

# **EFFECT OF SELENIUM ON THYROID BIOCHEMICAL AND HISTOPATHOLOGICAL CHANGES INDUCED BY SUBCHRONIC LEAD ACETATE EXPOSURE IN ADULT ALBINO RATS**

*BY*

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## **ABSTRACT**

*We evaluated the effect of selenium on thyroid functions, oxidative stress and histopathological changes induced by subchronic lead acetate exposure. The study was for eight weeks on fifty adult albino rats of both sexes divided into five equal groups: negative control group (I), positive control group (II), positive control selenium group (III) in a dose of 0.35 mg/kg bw; orally per day daily, lead acetate group (IV) in a dose of 60 mg/kg bw; orally per day and selenium (0.35 mg/kg bw; orally per day) + lead acetate (60 mg/kg bw; orally per day) group (V). After eight weeks rats were anaesthetized by ether and blood samples were collected to evaluate thyroid functions in form of Triiodothyronine (T3), Total Thyroxin (T4) and Thyroid Stimulating Hormone (TSH) levels as well as blood lead level. Then animals were sacrificed and thyroid tissue was obtained and examined histopathologically and estimate lead content, malondialdehyde (MDA) and antioxidative enzyme superoxide dismutase (SOD). Results revealed significant decrease in both T3, T4 levels and increase in TSH and blood lead level in lead acetate group (IV) when compared with control group (I). An increase in lead content as well as malondialdehyde and decrease in superoxide dismutase in thyroid tissue was observed in lead acetate group (IV). Histopathologically, there was numerous variable sized thyroid acini lined by hypertrophied columnar epithelium and filled with homogenous eosinophilic colloid material showing peripheral scalloping. Rats treated with selenium + lead acetate group (V) showed increase in both T3, T4 levels and decrease in TSH and blood lead level but not returned to normal when compared with lead acetate group (IV). Additionally, decrease in lead content and malondialdehyde of thyroid tissue and increase in superoxide dismutase activity was observed when compared with lead acetate group (IV). Histopathologically, there were some acini filled with cubical epithelium with no peripheral scalloping. We concluded that selenium has an ameliorative effect on lead acetate induced biochemical and histopathological changes on thyroid gland.*

**Key words:** *lead acetate - selenium - thyroid - oxidative stress.*

## **INTRODUCTION**

Lead (Pb) is considered to be one of the major environmental pollutants

and has been incriminated as a cause of accidental poisoning more than any other substance (Casas and Sordo, 2006).

One of the primary sources of lead contamination in air, soil and water is combustion of fuels containing lead additives. Other sources of ingestible lead include storage battery plates, radiator repair, secondary smelters, putty, linoleum, asphalt roofing, engine oil, insecticide baits, and contaminated feeds (Yu, 2004).

Despite of attempts for reducing the exposure to this metal, there are still some reports of cases with severe lead toxicity (Hershko, 2005).

Lead can cause profound hematological, neurological, gastrointestinal, renal, rheumatological and endocrine manifestations in man even at levels previously considered safe (Lyons and Pahwa, 2005).

Lead exposure also causes functional impairment of pituitary-adrenal axis as well as the pituitary-thyroid axis. In addition lead enhances the production of reactive oxygen species (ROS) in a variety of cells resulting to oxidative stress. ROS are highly reactive to membrane lipids, protein, and DNA. They are believed to be the major contributing factors to stress injuries and to cause rapid cellular damage (Singh and Dhawan, 1999 ; El-Beltagi et al., 2011).

The element selenium (Se) was discovered in 1817 and it is essential for normal life Processes (Köhrle, 2004).

Selenium is introduced into the food chain by plants, which absorb inorganic selenium salts from the soil and convert them into organic forms of the element (mainly as selenomethionine), which are then incorporated into proteins. The concentration of selenium in plants varies widely and depends on the selenium content and characteristics of soil (Pavlata et al., 2005).

Selenium is essential for normal thyroid function and thyroid hormone homeostasis (Beckett and Arthur, 2005) .This trace element acts as an antioxidant in the thyroid and a regulator of triiodothyronine (T3) production. It has been demonstrated that Se has a central role as supplementary therapy in Graves' disease because of its action through selenoenzymes both on transforming of the thyroid hormones and on the antioxidative defense of the organism (Vrca et al., 2004).

