused development of basophilic stippling and Howell-Jolly bodies, which are features of anemia due to lead intoxication. Moreover the control negative showed that lowered RBC count is other concordant hematological change was found in the group which lead acetate was administrated. RBCs have also been attributed to decrease in copper metabolism and iron consumption (*Sandhir et al., 1994*).

*Simsek et al. (2009)* found that the complete blood pictures values were significantly lowered in rats group taken to lead acetate than control group. Moreover, *Ancheva et al. (2003)* illustrated that lead be causing harmful to the RBCs membrane resulting in the rupture and destruction of red blood cells caused lead acetate was taken orally and also, decrease of blood iron level which may be the cause of decreased concentration of haemoglobine and hematocrit

value which caused the development of hypochromic anemia and hemolytic anemia.

Meanwhile, the all different groups were taken orally with 200 and 400 mg/kg/day pectin isolated rough and soft wheat bran, the resultant illustrated that the pectin isolated rough wheat bran give a good results nearly negative control followed by the results from pectin isolated from soft wheat bran. Pectin has been observed that controlling the absorption of lead (*Canfield et al., 2003*).

In some cases, orally administered pectin contributed to increased retention of heavy metal in tissues, in blood serum levels and in 24- hour urine collection, of children between the ages of 5 and 12 years (*Zhao et al., 2008*).

#### **Effects of pectin isolation from wheat bran on lipid profile in rats suffering from toxicity with lead acetate.**

The results from Table (4) showed that all lipid profile decreased in the positive control group, except LDL-C, as compared to the negative control group. Triglyceride drop in the treated birds may result from damage to the small intestine villi and impaired absorption of fatty acids. Moreover, damage to the liver may reduce the synthesis of triglyceride (*Hamidipour et al., 2016*). The progression of atherosclerosis and cardiovascular diseases are may be caused lipid and lipoprotein abnormalities (*Chrysohoou et al., 2004*). From the result in the same table of this work, there were increased mean values of High Density Lipoprotein (HDL) and decreased Low Density Lipoprotein (LDL), were observed in pectin fed rats when compared to positive control. Treated rats which suffer from toxicity with the two levels from pectin which extract from rough and soft wheat bran led to improvement of all lipid profile, as

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compared to the positive control group. Pectin is heteropolysaccharide consisting of mainly residues of galacturonic acid. This soluble fiber is present in all higher plants. 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 (*Ovodov, 2009*).

The biologically harmful substances that accumulate in the body such as steroids, cholesterol, bile acids, urea, bilirubin, serotonin, histamine, mast cell products and other, the Pectin is absorb the toxins and all harmful materials (*Ovodov, 2009*).

### **Effects of pectin isolation from wheat bran on serum uric acid, creatinine and urea in rats suffering from toxicity with lead acetate**

The data presented in Table (5) show that the effects of 200 and 400 mg/kg/day pectin isolated from rough and soft wheat bran on serum uric acid, creatinine and urea in rats suffering from toxicity with lead acetate. The results observed that the serum uric acid, creatinine and urea in positive group were increased than control negative and it was decreased than control positive.

Previous studies have revealed that Pb exposure could affect the function of the kidney. Pb exposure could promote the onset of oxidative stress (*Alya et al., 2015*). The mean values of serum urea, creatinine and uric acid decreased significantly with the two levels of pectin, on the other hand these parameters decreased gradually with increasing the level of pectin. These results occurred by *Adeyemi et al., (2009)* who found two consequences cause of

kidney diseases. The first step is the failure to keep protein, amino acids, sugar, water and ions in the kidney.

The second step is failure to excrete urea and creatinine and their elevated levels in the blood due to the presence of lead might have caused impairment of the brush border epithelial cells and making them not allowing urea and creatinine to pass through. The overall effect of this may be impaired kidney function and caused kidney failure, (*Oloyede et al., 2003*). These results also agree with *El-Zoghby and Sitohy(2001)* who found that renal function decreased significantly by feeding rats with pectin which provides ion exchange activity of the free carboxyl groups, the more the number of free carboxyl groups in the molecule, the more intensive ion exchange capacities the pectin has. Therefore, pectin with the low degree of esterification helps to reduce the blood arsenic level and diminish its adverse effects.

