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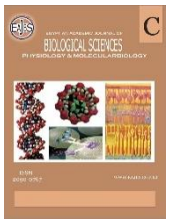

Evaluating Cytotoxic Effects of Lead Acetate on Pea (*Pisum sativum)* and White Albino Rats (*Rattus norvegicus albinus*)

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ABSTRACT

Due to human activities, heavy metals like lead are becoming more prevalent in contaminated soil, water and air, which has an unfavorable cytotoxic and genotoxic effects on all organisms. The current study set out to determine whether lead was cytotoxic to *Pisum sativum* root tips. In the study, three different lead concentrations—1.00, 5.00, and 10.00 ppm were used. It was discovered that the mitotic index decreased as the concentration increased. Control had the highest mitotic index percentage (27%), whereas lead (1.00 ppm) had the lowest mitotic index. When exposed to the maximum concentration, the germination rate dropped by 42%. Lead showed a variety of chromosomal abnormalities, including as micronucleus formation, clumping, bridge, laggards, C-metaphase, and stickiness. Loops and stickiness are examples of chromosomal abnormalities that are found mainly during the metaphase stage of cell division. In addition to histological analyses of liver tissues in male white mice, the effects of lead at 5 ml/1 liter were evaluated on blood enzymes such as plasma alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and creatine kinase (CK). (*Albinus Rattus norvegicus*). According to the results, the activities of the examined enzymes were significantly increased by the treated dose (5 ml/liter) of lead as compared to control. Lastly, histological examinations showed that, when compared to control, the tested dose of lead generated detrimental alterations in the liver tissues of the examined white male mice.

INTRODUCTION

Heavy metal pollution of the air, water, and soil is a serious issue that poses a risk to the environment in this age of industry and urbanization. A number of physiological and cytological processes in cells are disrupted by lead, a heavy metal pollutant that is stable in the environment but extremely harmful to biological organisms (Chander *et al.,* 2001; Zou *et al.,* 2006; Leuent *et al.,* 2009). Lead acetate is a common source of lead poisoning, as are automobile exhaust, petroleum refineries, auto repair shops, lead acid batteries, water pipes, industrial pollutants, and dust polluted with lead in older buildings. According to Haouas *et al.* (2014), lead is also a component of paints, building materials, ceramic glazes, and many other products. A white, crystalline substance with a faintly sweet flavor is lead (II) acetate.

$Pb(OAc)_2$ or $Pb(CH_3COO)_2$ are common expressions for its chemical formula, in which Ac stands for the acetyl group. It produces lead poisoning, just like a lot of other lead compounds. According to Dorunfemi *et al.* (2010), lead causes a number of chromosomal abnormalities in plant cells, which lowers the mitotic index and inhibits root growth. Lead also reduced seed germination or early seedling development are signs of plant toxicity caused by lead interfering with multiple metabolic processes (Sharma *et al.,* 1995 and Shanker *et al.,* 2005). Chromosome alteration analysis is a useful technique for determining a substance's potential for genotoxicity. Long-term exposure to lead also seems to have the potential to cause genotoxic as well as cytotoxic effects in a variety of tissues. According to Jarrar (2003), Sidhu and Nehru (2004), Taib *et al.* (2004), and Flora *et al.* (2006), the absorbed lead is conjugated in the liver and transferred to the kidney, where a small amount is eliminated in urine and the remainder builds up in various body organs. This affects numerous biological activities at the molecular, cellular, and intercellular levels and may cause morphological changes that persist even after Pb levels have decreased. Indeed, it has been demonstrated that lead acetate significantly increases the frequency of sister chromatid exchanges in cultures of Chinese hamster ovary cells (Lin *et al.,* 1994) and chromosomal abnormalities in human cells (Obe *et al.,* 1975). Furthermore, following lead treatment, mice's blood cells showed damage of DNA damage (Devi KD *et al.,* 2000; Allouche *et al.,* 2011). The current study set out to determine the cytotoxic and genotoxic effects of lead acetate on the meristematic root tips of *Pisum sativum* and rat's livers.

MATERIALS AND METHODS

1-Cytological Studies:

Distilled water was applied to rinse the *Pisum sativum* seeds for six hours under three concentrations of lead acetate (1.0, 5.0, and 10.0 ppm), the control set's seeds were soaked in distilled water. Three replicates of each concentration, each containing ten seeds, were then left to germinate in a petri dish lined with Watman's filter paper until the roots reached a length of three to four centimeters. After being sliced, the root tips were fixed in a fixative solution (ethanol: acetic acid (3:1)) for 24 hours in the refrigerator. They were then stored in 70% ethanol until they were processed. For ten minutes, root tips were warmed in a 2% acetocarmine solution. For the purpose of preparing squash and making cytological observations, stained root tips were moved to a slide, squashed and cover with cover slide and examined under light microscope.

2-Tested rat and Experimental Design:

A rat experiment was carried out at Benha University's Faculty of Agriculture's Plant Protection Department. The laboratory animal research center at Benha University's Faculty of Veterinary Medicine provided 18 adult male white albino rats (*Rattus norvegicus albinus* (Berk)) weighing 120 ± 10 g. The rats were kept in metal cages and fed with enough food that included water, 21% protein, 4.59% fat, and 4.20% fiber. Every animal was monitored every day prior to the administration of therapy. Prior to doing the experiment, albino male rats (*R.norvegicus albinus*), a laboratory strain, were housed for two weeks. Rats were divided into two groups at random, with three animals in each group. The second group was fed feed and pure distilled water as a control, while the first group was given feed and water containing Lead (II) acetate Pb $(CH_3COO)_2$ at a concentration of 5 ml/L water. For each treatment, three replicates were conducted. The course of treatment lasted for 30 days straight. Rats livers were examined histologically and enzymeatically and biochemically to assess structural damage. Lead's impact on alkaline phosphatase (ALP), aspartate aminotransferase (AST), and alanine aminotransferase (ALT). The impact of lead on the liver organ was examined histopathologically.