Selenium was used in treatment of free radical-associated disease such as diabetes, aflatoxin B1-induced lipid peroxidation, and in development of chemopreventive antioxidant drugs (Kahler et al., 1993).

The aim of this work is to study the biochemical and histopathological effects of lead acetate on the thyroid and evaluate the role of selenium in amelioration of the toxic changes induced by lead acetate.

## MATERIAL AND METHODS

### Chemicals:

- **Lead acetate:** lead acetate was purchased from Sigma Chemical (St. Louis, Mo).
- **Selenium:** sodium selenite ( $\text{Na}_2\text{SeO}_3$ ) was purchased from Merck (Dormstadt, Germany). The chemicals used were of the highest purity.

### Animals:

This study was conducted for 8 weeks on 50 adult albino rats of both sexes (weight 100-150gm), divided into 5 groups, each consisted of 10 rats:

- **Group I** (-ve control group): the rats were given the basal diet and water.
- **Group II** (+ve control group): Each rat was gavaged orally with 1 ml of distilled water (vehicle of lead acetate and selenium) once daily.
- **Group III** (+ve control selenium group): Each rat was gavaged orally with selenium in a dose of (0.35 mg/kg b.wt. per day) (Jamba et al., 1997) dissolved in 1 ml of distilled water.
- **Group IV** (lead acetate group): Each rat was gavaged orally with lead acetate in a dose of (60 mg/kg b.wt. per day) (Karamala et al., 2011) dissolved in 1 ml of distilled water. This dose represents 1/10 LD50.
- **Group V** (selenium + lead acetate group): Each rat was gavaged orally

with selenium(0.35 mg/kg b.wt. per day) +lead acetate (60 mg/kg b.wt. per day), each dose was dissolved in 1 ml of distilled water.

### Methods:

#### (I) Biochemical study:

After 8 weeks, rats of all groups were anesthetized and blood samples were collected from the retro-orbital plexus to assess the thyroid functions in form of total Triiodothyronine (T3), Total Thyroxin (T4) and Thyroid Stimulating Hormone (TSH) were estimated in serum samples by radio-immuno assay (RIA) using DIMA GmbH Diagnostics kits, Goettingen, Germany according to the method described by Young et al, (1975) and Sterling (1975) and (Kieffer et al., 1975) respectively. Blood lead level was measured by atomic absorption spectrometry according to Parsons and Slavin, (1993).

Lipid peroxidation process indicator, which is thyroid malondialdehyde (MDA) was estimated according to Ohkawa et al., (1979) and antioxidant enzyme superoxide dismutase (SOD) activity was measured in thyroid tissue according to the method of Martin et al. (1987).

Then rats were sacrificed by doing longitudinal incision in the neck and thyroid specimens were obtained to assess the lead content by atomic absorption spectrometry.

**(II) Histopathological study:**

The thyroid specimens were stained by Hematoxylin and Eosin and subjected to histopathological examination by light microscope.

Results will be collected, analyzed by ANOVA test and Post hoc least significant difference (LSD) test.

**RESULTS****(I) Biochemical results:**

As comparing the control groups (I, II, III) no significant differences ( $P > 0.05$ ) in serum T3, T4 and TSH as well as blood lead level and thyroid lead content and also thyroid (MDA and SOD) were detected. Therefore, the (-ve) control group (I) was used in the statistical comparison with the other treated groups (Table 1).

The statistical study revealed a high significant decrease ( $P < 0.0001$ ) in T3 and T4 values and high significant increase ( $P < 0.0001$ ) in TSH values when compared all treated groups with (-ve) control group (I) all over the period of the study. While a high significant increase ( $P < 0.01$ ) in both T3 and T4 values and high significant decrease ( $P < 0.0001$ ) in TSH values when compared (lead acetate + selenium) group (V) with lead acetate group (IV) (Table 2).