#### **Effects of pectin isolation from wheat bran on serum liver enzymes in rats suffering from toxicity with lead acetate**

From the Table (6) it could be illustrated that there was a significant increase in the level of activity of serum liver enzymes, the mean value of aspartate amino transferase (AST) and alanine amino transferase (ALT) of positive control ( $130.12 \pm 6.76$  and  $65.95 \pm 2.98$  U/L respectively) increased that of control negative ( $82.11 \pm 2.67$  and  $40.60 \pm 3.76$  U/L respectively). *Todorvic et al., (2005)* reported that the accumulated lead in the liver can be directly harmful to the hepatocytes mainly by damaging the quality of the cell membrane and it was increased the activities of liver function enzymes was most likely a result of the hepatotoxic effect of lead. This result due to the lead taken orally was delivered to the liver through the blood circulation and go into the body circulation. This

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metal accumulates in the hepatic cells and causes structural disorganisation such as necrosis and hydropic degeneration in these cellular areas (*Ozkaya , 2016*).

Moreover, *Ozkaya et al. (2017)* reported that the lead had obvious effect on levels of liver function enzymes activities which were significantly elevated to a higher position therefore become significantly higher than those in the control groups.

However, a significant increase in the level activity of alkaline phosphatase (ALP) of control positive ( $196.24 \pm 9.55$  U/L) compared with control negative ( $120.21 \pm 7.85$  U/L). The ALP plays an important role in the metabolism of glycogen and can disable the phosphorylase enzyme therefore it can stimulate glycogen synthesis in the liver. Therefore, the inhibition of enzyme activity in liver associated with glycogen breakdown to provide required energy under stressful conditions reduces the rate of phosphorylation or prevents oxidative phosphorylation in respiratory chain (*Saha and Kaviraj, 2009*). Therefore, treatment of rats with pectin significantly reduced lead retention in tissues; these results suggest that pectin may be considered as perspective dietary compounds removing environmental lead from the body (*Serguschenko et al., 2007*).

Pectin binds and eliminates lead from the liver, cavity of the stomach and intestine (*Khotimchenko et al., 2007*). Besides pectin cleanses the blood of the products of lipid peroxidation, cholesterol and other toxins and slags (*Khasina et al., 2003*). It is known its beneficial effect on blood creation and anemia correction (*El-Nahal, 2010*).

**Effects of pectin isolation from wheat bran on pancreatic function in rats suffering from toxicity with lead acetate.**

Table (7) showed that the results effect of pectin isolated from wheat bran on pancreatic function as, blood sugar,  $\alpha$ -amylase enzyme and lipase enzyme in rats suffering from toxicity by lead acetate. From the resultant it could be noticed that the hypoglycemia in group rats suffering toxicity fed on 200 and 400 mg/kg/day pectin were taken orally than control positive hyperglycemia. The increase in glucose may be partly influenced by changes in the endocrine glands and an increase in cortisol, which consequently increases the metabolism of glucose glycolysis (*Levesque et al., 2002*). Increase blood glucose in treated poultry may have various reasons, such as lead poisoning, impaired carbohydrate metabolism, increase energy demand of cell, cellular ATP decrease or even decrease level of the acetylcholinesterase. The increase in blood glucose level or hyperglycemia indicates disturbances in the metabolism of carbohydrates, caused by an increase in liver glycogen breakdown (*John, 2007*).

The concentration of glucose in poultry treated with lead significantly increased. Glucose density adjusts by complex mechanisms hormones such as glucagon, insulin and other hormones, such as corticosteroids, epinephrine and thyroxine. However, environmental stress and tension could result in a significant increase in plasma glucose levels (*Agrahariet al., 2007*).

The data in the same table observed that decreasing of  $\alpha$ -amylase activity in the group rats suffering toxicity fed on 200 and 400 mg/kg/day pectin were taken orally than control positive may be caused decreasing polysaccharides hydrolysis in animals which become intoxicated due to lower  $\alpha$ -amylase enzyme and pancreatic

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enzymes activity in the small intestine in toxicity rats decreasing the hydrolysis of sugar complex in food by brush border enzymes (*Kostjuevich, 2008*). These changes give to lower the hydrolytic ability in the small intestine and it was caused reducing body weight of animals which was taken lead acetate by orally. Whilst, the results from lipase enzymes were parallel and occurred to the results from blood glucose and amylase enzyme.