3-Blood and Tissue Samples:

All biochemical parameters were

evaluated in Dr. Mahmoud Abou El.makarem's laboratory in Toukh, Qalubia Governorate, Egypt. Blood samples were taken in serum tubes. Serum levels of AST, ALT, ALP, urea, and creatinine were measured using spectrophotometric analysis, which is a routine procedure. Blood samples from the retroorbital sinus or venous puncture were taken on day fifteen (in tubes containing heparin) and centrifuged for ten minutes at 4000 rpm. For the biochemical test, serum plasma from the resulting clear supernatant was extracted. In order to separate the liver organs for the histopathology investigations, all of the tested mice were killed at the conclusion of the experiment.

3-1-Enzymes Measurements:

Rats livers were examined histologically and enzymeatically and biochemically to assess structural damage. Lead's impact on alkaline phosphatase (ALP), aspartate aminotransferase (AST), and alanine aminotransferase (ALT). The impact of lead on the liver organ was examined histopathologically.

In vivo measurements of plasma alanine aminotransferase, ALT (sGPT), and aspartate aminotransferase, AST (sGOT), were conducted using the IFCC's 1986 methodology. ALP (alkaline phosphatase) was measured using biodiagnostics kits. Urea and creatine kinase (CK) were found in accordance with IFCC (1989).

4-Histopathological Studies:

The liver organs under treatment and management underwent histopathological investigations. After removing any excess material and fixing it in neutral buffer (10% formalin), the isolated liver was dehydrated in increasing alcohol grades, cleaned in xylene, embedded in paraffin, cut into paraffin sections that were 4 microns thick, and stained with hematoxylin and eosin using normal methods. In 1996, Banchroft *et al.* A digital camera attached to a computer was used to evaluate and take pictures of the pieces. These methods were used at Benha University's Faculty of Medicine, Pathology Department.

5-Statistical Analysis.

Analysis of variance (ANOVA) was used in SPSS to statistically analyze the data for the cytological investigations. The data were statistically evaluated for the enzyme analysis in accordance with SAS (2007) and Steel and Torrie (1980) to obtain L.S.D. The values were recorded as mean ± S.D.

RESULTS AND DISCUSSION

Cytological Analysis:

Lead's cytotoxic effects are clearly visible at different concentrations (1, 5 and 10 ppm), resulting in a steady decline in mitotic index of 24%, 18%, and 11%, respectively, whereas the control exhibits the greatest mitotic index of 39% (Fig.1). While treated sets display a considerable number of chromosomal abnormalities, as indicated in Table 1, the control group exhibits nonsignificant aberrant cell division. Stickiness, bridge, lagging, clumping, fragment, and c-mitosis, as depicted in Figure 3, are examples of aberrations that occur during the investigation. At the maximum concentration, chromosome breakdown and loop formation were produced. Throughout all phases of mitosis, stickiness is noticeable.

The results unequivocally showed that the lead caused a significant quantity of genotoxicity. According to Smaka-kinel *et al.* (1996), the mitotic index is an excellent tool for identifying cytotoxicity in all living things. According to Figure 3, the 2.72 percent of all the cells that were analyzed were in C-metaphase. By increasing the concentration of the treated dose of lead acetate (4.93% in the case of 1 ppm, 5.78 % in the case of 5 ppm, and 7.48 % in the case of 10 ppm as shown in Figure 5, respectively, Table 1), the proportion of cells with C-metaphase was raised.

In the control group, 2.93 percent of the cells under examination (Fig. 3), and in the cases of 1 ppm of lead acetate (Fig. 4), the 6.65 percent of the cells under examination had a delayed anaphase stage, and 7.77 percent of the cells acquired as a consequence of 10 ppm of lead acetate (Fig. 6), the delayed anaphase case was detected. In all treatments,

sticky chromosomes were found; in cells treated with 1 ppm of lead acetate, they were 4.20 percent; in cells treated with 5 and 10 ppm of lead acetate, they were 3.54% and 3.59%, respectively, as shown in Figures. 3 and 5. As seen in Figures. 3, 4, 5, and 6, the 1.08 percent of the cells under examination had lagging chromosomes at the treated dose of 1 ppm. Following treatment with 5 ppm of lead acetate, 2.21 percent of the analyzed cells had lagging chromosomes, whereas 2.67 percent of the screened cells had lagging chromosomes following treatment with 10 ppm of lead acetate. Bridges were also identified, and it was discovered that they increased with the rise in treatment dose: 1.51 percent for 1 ppm of lead acetate, 1.81 percent for 5 ppm, and 2.24 percent for 10 ppm. As demonstrated by earlier research, an increase in concentration is linked to a decrease in the mitotic index. Numerous chromosomal abnormalities, including as laggards, anaphase bridges, and telophase bridges, are brought on by heavy metals and may be caused by an uneven exchange of dicentric chromosomes. One of the effects of inactivating the spindle machinery linked to centromere division delay is C-mitosis (Mann 1997, Kirkla 1998, and Aremu *et al.,* 2006). Stickiness and clumping are noticeable at every stage of the current investigation. Stickiness is a more serious disruption in cytology than C-mitosis and chromosome bridges (Al Achkar *et al.,* 1989; Yücel *et al.,* 2008 and Zheng *et al.,* 2009). Several authors have also noted stickiness in their studies on environmental influences. At any stage of the cell cycle, chromosomal clustering is a characteristic of chromosome stickiness. The current study found that lead was cytotoxic to *Pisum sativum*, increasing chromosomal aberrations and, most significantly, decreasing germination rates, both of which had a direct impact on yield.