In addition, there was a high significant increase ( $P < 0.0001$ ) in both blood lead lev-

el and thyroid lead content when compared all treated groups with (-ve) control group (I) all over the period of the study. Comparison between (lead acetate + selenium) group (V) with lead acetate group (IV), the results showed a high significant decrease ( $P < 0.0001$ ) (Table 3).

Lipid peroxidation process evaluation via estimation of thyroid MDA and SOD, in this study results revealed a high significant increase ( $P < 0.0001$ ) in MDA level and a high significant decrease ( $P < 0.0001$ ) in SOD values when compared all treated groups with (-ve) control group (I) all over the period of the study. Regarding the comparison of (lead acetate + selenium) group (V) with lead acetate group (IV) there was significant decrease ( $P < 0.01$ ) in thyroid MDA values and significant increase ( $P < 0.001$ ) in thyroid SOD values (Table 4).

**(II) Histopathological results:**

The examination of thyroid of control groups (I, II, III) revealed normal thyroid follicles lined with cubical epithelium lie on the periphery of the gland and colloids concentrated in the centre (Figure 1).

After 8 weeks rat's thyroid of lead acetate group (IV) showed numerous variable sized thyroid acini lined by columnar epithelium filled with homogenous eosinophilic colloid material showing peripheral vacuoles (scalloping) (Figure 2). After add-

ing selenium in group (V), examined sections of this group showed improvement in form of presence of some acini lined by cubical epithelium filled with homogeneous eosinophilic colloid material with no peripheral vacuoles (scalloping) (Figure 3).

### **DISCUSSION**

The pathogenesis of lead toxicity is multifactorial as it directly interrupts enzymes activation, completely inhibits trace minerals absorption, binds to sulfhydryl protein, lowers the level of available sulfhydryl antioxidants and alters calcium homeostasis (Khotimchenko et al., 2004).

Lead acetate group (IV) showed a high significant changes in T3, T4 and TSH values. In addition, there was a high significant increase in both blood lead level and thyroid lead content. Regarding the thyroid MDA there was significant increase in thyroid MDA values, while there was significant decrease in thyroid SOD values when compared with control(I).

These findings coincide with Lau et al., (1991) and El-Nahal, (2010) who found that administration of the lead solution through a gavage resulted in considerable alterations of thyroid function in form of marked decrease of blood thyroid hormones level (T3 and T4) and influence the thyrotrophin-releasing hormone synthesis

and increase the binding of this hormone to anterior pituitary receptors resulting in consequent rise in release of TSH.

Yousif and Ahmed, (2009) explained that the tendency towards an increase in the serum TSH concentration observed at exposure to lead is a likely response to decreased serum T3 and T4 levels as a feedback mechanism.

In contrast to our results Shyam et al., (1997) reported that declined concentration of serum T3 and marginal increase of T4 (non significant increase) in lead treated rats might be due to decreased of transformation rate from T4 to T3 according to inhibition of type-I iodothyronine5-monodeiodinase (5-D) activity, being a selenoenzyme containing a selenocysteine residue as its active site. Lead can inhibit 5-D activity through binding to sulfhydryl groups of this enzyme. To explain the previous data, Langer and Gschwendtova, (1991) stated that T4 may be not significantly increased despite inhibition of (5-D) activity as it is utilized in other metabolic pathways.

Contradictory reports were also available in human subject on the level of circulating thyroid hormones after lead exposure mentioned that long-term low-level lead exposure may lead to reduced T4 level without significant changes in T3 level in adolescents even at low lead blood lev-

els. (Schantz and Widholm, 2001 ;Dundara et al., 2006).

Results of blood lead level and thyroid lead content of the present study go in agreement with Khotimchenko et al., (2004) and Omotosho et al., (2011) who stated that the mean blood lead concentration in the negative control group was substantially lower than among lead-exposed groups and accumulation of considerable amount (6.5 fold higher) of lead in the gland as well as in the blood of rats received lead acetate for 3 weeks.