It can be concluded that the uses of taken orally pectin 200 and 400 mg/kg/day prepared from rough and soft wheat every day for another four weeks has a correcting effect on complete blood picture, liver, kidney and pancreatic functions which caused by 0.13% lead acetate in drinking water. Pectin is a delay of the lead absorption into blood circulation (*Khasina et al., 2003*). Moreover, 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 (*Khasina et al., 2003*).

### **Histopathological experimental:**

#### **Effects of pectin on histopathological in rats suffering from toxicity with lead acetate.**

The lead toxicity is remains an important public health problem because of the large amount of different sources from household and environment. Therefore, the lead is a toxic heavy metal and it is effecting on the blood, kidney, liver and the stomach and the intestines tract (*Paoliello and De Capitani , 2005*).

The light microscopic examination of normal control group liver sections Figure (1) showed that normal structure of the liver. The unit of liver tissue is the classic hepatic lobule, which is surrounded

by connective tissue. In the center of hepatic lobule there is a central vein.

It is clear that from Figure (2) represented that the light microscopic examination of injured group which received 0.13% lead acetate drinking for four weeks there were extensive severe vascular degenerative changes in the hepatocytes. Few hepatocytes show granular degenerative changes. Moreover the same group Figure (3) showed that congestion of portal blood vessels accompanied by severe degenerative changes hepatocytes are vacuolated and enlarged, cytoplasm appears faintly stained and the nuclei are shrunken.

Figures (4) showed that the light microscopic examination of liver sections which received 0.13% lead acetate and 200 mg /kg<sup>-1</sup>, body weight from pectin isolated from rough wheat bran for four week showed that some hepatocytes show necrosis, others were suffer from granular derivative changes. Whereas, Figure (5) showed that the light microscopic examination of liver sections which received 0.13% lead acetate and 200 mg /kg<sup>-1</sup>, body weight from pectin isolated from rough wheat bran for four week showed that hepatocytes showed that the slight diffuse vascular degenerative changes, others were necrotic.

Figure (6) showed that the light microscopic examination of liver sections which received 0.13% lead acetate and 200 mg /kg<sup>-1</sup>, body weight from pectin isolated from soft wheat bran for four week showed that some hepatocytes show necrosis, others were suffer from granular derivative changes. Moreover, Figure (7) represented that the light microscopic examination of liver sections which received 0.13% lead acetate and 400 mg /kg<sup>-1</sup>, body weight from pectin

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isolated from soft wheat bran for four week showed that hepatocytes show slight granular derivative changes and others were necrotic.

### ***Conclusion***

The results revealed in increasing of the complete blood picture, liver, kidney and pancreatic functions in control positive may be due to lead acetate 0.13% in drinking water. Oral administration pectin (200 and 400 mg/kg/day) isolated from soft and rough wheat bran to intoxicated the different rats group has a normalizing effect on the complete blood picture, liver, kidney and pancreatic functions. The obviously results supported that the possible effect of pectin isolated from rough and soft wheat bran against lead acetate may be treated induced hepatic damage in rats

**Table (1):** Physic-chemical characteristics of isolated pectin from wheat bran.

Physic-chemical	Wheat soft bran	Wheat rough bran
Moisture	10.83	10.33
Ash content	1.89	1.97
pH value	3.0 ±0.2	3.0 ±0.2
Equivalent weight	490.21	529.34
Methoxyl content%	7.32	7.54
Anhydrogalactouronic acid %	69.34	72.45
Degree of Estrification %	57.24	59.73
Estrification %	41.79	46.2
Natural sugar %	8.12	7.38

**Table (2):** Effects of pectin isolation from wheat bran on body weight gain and food efficiency ratio in rats suffering from toxicity with lead acetate.