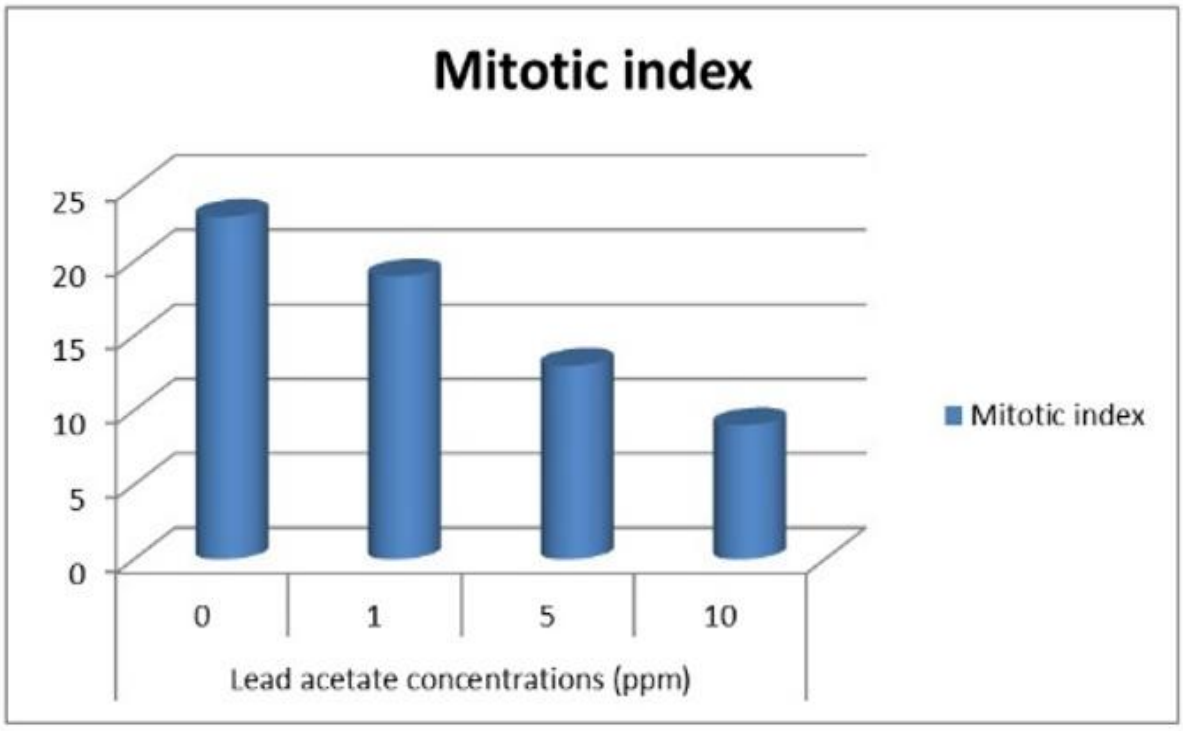


Fig.1: Effect of lead on mitotic index of *Pisum sativum*.

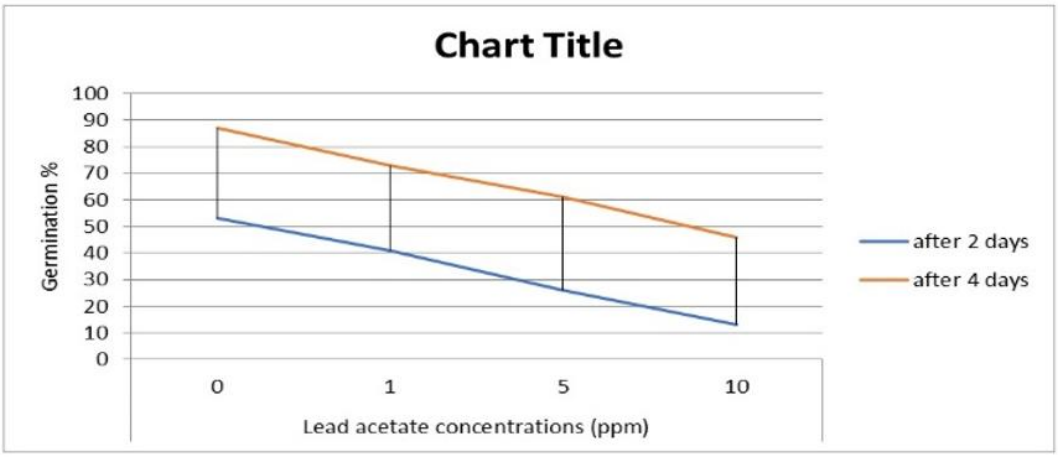


Fig.2: Effect of lead acetate on germination percentage of *Pisum sativum*.