Results of this study showed significant increase in MDA values in lead acetate group (IV), these findings keep matching with results reported by Oladipo, (2010) and Ambali et al., (2011) who stated that increased thyroid MDA concentration is an indicator of oxidative damage to the thyroid gland. This may be attributed to the high metabolic rate, high level of free radicals accumulation, and low level of endogenous antioxidant in the thyroid gland, and this oxidative stress induction is one of the molecular mechanisms of lead poisoning. Also, the decrease in SOD activity in results of this study can be explained by lead induced alteration of antioxidant activities by inhibiting functional SH groups in several enzymes such as aminolevulinic acid dehydratase (ALAD), superoxide dismutase (SOD), catalase (CAT), glutathione peroxidase (GPx), and

glucose- 6-phosphate dehydrogenase (G6PD) as reported by Chiba et al., (1996).

As regard the histopathological results of the present study, rats received lead acetate for 8 weeks group (IV) showed alteration in the thyroid structure in form of increase in the height of the follicular epithelium , vaculation and fainting of the colloid. These findings matching with Khotimchenko et al., (2004) who reported that thyroid gland removed from rats given lead acetate showed that administration of lead acetate resulted in formation of hypertrophied epithelium of follicles presented with cubical or columnar epithelial cells. Colloid appeared to have a faint coloration compared to control group indicating the low contents of thyreoglobulin. In addition there were a plenty of vacuoles in peripheral as well as in central parts of the gland.

The present study showed a significant increase in (T3, T4) and significant decrease in TSH. As regard blood lead level and thyroid lead content, there was significant decrease of both. As well as significant decrease for thyroid MDA and significant increase for thyroid SOD when the selenium added in group (V), this was observed all over the period of the study when compared with lead acetate group (IV). In keeping with these findings Dhingra and Bansal, (2005) reported that selenium is a critical element of seleno-enzyme,

type I iodothyronine deiodinase (5'-DI), which is required for hepatic conversion of T<sub>4</sub> to 3, 3', 5-tri-iodothyronine (T<sub>3</sub>). In agreement with another study by Jin-sang et al., (1996) who suggested that lead can cause toxic effects on thyroid and selenium seems to have a protective effect on specific reaction by lead induced organic function toxicity.

The protective effect of selenium against lead might be attributed to the formation of inactive selenium-lead complexes which can explain the reduction of blood lead level and also the thyroid lead content as shown in this study. However, it seemed that such interactions could not constitute the sole mechanism for the observed blockade by selenium of lead-induced biochemical changes in tissues (Whanger, 1992).

Lipid peroxidation process amelioration, which was detected in this study as an effect of selenium can be possibly due to the mechanism of its action as demonstrated by Othman and El Missiry, (1998) who declared that selenium was found to enhance the antioxidant capacity of cells by increasing the activities of SOD and glutathione reductase and augmenting the glutathione content. Three possible mechanisms were proposed for the protective effect of selenium: (i) formation of an inactive selenium-lead complex; (ii) stimulating radical scavenging by increasing the

activity of SOD, thereby increasing the removal of the superoxide radical; and (iii) increasing the antioxidant capacity of cells indirectly by increasing the activity of glutathione reductase, which has a major role in maintaining a sufficient content of glutathione in the reduced form.

As regard the histopathological results of the present study, rats received (lead acetate + selenium) group (V) for 8 weeks showed improvement in form of presence of some acini lined by cubical epithelium filled with homogenous eosinophilic colloid material with no peripheral vacuoles, these findings may be attributed to accumulation of lead in the organism inducing damage to cell membranes and disorders of the oxido-reductive processes in the cells (Lasisz et al., 1992). Therefore thyroid dysfunction in this study might be related to structural damage of thyroid follicular cells due to accumulation of lead in the thyroid gland.

In addition, Singh and Dhawan, (1999) found that lead exposure impaires uptake of iodine in thyroid gland, leading to decreased volume of the follicles and contributing to formation of the so-called relative iodine deficiency. It leads to expansion of connective tissue and reduction of the thyroglobulin concentration in the follicles of the gland.

As the fact that essential trace element

selenium is required for thyroid hormone synthesis and metabolism. The thyroid gland has the highest selenium content covalently incorporated into several selenoproteins such as the families of glutathione peroxidases, thioredoxin reductases and deiodinases. These contribute to thyroid hormone biosynthesis, antioxidative defense and redox control of thyrocytes as well as to thyroid hormone metabolism (Köhrle and Gärtner, 2009).