Groups	Initial body weight (g)	Final body weight (g)	Gain body weight (g)	Total food intake (g)	Food efficiency ratio
Control negative	167.5 ±7.6 <sup>b</sup>	194.5 ±6.6 <sup>a</sup>	27.0 ±1.25 <sup>a</sup>	462.7 ±26.0 <sup>a</sup>	5.84 ±0.2 <sup>a</sup>
Control positive	170.3 ±8.0 <sup>ab</sup>	177.0 ±7.0 <sup>b</sup>	6.7 ±0.51 <sup>c</sup>	348.6 ±28.3 <sup>b</sup>	1.92 ±0.6 <sup>c</sup>
Group 1	171.8 ±7.8 <sup>b</sup>	189.8 ±8.1 <sup>a</sup>	18.0 ±1.12 <sup>b</sup>	420.0 31.2 <sup>a</sup>	4.29 ±0.2 <sup>b</sup>
Group 2	173.2 ±7.6 <sup>b</sup>	193.3 ±9.5 <sup>a</sup>	20.1 ±1.14 <sup>b</sup>	430.4 ±15.4 <sup>a</sup>	4.67 ±0.4 <sup>b</sup>
Group 3	180.5 ±8.6 <sup>a</sup>	198.3 ±7.9 <sup>a</sup>	17.8 ±0.98 <sup>b</sup>	455.9 ±30.8 <sup>a</sup>	3.90 ±0.6 <sup>b</sup>
Group 4	175.3 ±6.7 <sup>b</sup>	195.1 ±8.4 <sup>a</sup>	19.8 ±1.03 <sup>b</sup>	450.5 ±22.3 <sup>a</sup>	4.39 ±0.2 <sup>b</sup>

Each value represents the mean ±SD. Mean followed by different superscript letters in each column are significantly different (p<0.05).

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**Table (3):** Effects of pectin on complete blood picture in rats suffering from toxicity with lead acetate.

Groups	Hemoglobin (g/dl)	Hematocrit (%)	Red blood cells (m/cm)	White blood cells (cm)	Platelets ( cm )
Control negative	12.7± 0.81 <sup>a</sup>	38.0± 2.42 <sup>a</sup>	6.97± 0.30 <sup>a</sup>	5.67± 0.93 <sup>a</sup>	753.3±37 .8 <sup>a</sup>
Control positive	9.50± 0.61 <sup>c</sup>	30.0± 1.91 <sup>c</sup>	4.59± 0.52 <sup>c</sup>	4.12± 0.81 <sup>c</sup>	552.3± 30.21 <sup>c</sup>
Group 1	10.70± 0.65 <sup>b</sup>	33.74± 1.73 <sup>b</sup>	5.56± 0.51 <sup>b</sup>	4.94± 0.76 <sup>b</sup>	650.1± 32.28 <sup>b</sup>
Group 2	12.15± 0.71 <sup>a</sup>	37.25± 1.64 <sup>a</sup>	6.54± 0.43 <sup>a</sup>	5.50± 0.59 <sup>a</sup>	710.2± 30.58 <sup>a</sup>
Group 3	10.11± 0.72 <sup>b</sup>	32.58± 1.79 <sup>b</sup>	5.15± 0.28 <sup>b</sup>	4.42± 0.62 <sup>b</sup>	610.1± 37.46 <sup>b</sup>
Group 4	11.98± 0.68 <sup>a</sup>	36.45± 1.83 <sup>a</sup>	5.92± 0.37 <sup>a</sup>	5.16± 0.49 <sup>a</sup>	695.3± 35.22 <sup>a</sup>

Each value represents the mean ±SD. Mean followed by different superscript letters in each column are significantly different (p<0.05).