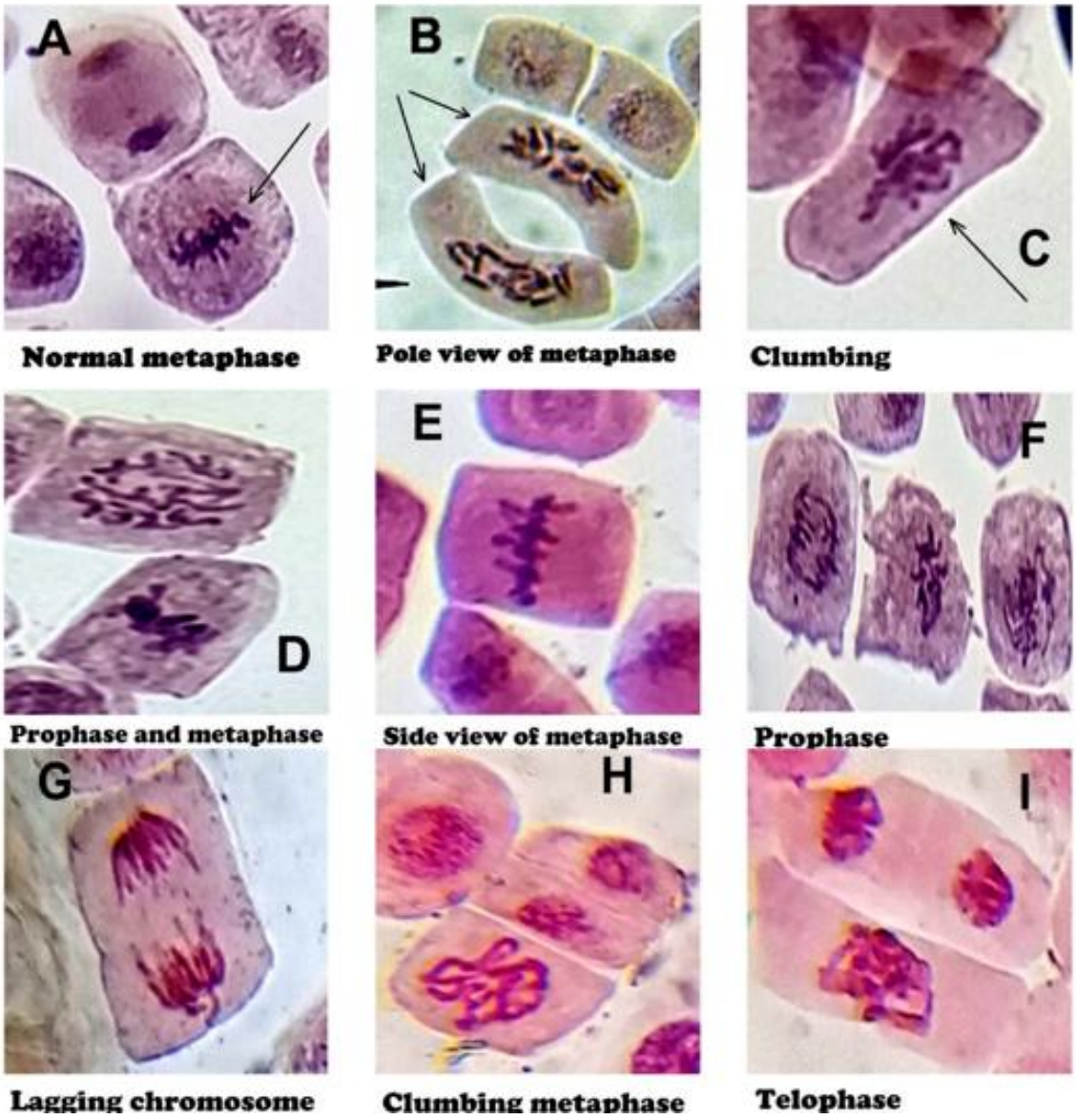


Fig. 3: Mitotic cells induced by lead acetate treatment in *Pisum sativum*. A to C: Displaying telophase, C-metaphase, sticky metaphase, and typical anaphase D: The sticky phase F: Ansynchronus anaphase, E: Sticky metaphase, G: Lagging fragment anaphase, Scale bar = 10µm, H: Clumping, I: Loop creation.