The use of selenium can increase the thyroglobulin production and disappearance of scalloping (vacuoles) and the fol-

licular epithelium become cubical or low columnar because these cells need no more hyperactivation to produce thyroid hormones. As well as the improvement of the redox state of the gland will reduce the lead induced thyroid damage.

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**Table (1):** Comparison of thyroid functions (T3), (T4) and (TSH), blood lead level, thyroid lead content and thyroid (MDA), (SOD) values in (– ve) control (I), lead acetate (IV) and lead acetate + selenium (V) groups after 8 weeks.

	Negative control (I) Mean ± SD	Positive control (II) Mean ± SD	Positive control selenium (III) Mean ± SD	P
<b>T3 (ng/ml)</b>	2.87 ± 0.18	2.92 ± 0.38	2.81 ± 0.16	>0.05
<b>T4 (ng/ml)</b>	33.24 ± 3.81	31.17 ± 4.23	34.53 ± 4.49	>0.05
<b>TSH (uIU/ml)</b>	1.39 ± 0.24	1.41 ± 0.28	1.36 ± 0.21	>0.05
<b>Blood lead level (µg/dl)</b>	10.7 ± 0.87	9.8 ± 1.47	10.2 ± 1.51	>0.05
<b>Thyroid lead content (ug/g)</b>	8.41 ± 2.2	8.44 ± 2.3	8.35 ± 1.7	>0.05
<b>thyroid malondialdehyde (nmol / g)</b>	5.12 ± 0.11	5.22 ± 0.29	5.16 ± 0.18	>0.05
<b>thyroid superoxide dismutase (U/mg)</b>	1.67 ± 0.11	1.71 ± 0.24	1.75 ± 0.19	>0.05

**Table (2):** Comparison of T3, T4 and TSH values in (– ve) control (I), lead acetate (IV) and lead acetate + selenium (V) groups after 8 weeks.

	Negative control (I) Mean ± SD	Lead acetate (IV) Mean ± SD	Lead acetate + selenium (V) Mean ± SD	P
<b>T3 (ng/ml)</b>	2.87 ± 0.18	1.68 ± 0.22 <sup>a</sup>	2.01 ± 0.25 <sup>b,c</sup>	< 0.0001*
<b>T4 (ng/ml)</b>	33.24 ± 3.81	24.01 ± 2.21 <sup>a</sup>	28.01 ± 2.10 <sup>b,c</sup>	< 0.0001*
<b>TSH (uIU/ml)</b>	1.39 ± 0.24	6.09 ± 0.50 <sup>a</sup>	4.11 ± 0.52 <sup>b,c</sup>	< 0.0001*

\* Significant difference (P < 0.0001) as compared by ANOVA.

**a** Significant difference (P < 0.0001) compared with “control I” group using post hoc LSD test.

**b** Significant difference (P < 0.0001) in T3 and TSH, (P < 0.01) in T4 compared with “control I” group, using post hoc LSD test.

**c** Significant difference (P < 0.01) in T3 and T4, (P < 0.0001) in TSH compared with “lead acetate IV” group, using post hoc LSD test.

n=10 in all groups

**Table (3):** Comparison of blood lead level and thyroid lead content values in (-ve) control (I), lead acetate (IV) and lead acetate + selenium (V) groups after 8 weeks.

	<b>Negative control (I)</b> Mean $\pm$ SD	<b>Lead acetate (IV)</b> Mean $\pm$ SD	<b>Lead acetate + selenium (V)</b> Mean $\pm$ SD	<b>P</b>
<b>Blood lead level (<math>\mu\text{g/dl}</math>)</b>	10.7 $\pm$ 0.87	23.0 $\pm$ 2.14 <sup>a</sup>	16.2 $\pm$ 2.1 <sup>b'c</sup>	< 0.0001*
<b>Thyroid lead content (<math>\mu\text{g/g}</math>)</b>	8.41 $\pm$ 2.2	37.12 $\pm$ 3.14 <sup>a</sup>	23.4 $\pm$ 2.2 <sup>b'c</sup>	< 0.0001*

\* Significant difference ( $P < 0.0001$ ) as compared by ANOVA.