**Table (4):** Effects of pectin on lipid profile in rats suffering toxicity lead from acetate

Groups	T. Lipid (mg/dl)	Triglycerides (mg/dl)	T. cholesterol (mg/dl)	HDL (mg/dl)	LDL (mg/dl)
Control negative	0.55 ± 0.03 <sup>a</sup>	186.3 ± 6.1 <sup>a</sup>	150.3 ± 1.1 <sup>a</sup>	95.7 ± 8.0 <sup>a</sup>	17.34 ± 1.56 <sup>d</sup>
Control positive	0.32 ± 0.17 <sup>c</sup>	107.7 ± 27.9 <sup>d</sup>	120.3 ± 6.5 <sup>c</sup>	47.3 ± 4.2 <sup>c</sup>	51.46 ± 20.2 <sup>a</sup>
Group 1	0.42 ± 0.97 <sup>b</sup>	140.38 ± 10.24 <sup>c</sup>	132.38 ± 7.16 <sup>a</sup>	73.94 ± 5.39 <sup>b</sup>	30.28 ± 2.43 <sup>b</sup>
Group 2	0.45 ± 0.15 <sup>b</sup>	160.64 ± 7.11 <sup>b</sup>	128.68 ± 5.24 <sup>b</sup>	77.35 ± 4.58 <sup>b</sup>	19.20 ± 2.67 <sup>c</sup>
Group 3	0.41 ± 0.68 <sup>b</sup>	130.19 ± 9.27 <sup>c</sup>	130.12 ± 6.35 <sup>a</sup>	72.26 ± 3.67 <sup>b</sup>	31.82 ± 2.94 <sup>b</sup>
Group 4	0.43 ± 0.34 <sup>b</sup>	150.69 ± 8.25 <sup>b</sup>	125.68 ± 11.27 <sup>b</sup>	75.34 ± 4.19 <sup>b</sup>	20.20 ± 1.99 <sup>c</sup>

Each value represents the mean ±SD. Mean followed by different superscript letters in each column are significantly different (p<0.05).

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**Table (5):** Effects of pectin on serum uric acid, creatinine and urea (mg/dl) in rats suffering from toxicity with lead acetate.

Groups	Urea (mg/dl)	Creatinine (mg/dl)	Uric acid (mg/dl)
Control negative	26.12 ±2.4 <sup>d</sup>	0.49 ±0.11 <sup>c</sup>	2.56 ±0.25 <sup>c</sup>
Control positive	42.26 ±3.0 <sup>a</sup>	1.17 ±0.24 <sup>a</sup>	5.73 ±0.61 <sup>a</sup>
Group 1	35.49±1.72 <sup>o</sup>	0.60±0.21 <sup>o</sup>	3.702±0.24 <sup>o</sup>
Group 2	29.56±1.56 <sup>c</sup>	0.52±0.15 <sup>c</sup>	2.82±0.29 <sup>c</sup>
Group 3	39.59±1.83 <sup>o</sup>	0.63±0.32 <sup>o</sup>	3.65±0.31 <sup>o</sup>
Group 4	33.96±1.94 <sup>c</sup>	0.55±0.22 <sup>c</sup>	2.91±0.45 <sup>c</sup>

Each value represents the mean ±SD. Mean followed by different superscript letters in each column are significantly different (p<0.05).

**Table (6):** Effects of pectin on aspartate aminotransferase (AST), alanine aminotransferase (ALT) and alkaline phosphatase (ALP) (U/L) in rats suffering from toxicity with lead acetate

Groups	AST (U/L)	ALT(U/L)	ALP (U/L)
Control negative	82.11±2.67 <sup>d</sup>	40.60±3.76 <sup>d</sup>	120.21±7.85 <sup>d</sup>
Control positive	130.12±6.76 <sup>a</sup>	65.95±2.98 <sup>a</sup>	196.24±9.55 <sup>a</sup>
Group 1	90.28±4.25 <sup>ab</sup>	48.29±2.57 <sup>ab</sup>	178.59±7.22 <sup>b</sup>
Group 2	85.64±5.12 <sup>c</sup>	43.58±3.11 <sup>c</sup>	169.38±6.94 <sup>b</sup>
Group 3	95.39±3.98 <sup>o</sup>	50.17±1.94 <sup>o</sup>	158.68±7.38 <sup>c</sup>
Group 4	88.82±4.87 <sup>ab</sup>	46.34±2.37 <sup>ab</sup>	144.37±4.51 <sup>c</sup>

Each value represents the mean ±SD. Mean followed by different superscript letters in each column are significantly different (p<0.05).