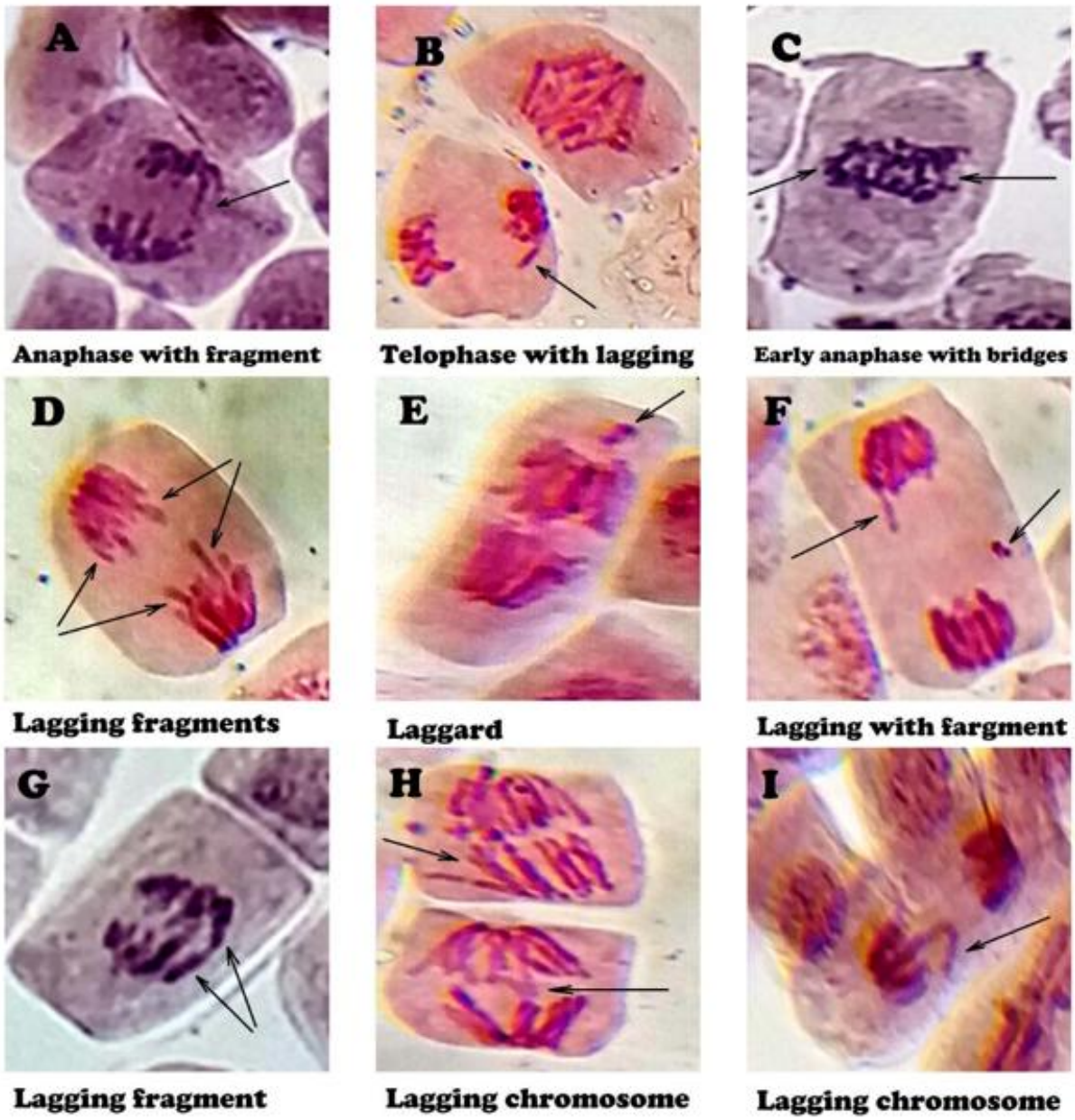


Fig. 4: Mitotic cells induced by lead acetate treatment in *Pisum sativum*. A to C: Displaying typical telophase, metaphase, and anaphase D: Bridge of anaphase, E: Fragmentary sticky anaphase, F: Anaphase ansynchronus, G: Metaphase with fragment, H: Clumping, I: Formation of loops, J: Telophase with laggards, K: Disintegration of chromosomes, L: C-mitosis. 10µm is the scale bar.

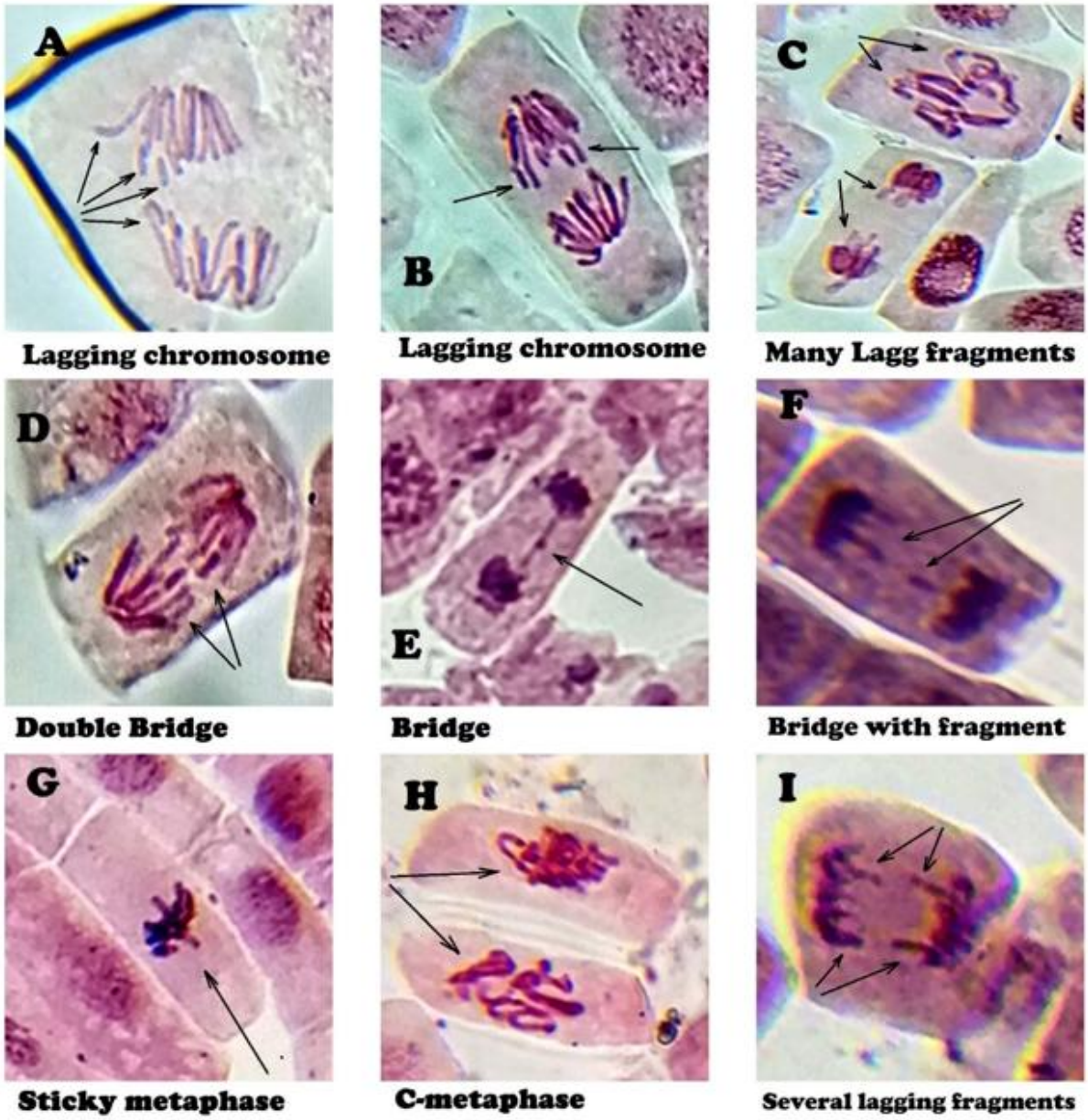


Fig. 5: Mitotic cells induced by lead acetate treatment in *Pisum sativum*. A to C: Displaying typical telophase, metaphase, and anaphase D: Bridge of anaphase, E: Fragmentary sticky anaphase, F: Anaphase ansynchronus, G: Metaphase with fragment, H: Clumping, I: Formation of loops, J: Telophase with laggards, K: Disintegration of chromosomes, L: C-mitosis. 10µm is the scale bar.