**a** Significant difference ( $P < 0.0001$ ) compared with "control I" group, using post hoc LSD test .

**b** Significant difference ( $P < 0.0001$ ) compared with "control I" group, using post hoc LSD test .

**c** Significant difference ( $P < 0.0001$ ) compared with "lead acetate IV" group, using post hoc LSD test .

n=10 in all groups

**Table (4):** Comparison of thyroid (MDA) and thyroid (SOD) values in (-ve) control (I), lead acetate (IV) and lead acetate + selenium (V) groups after 8 weeks.

	<b>Negative control (I)</b> Mean $\pm$ SD	<b>Lead acetate (IV)</b> Mean $\pm$ SD	<b>Lead acetate + selenium (V)</b> Mean $\pm$ SD	<b>P</b>
<b>Thyroid malondialdehyde (nmol / g)</b>	5.12 $\pm$ 0.11	8.03 $\pm$ 0.14 <sup>a</sup>	7.01 $\pm$ 1.0 <sup>b'c</sup>	< 0.0001*
<b>Thyroid superoxide dismutase (U/mg)</b>	1.67 $\pm$ 0.11	0.7 $\pm$ 0.13 <sup>a</sup>	1.04 $\pm$ 0.2 <sup>b'c</sup>	< 0.0001*

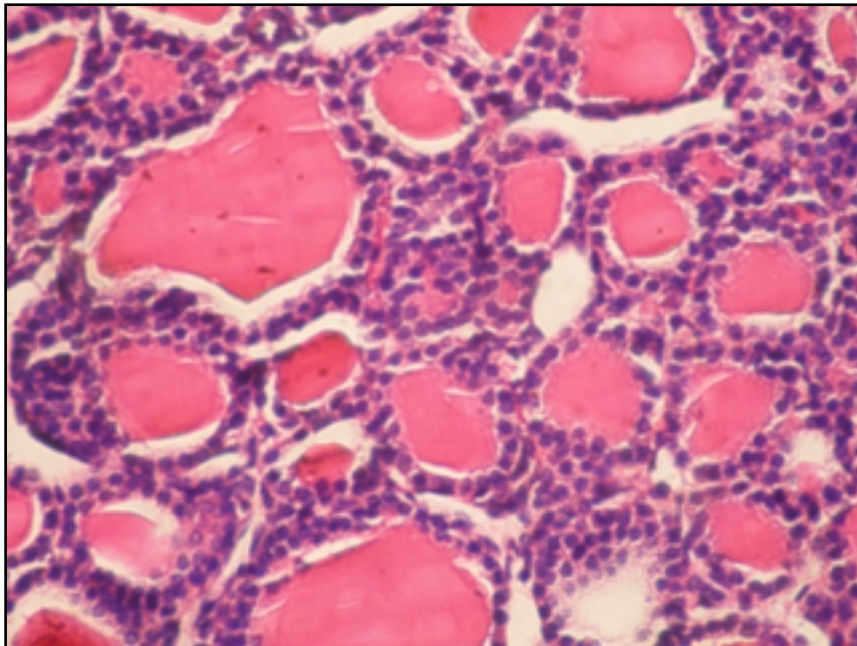
\* Significant difference ( $P < 0.0001$ ) as compared by ANOVA.