**Table (7):** Effects of pectin on pancreatic function in rats suffering from toxicity with lead acetate.

Groups	Blood sugar (mg/dl)	Amylase enzyme (U/L)	Lipase enzyme (U/L)
Control negative	176.3 ±5.72 <sup>d</sup>	1000.0 ±5.0 <sup>d</sup>	77.33 ±2.54 <sup>d</sup>
Control positive	219.3 ±3.84 <sup>a</sup>	3382 ±137.4 <sup>a</sup>	88.66 ±3.00 <sup>a</sup>
Group 1	185.12±4.25 <sup>b</sup>	2045±120.6 <sup>b</sup>	82.33±2.98 <sup>b</sup>
Group 2	180.28±5.16 <sup>c</sup>	1473±131.8 <sup>c</sup>	79.54±3.51 <sup>c</sup>
Group 3	187.36±3.28 <sup>b</sup>	2082±125.9 <sup>b</sup>	83.68±2.74 <sup>b</sup>
Group 4	182.68±4.59 <sup>c</sup>	1538±128.7 <sup>c</sup>	80.67±4.11 <sup>c</sup>

Each value represents the mean ±SD. Mean followed by different superscript letters in each column are significantly different (p<0.05).

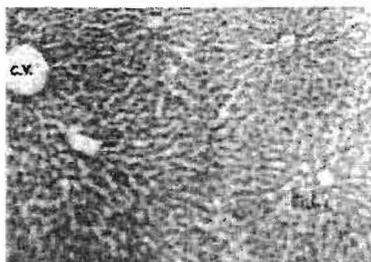


Fig. (1): Liver of rats from negative control, untreated group showing normal central vein and normal hepatocytes.

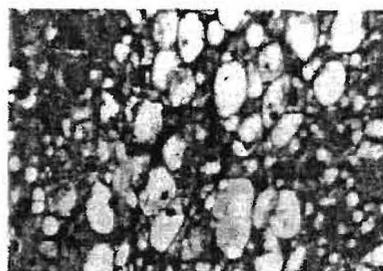


Fig. (2): Liver of rats from positive control receiving 0.13% lead acetate showing granular degenerative changes (H. E. X:650).

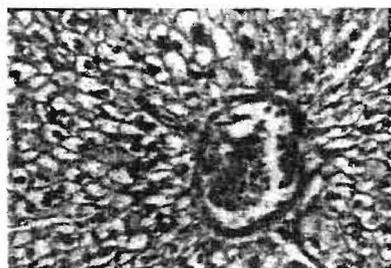


Fig. (3): Liver rats in positive control showing congestion of portal blood vessels, accompanied severe changes changes of hepatocytes (H.E.X:400).

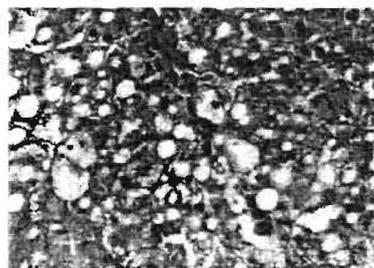


Fig (4): Liver rats received 200mg /kg/day body weight from pectin isolated from rough wheat bran showing vascular of some hepatocytes (H. E. X:650).

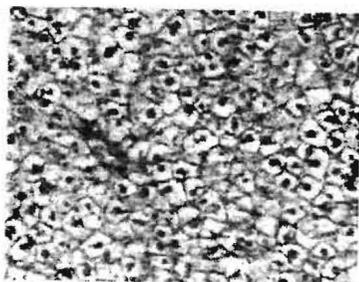
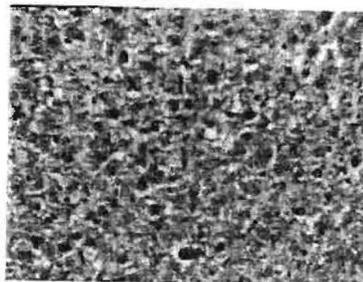


Fig. (5): Liver rats received 400 mg /kg-1, Fig day body weight from pectin isolated from rough wheat bran showing diffuse vascular degenerative changes of hepatocytes others were necrosis (H. E. X:400).



6): Liver rats received 200 mg /kg/ day body weight from pectin isolated from soft wheat bran and 0.13% lead acetate showing necrosis of some hepatocytes (H. E. X: 400).

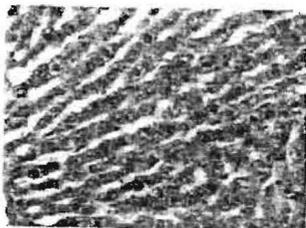


Fig (7): Liver rats received 400 mg /kg/ day, from pectin isolated from soft wheat bran and 0.13% lead acetate showing infiltration of edema in between the hepatocytes (H. E. X: 400).