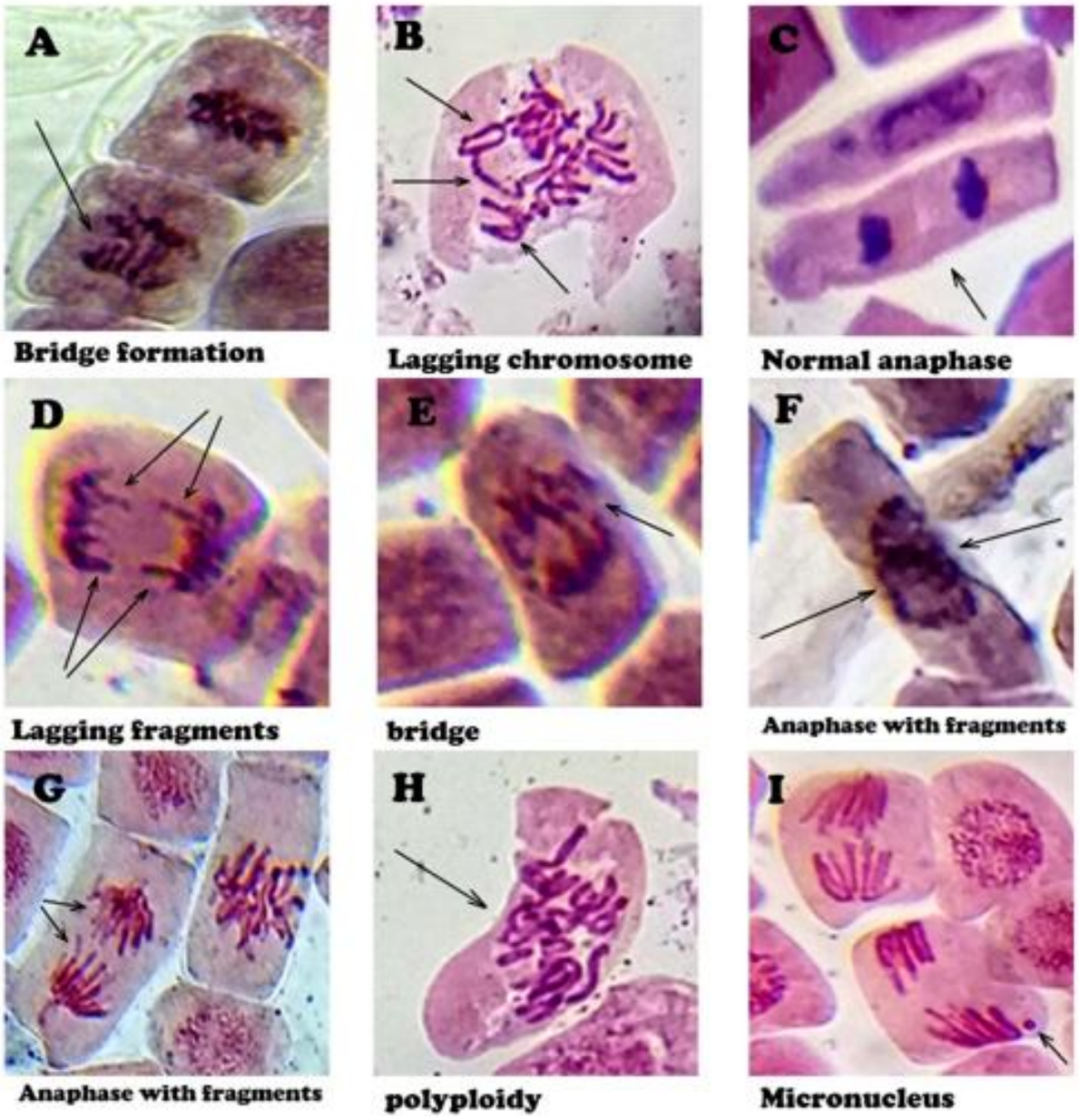


Fig. 6: Mitotic cells induced by lead acetate treatment in *Pisum sativum*. Anaphase, metaphase, and telophase are all displayed normally in A to C. D: Bridge of anaphase, E: Fragmentary sticky anaphase, F: Anaphase ansynchronus, G: Metaphase with fragment, H: Clumping, I: Formation of loops, J: Telophase with laggards, K: Disintegration of chromosomes, L: C-mitosis. 10µm is the scale bar.

Table 1: Mitotic aberration induced by lead acetate treatment in *Pisum sativum* with three different concentrations (1.0, 5.0 and 10.0 ppm).

	Concentration of lead acetate (ppm)			
	Control	**1.0**	**5.0**	**10.0**
No. of examined cells	1397	1379	1382	1390
C metaphase	38 2.72 %	68 4.93 %	80 5.78 %	104 7.48 %
Delayed anaphase	41 2.93 %	64 4.64 %	92 6.65 %	108 7.77 %
Stickiness	4 0.28 %	58 4.20 %	49 3.54 %	50 3.59 %
Laggard	0 0 %	15 1.08 %	30 2.21 %	38 2.67 %
Abnormal anaphase	0 0 %	7 0.50 %	11 0.79 %	17 1.22 %
Abnormal metaphase	10 0.71 %	28 2.03 %	25 1.80 %	20 1.43 %
Chromosome Bridge	0 0 %	21 1.52 %	26 1.81 %	32 2.24 %
Chromatid bridge	0 0 %	0 0 %	0 0 %	8 0.57 %
Other aberrations	0 0 %	22 1.59 %	15 1.08 %	31 2.23 %
Number of abnormal cells	93	301	328	409
	6.65	21.82 %	23.73 %	29.42 %

Biochemical Measurements:

1-Enzymes Measurements.