**a** Significant difference ( $P < 0.0001$ ) compared with "control I" group, using post hoc LSD test .

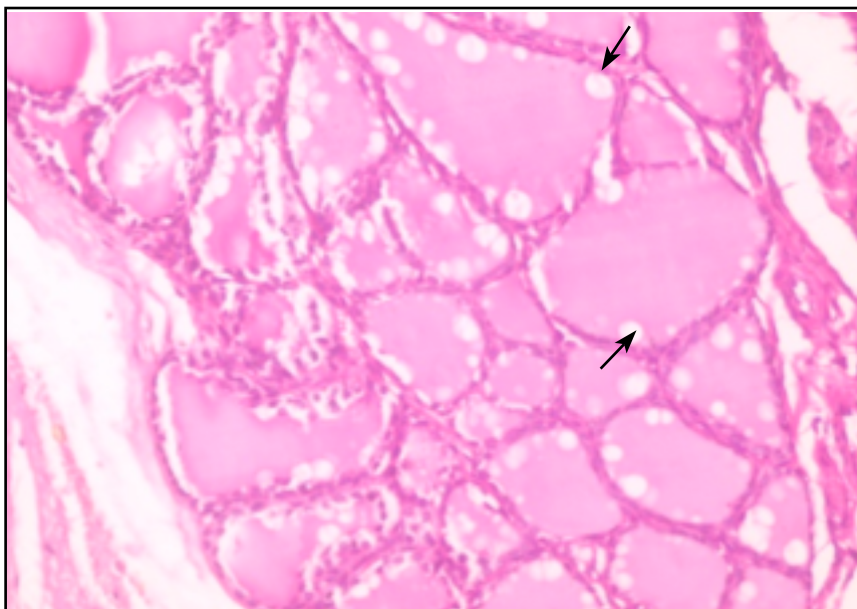
**b** Significant difference ( $P < 0.0001$ ) compared with "control I" group, using post hoc LSD test .

**c** Significant difference ( $P < 0.01$ ) in MDA and ( $P < 0.001$ ) in SOD compared with "lead acetate IV" group, using post hoc LSD test .

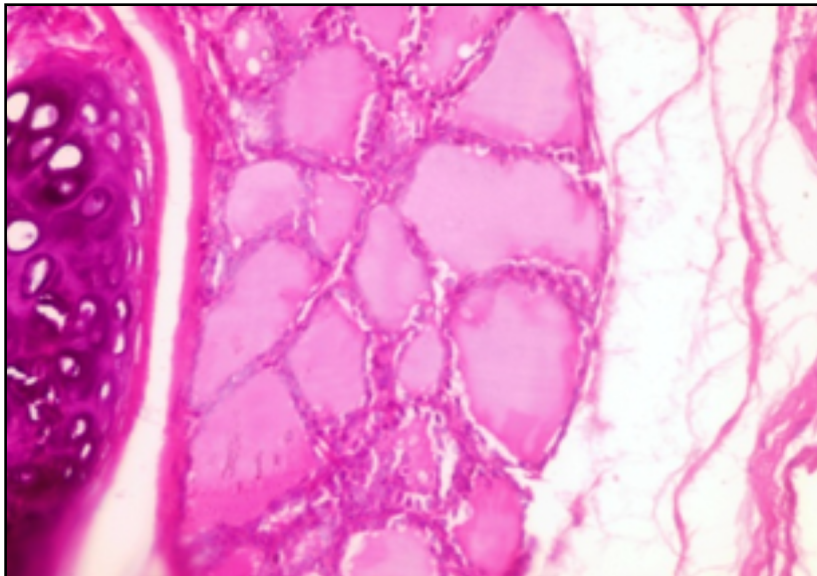
n=10 in all groups



**Figure (1)** : A photomicrograph of section in rat's thyroid of control groups revealed normal thyroid follicles lined with cubical epithelium lie on the periphery of the gland and colloids concentrated in the centre (H&E 400X).



**Figure (2)** : A photomicrograph section in a rat's thyroid of lead acetate group (IV) after 8 weeks showing numerous variable sized thyroid acini lined by hypertrophied columnar epithelium and filled with homogenous eosinophilic colloid material with peripheral scalloping (arrow) (H&E 400X).



**Figure (3)** : A photomicrograph section in a rat's thyroid of (lead acetate + selenium) group (V) after 8 weeks showing some acini lined by cubical epithelium and filled with homogenous eosinophilic colloid material with no peripheral scalloping and some acini lined by cubical to low columnar epithelium and the colloid showed focal peripheral scalloping (arrow) (H&E 400X).