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## الإستفادة من نخالة القمح كمنتج ثانوي لإنتاج البكتين لتكون فعالة على سمية الرصاص في الفئران

اميره مرسى المسلمانى<sup>١</sup>

قسم اتغذية وعلوم الأظعمة ، كلية الإقتصاد المنزلى ، جامعة الأزهر ، مصر

### الملخص العربى

قد أجريت هذه الدراسة لاستخدام نخالة القمح (الخشنه والناعمه) كمنتج ثانوي لإنتاج البكتين لتكون فعالة على سمية خلات الرصاص في الفئران. تم تصميم التجربة البيولوجية باستخدام فئران ذكور (٣٦ فئران) مقسمة إلى ست مجموعات وتم تغذيتها على الغذاء الأساسى لمدة أربعة أسابيع. تم استخدام المجموعه الأولى كمجموعه ضابطه سالبه تغذت على الغذاء الأساسى بالإضافة الى تناول مياه الشرب العاديه. المجموعه الثانيه وتحتوى على (٦) فئران وتتغذى على الغذاء الأساسى بالإضافة الى تناول مياه الشرب التى تحتوى على خلات الرصاص بتركيز (٠.١٣%). أما المجموعه الثالثه والرابعه فكانت تتغذى على الغذاء الأساسى وتتناول مستخلص البكتين الناتج من الرده الخشنه بتركيز ٢٠٠ و ٤٠٠ ملليجرام/كجم من وزن الفأر بالإضافة الى (٠.١٣%) من خلات الرصاص فى مياه الشرب. أما المجموعه الخامسه والسادسه فكانت تتغذى على الغذاء الأساسى وتتناول مستخلص البكتين الناتج من الرده الناعمه بتركيز ٢٠٠ و ٤٠٠ ملليجرام/كجم من وزن الفأر بالإضافة الى (٠.١٣%) من خلات الرصاص فى مياه الشرب. فى نهاية مدة التجربه (٣٠ يوم) تم حساب التقديرات البيولوجيه والحصول على الدم لتقدير كرات الدم الحمراء والبيضاء وفصل الباقي لتقدير دهون الدم الكامله ووظائف الكبد ووظائف الكلى ووظائف البنكرياس وتم عمل فحص هستوباثولوجى للكبد. أوضحت النتائج أن خلات الرصاص أدت إلى انخفاض ملحوظ في عدد كرات الدم الحمراء وعدد كريات الدم البيضاء وهيموجلوبين الدموالهيماتوكريت والصفائح الدمويه فى المجموعه الضابطه الموجه بالمقارنه بالمجموعه الضابطه السالبه. على العكس من ذلك ، فقد ازدادت انزيمات الكبد وانزيمات الكلى وانزيمات البنكرياس بشكل ملحوظ فى المجموعه الضابطه الموجه بالمقارنه بالمجموعه الضابطه السالبه بينما جميع دهون الدم انخفضت فى المجموعه الضابطه الموجه بالمقارنه بالمجموعه الضابطه السالبه ماعدا الليبوبروتينات منخفضة الكثافه. تناول مستخلص البكتين الناتج من الرده الخشنه والناعمه ادى الى تحسن فى جميع الوظائف التى ارتفعت بسبب خلات الرصاص كما اوضح فحص هستوباثولوجى ان تناول البكتين المستخلص من الرده الخشنه والناعمه قد قلل من التأثيرات الضاره التى أحدثتها خلات الرصاص على أنسجة الكبد. من النتائج الواضحه يمكن التوصيه بأن البكتين من نخالة القمح الخشنه والناعمه له تأثيرات مفيدة ويمكن أن يكون قادرا على أن يصبح مضادا لسمية الرصاص. علاوة على ذلك ساهم فى القضاء السريع على السمية وإزالتها من الدم. يمكن التوصيه بأن البكتين من نخالة القمح الخام والناعمه له تأثير كبير على ربط الرصاص ويجب تحسين تغذية الإنسان لخفض تلوث الرصاص فى الطعام والماء.