Lead's effects on the enzymes creatine kinase (CK), alkaline phosphatase (ALP), aspartate amino transferase (AST), and alanine aminotransferase (ALT) are shown in Table (2) as mean ± standard deviation.

Table 2: Effect of that Lead (II) acetate Pb $(CH_3COO)_2$ on ALT, AST, ALP, urea and creatinine in blood of rats.

	Creatinine	**Urea**	**ALP**	**AST**	**AL**
Control	0.69 ± 0.018^{B}	42.367 ± 0.179^{B}	85.967 ± 2.291^{B}	178.967 ± 3.705^{B}	21.333 ± 0.337^{B}
Lead acetate 5 ml/1 Litre	0.32 ± 0.018^{A}	60.2 ± 0.179^{A}	102.433 ± 2.291^{A}	313.733 ± 3.705^{A}	36.3 ± 0.337^{A}

Because it is stored in the liver following exposure to lead, the liver is regarded as one of the main target organs affected by lead toxicity. Furthermore, heavy metal poisoning is relevant since the liver is one of the primary organs involved in the storage, biotransformation, and detoxification of hazardous chemicals (Herman and Geraldine, 2009). The liver is the first organ for which histological analysis can be used to examine the morphological changes that reflect possible lead effects on somatic cells because soft tissues, particularly the liver, store absorbed lead through the portal vein (Patrick L., 2006) (Sidhu and Nehru, 2004; Abdou and Newairy, 2006).

At the studied dose, lead markedly elevated the ALT enzyme activity in the blood of white male albinos (*Rattus norvegicus albinus*). Comparing the growing percentage to the control, which was 21.33, the result was +36.3.

AST and ALT, two liver enzymes, are regarded as a crucial diagnostic for identifying lead hepatotoxicity. Our findings showed that, in comparison to the control

group, lead significantly raised AST and ALT levels. The primary cause of the elevated AST and ALT levels in treated rats' plasma is the enzymes' leakage into the bloodstream from the liver cytosol (Concepción Navarro *et al.,* 1993). Enzyme leakage from the damaged hepatic cells into the bloodstream causes a marked increase in the AST level in the plasma. When an organ experiences cellular degeneration or destruction, ALT levels in plasma similarly rise (Hassoun and Stohs, 1995).

Abdel-Kader *et al.* (2011) observed similar outcomes after giving rats 1000 ppm of lead acetate in water for four weeks. Sivarprasad *et al.* (2004) demonstrated that male Wistar rats exposed to 0.2% lead acetate in water for five weeks had higher levels of serum transaminases. Lead significantly increased the AST enzyme's activity in the blood of white male mice as well, but not as much as ALT (313.73 versus 178.96). Thus, the findings are consistent with Dacie and Lewis (1995), who discovered that whereas AST is present in a variety of tissues, including the heart, bones, muscles, kidney, and brain, ALT is mostly located in the liver.

In the case of the measured dose of lead in the blood of white albino rats, the alkaline phosphatase enzyme (ALP) activity was likewise significantly elevated, reaching 102.43 as opposed to the control level of 85.96. Young (2000) found that elevated ALP levels are linked to bone disorders, suggesting that lead may be responsible for certain bone disorders at sublethal levels assessed on male white albino rats (*Rattus norvegicus albinus*).

The activity of the creatine kinase (CK) enzyme was shown to be lower, reaching 0.32 as opposed to 0.69 for the control. According to Young (2000), myocardial infraction or skeletal muscle disorders may be linked to the subsequent elevation of CK levels brought on by lead therapy. Based on these results, it can be said that the tested dose of lead may have a negative impact on a number of organs in connection to the white albino rat's (*Rattus norvegicus albinus*) ALT, AST, ALP, urea, and CK.

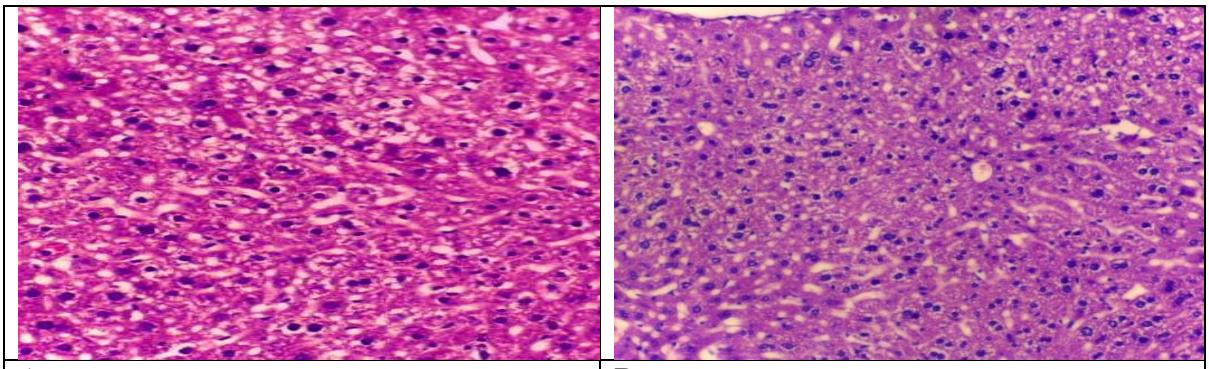
2.Histopathological Effects on Liver:

The group of white male mice used as a control showed no histopathological alterations, and the normal histological structure of the central veins and surrounding hepatocytes in the parenchyma was seen (Fig 7a). Mice given 5 ml/Litre of lead showed intracytoplasmic vacuolar degeneration in their hepatocytes (Fig 7). In white albino rats, a 5 ml/Litre dosage of lead resulted in the development of diffuse Kupffer cells between the hepatocytes (Fig 7b). In addition to vacuolar degeneration in the hepatocytes and significant dilatation in the portal veins, the histological examinations of the liver of white albino rats treated with 5 ml/Litre of lead revealed early dysplasia with early fatty alterations.