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## تأثير السيلينيوم على التغيرات البيوكيميائية والباثولوجية بالغدة الدرقية الناجمة عن التعرض المزمن لخلات الرصاص فى الجرذان البيضاء البالغة

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استهدفت الدراسة الحالية تقييم تأثير السيلينيوم على وظائف الغدة الدرقية، وكذا التوتر المؤكسد والتغيرات المرضية فى أنسجتها الناجمة عن التعرض لخلات الرصاص بشكل مزمن. لقد أجرى هذا البحث لمدة 8 أسابيع على جرذان التجارب البيضاء البالغة وعددهم 50 جرد وتم تقسيمهم إلى 5 مجموعات كل مجموعة مكونة من 10 جرذان استخدمت المجموعة الأولى كمجموعة ضابطة سالبة (I) والثانية كمجموعة ضابطة موجبة (II) واعطيت المجموعة الثالثة السيلينيوم يوميا بجرعة 0,35 مجم/كجم من وزن الجسم كمجموعة ضابطة موجبة (III) واعطيت المجموعة الرابعة خلوات الرصاص بجرعة (60 مجم/كجم) مجموعة (IV) بينما اعطيت المجموعة الخامسة نفس جرعة خلوات الرصاص السابقة بجانب جرعة السيلينيوم كما فى المجموعتان الثالثة والرابعة. بعد مرور 8 أسابيع تم استخدام عينات الدم وأنسجة الغدة الدرقية من الجرذان لتقييم مستوى هرمونات الغدة الدرقية (تراي أيدودوثيرونين والثيروكسين والهرمون المحفز للغدة الدرقية) وكذلك مستوى الرصاص فى الدم والغدة الدرقية و الدهون فوق المؤكسدة (مالونداي ألدهيد) والإنزيمات المضادة للأكسدة (سوبر أكسيد ديسميوتيز) وأتبع هذا دراسة ميكروسكوبية لأنسجة الغدة الدرقية لتحديد التغيرات الباثولوجية بها باستخدام صبغة الهيماتوكسيلين أوسين وفحصها بواسطة المجهر الضوئى. كشفت النتائج عن انخفاض ملحوظ فى كل من مستوى هرمونات الغدة الدرقية (تراي أيدودوثيرونين والثيروكسين) وارتفاع فى مستوى الهرمون المحفز للغدة الدرقية وكذلك فى مستوى الرصاص فى الدم والغدة الدرقية فى مجموعة خلوات الرصاص (IV) بالمقارنة مع المجموعة الضابطة (I). وقد وجدت زيادة فى كمية الرصاص، وكذلك الدهون فوق المؤكسدة (مالونداي ألدهيد) وانخفاض فى الإنزيمات المضادة للأكسدة (سوبر أكسيد ديسميوتيز) فى أنسجة الغدة الدرقية فى مجموعة خلوات الرصاص (IV). أما عن التغيرات الباثولوجية فى الأنسجة، كان هناك العديد من الحويصلات متغيرة الحجم فى الغدة الدرقية مبطنة بواسطة خلايا عمودية وملبئة بمواد متجانسة غروية مع ظهور فجوات طرفية. ومن ناحية أخرى، أظهرت جرذان المجموعة الخامسة التى تعاطت السيلينيوم مع خلوات الرصاص (V) تحسن فى مستويات هرمونات الغدة الدرقية (تراي أيدودوثيرونين والثيروكسين والهرمون المحفز للغدة الدرقية) وانخفاض فى مستوى الرصاص فى الدم والأنسجة عند مقارنتها مع مجموعة خلوات الرصاص (IV) بالإضافة إلى ذلك، أنخفض مستوى الرصاص فى الدم والأنسجة وكذلك المألون. داي ألدهيد فى أنسجة الغدة الدرقية وزيادة فى النشاط الإنزيمات المضادة للأكسدة (سوبر أكسيد ديسميوتيز) عند مقارنتها مع مجموعة خلوات

الرصاص (IV) . كما لوحظ أن هناك تحسن في التغيرات الباثولوجية بالأنسجة، حيث كانت هناك بعض حويصلات مليئة بخلايا مكعبة مع عدم وجود الفجوات الطرفية.

نخلص من ذلك إلى أن السيلينيوم له تأثير ايجابي على التغيرات البيوكيميائية والباثولوجية في الغدة الدرقية الناجمة عن التسمم بخلات الرصاص.