These findings could have a significant impact on a number of organs in regard to the white albino rat (*Rattus norvegicus albinus*) enzymes that were evaluated. According to histological investigations, the tested amount of lead caused detrimental changes in the white albino rat's liver tissues when compared to the control. Histological examination revealed a number of alterations, including the disarray of the hepatic cords and the enlargement of the hepatocytes. Suradkar (2010) reported similar findings in rats given 1000 ppm lead acetate for 28 days. According to Majno and Joris (1995), Searle *et al.* (1982), Rosser and Gores (1995), and Thompson (1995), the swelling of intracellular organs, particularly the mitochondria and endoplasmic reticulum, was the cause of the hypertrophy.

Additionally, cell vacuolation, a cellular defense mechanism against harmful compounds, is one way that liver toxicity manifests itself (Mollendorf, 1973; Taib *et al.,* 2004). Due to their segregation within vacuoles, these chemicals were unable to disrupt cellular metabolism. Additionally, it has been proposed that disruptions in lipid inclusions and fat metabolism are the primary cause of cytoplasmic vacuolation (Zhang and Wang, 1984). Following lead therapy, lymphocytic infiltration and sinusoidal blood congestion are signs of liver injury. EL-

Sokkary *et al.* (2005), Joher *et al.* (2004), Liu *et al.* (2012), Sharma *et al.* (2010), and Mudipalli (2007) have also published similar studies. Cell irritability, inflammation, and hypersensitivity to the toxicant utilized are all evident in the lymphocytic infiltrates seen in this study after lead treatment. Furthermore, our results demonstrated central vein dilatation and portal tact, which are consistent with the findings of Ibrahim *et al.* (2012) and El-Sokkary *et al.* (2005), who gave rats 100 mg of lead acetate.



A B

Fig. 7. (A) White albino rat liver in control: showing normal histological structure of the hepatocytes surrounding the central vein in the hepatic parenchyma. **(B)** White albino rat liver treated with 5 ml/l of lead acetate, showing early fatty alterations and dysplasia. × 40.

Declarations:

Ethical Approval: Not applicable.

Competing interests: The authors declare no conflicts of interest.

Contributions: All authors contributed equally, and have read and agreed to the published version of the manuscript.

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ARABIC SUMMARY

تقييم التأثيرات السامة لأسيتات الرصاص على نبات البازلاء (*Pisum sativum*) والفئران البيضاء (*Rattus norvegicus albinus*).

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بسبب الأنشطة البشرية، تزداد المعادن الثقيلة، مثل الرصاص، انتشارًا في التربة والهواء الملوثين، مما يؤثر سلبًا على جينات الخضراوات مثل البازلاء. هدفت الدراسة الحالية إلى تحديد ما إذا كان الرصاص سامًا جينيًا لأطراف جذور نبات البازلاء. في هذه الدراسة، استُخدمت ثلاثة تراكيز مختلفة من الرصاص - 1.00، 5.00، و10.00 جزء في المليون. وقد لوحظ انخفاض في مؤشر الانقسام الفتيلي مع ارتفاع التركيز. سجلت المجموعة الضابطة أعلى نسبة مؤشر انقسامي (27%)، بينما سجلت المجموعة الرصاصية (1.00 جزء في المليون) أدنى نسبة مؤشر انقسامي. عند التعرض لأعلى تركيز، انخفض معدل الإنبات بنسبة 42%. أظهر الرصاص مجموعة متنوعة من التشوهات الكروموسومية، بما في ذلك النوى الصغيرة، والتكتل، والجسر، والتأخر، والطور الاستوائي C، والالتصاق. تُعد الحلقات والالتصاق أمثلة على التشوهات الكروموسومية التي تُلاحظ بشكل أساسي خلال مرحلة الطور الاستوائي من انقسام الخلايا. بالإضافة إلى التحليلات النسيجية لأنسجة الكبد في ذكور الفئران البيضاء، تم تقييم آثار الرصاص بالجرعة المعاملة (مللى5 /1 لتر) على إنزيمات الدم مثل ناقلة أمين الألانين البلازمية (ALT) وأسبارتات أمينوترانسفيراز (AST) والفوسفاتيز القلوي (ALP) وكيناز الكرياتين (*Albinus Rattus norvegicus*) .(CK). ووفقًا للنتائج، ازدادت أنشطة الإنزيمات المفحوصة بشكل ملحوظ عند الجرعة (5 مل/لتر) من الرصاص، على الرغم من أن الأنشطة كانت أقل تغيرًا عند المجموعة الضابطة. وأخيرًا، أظهرت الفحوصات النسيجية أنه عند مقارنتها بمجموعة التحكم، أحدثت جميع جرعات الرصاص المختبرة تغيرات ضارة في أنسجة الكبد لدى ذكور الفئران البيضاء المفحوصة.

الكلمات المفتاحية: الرصاص، التشوهات الكروموسومية، السمية الجينية، ALT، AST، ALP، دراسات نسيجية مرضية على الكبد، ذكور الفئران البيضاء (*Rattus norvegicus albinus*).