EFFECT OF LEAD, CADMIUM AND THEIR COMBINATION ON THE TESTES AND SEMINAL VESICLE OF RATS

BY

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ABSTRACT

This study was done to evaluate the effect of lead or cadmium, and their combination on testes and seminal vesicles of male albino rats because workers in many industries may exposed to them together. A total of 24 healthy adult male albino rats were used divided into 4 groups, 6 rats in each group. The control group was injected intraperitoneally (Ip) by distilled water daily for 21 days. The second group was injected Ip by 10 mg/kg lead acetate dissolved in distilled water daily for 21 days. The third group was injected Ip by 0.5 mg/kg cadmium chloride (CdCl2) dissolved in distilled water daily for 21 days. On the other hand the fourth group was injected Ip by combination of 10 mg/kg lead (Pb) acetate and 0.5 mg/kg cadmium chloride (CdCl2) dissolved in distilled water daily for 21 days. The rats were sacrificed after twenty four hours from the last dose. The weight of testes and seminal vesicles were measured and compared to control group. The level of reduced glutathione and catalase, as a protective marker against oxidative stress in the tissue of testes and seminal vesicles were also measured and compared to control group. Then testes and seminal vesicles prepared for histopathological examination. Sections stained with eosin and hematoxylin stain and examined under light microscope. The results of this study revealed presence of a significant decrease in the weight of testes and seminal vesicles in rats treated with lead acetate or cadmium chloride and their combination. Furthermore the level of reduced glutathione and cataluse of testes and seminal vesicles in rats treated with lead acetate or cadmium chloride and in combination compared to control group were significantly decreased. In addition, there was degenerative changes in structure, size, and shape of cells of testes and seminal vesicles in rats treated with lead acetate and cadmium chloride either alone or in combination. The results of this study revealed no additive effect resulting from the combination of lead acetate and cadmium chloride in all biochemical and histological examination.

Key words: Lead, cadmium, testes, seminal vesicles, antioxidants.

INTRODUCTION

Levels of heavy metals in air, water, and soil have been increased during the last years, both in urban and rural areas. In terms of potential adverse effects on animals and human health, cadmium and lead are amongest the elements that have caused most concern. They are considered the major contaminants of our environment (Tätrai et al., 2001). The involvement of heavy metals including lead and cadmium has been implicated in the aetiology of male infertility (Batra et al., 2001).

Exposure to lead continues to be a widespread problem. The general population may get exposed to lead due to food or water contamination, and air pollution caused by industrial emission and gasoline containing lead compounds. Lead is one of the most widely used metals in industries in many countries. Batteries, paints, pigments, plastic, ceramic, secondary foundries and welding being the most important occupational setting (Ercal et al., 1996). Environmental emission of lead (Pb) have been reduced in many countries, nevertheless there is still public concern about exposure to toxic effects of Pb upon the general population, especially developing countries (Hernández-Ochoa et al., 2005). Environmental exposure to toxic levels of lead occurs in a number of industries with potential adverse effects on the reproductive capacity

of exposed men (Abul-Nasr et al., 1999). The consequence of lead in male reproductive system is far-reading and is attracting increasing attention in the recent times (Wang, 2006).

Most data are consistent with the hypothesis that lead toxicity is directed at the hypothalamic pituitary axis. In addition to this CNS effects, a direct testicular toxicity may occur. A number of investigators has reported the toxicity of lead on the testes and spermatogenesis (Sokol et al., 1994). Lead has been correlated with reduced human semen quality (Alexander et al., 1996). Emerging data suggests that some of the effects of lead on testes may be due to production of reactive oxygen species (ROS) (Mariola et al., 2004).

Cadmium represents a serious industrial and environmental pollutant. Its toxicity and adverse effects have evoked a great concerns about the concequences of cadmium exposure to human (Vtdislav et al., 2006). It is a typical cummulative xenobiotic with extremely long biological half life. It accumulates in human tissues particularly in liver, kidney, heart, lungs and testes (Nogyova et al., 1994).

Workers engaged in occupations such as electroplating, plastic, cement and phosphate fertilizers could be exposed to large amounts of Cd (Cook and Morrow, 1995). Cigarette smoke is a large source of Cd intake. It is also released to the air, land and water by human activities during combustion of coal and mineral oils, smelting, alloy processing and minimg operations (Patra et al., 1999). The mechanisms by which Cd induces its toxic effects are quite variable. The toxicity and carcinogenicity induced by cadmium involve an oxidative stress with subsequent oxidative tissue damage (Stohs et al., 2001).

The aim of this study was to evaluate the effects of lead or cadmium, and their combination on the weight, antioxidant markers (catalase, and intracellular reduced glutathione), and histological structure of testes and seminal vesicles, because workers in many industries may exposed to them together.

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MATERIAL AND METHODS

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(1) Chemicals:

Cadmium chloride, reduced glutathione, Ellman's reagent [(5,5-Dithiobis (2-nitrobenzoic acid), DTNB)] and catalase standard were purchased from ICN pharmaceutical company, (USA). Lead acetate was obtained from Kemex Fleming, (Egypt). All other chemicals were of analytical grades.

(2) Animals and Treatments:

Twenty four (24) adult healthy Wister albino rats, with an average weight of 150-

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200 gm were used in this study. They were kept under routine healthy laboratory conditions and were fed normal Purina chow and tap water ad libitum. The animals were divided into four groups, six rats each.

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Group I served as control group and was daily injected intraperitonealy (Ip) with 1ml distilled water which was used as solvent to dissolve all compounds injected in a fixed volume 1ml. Group II was injected Ip by 10 mg/kg lead acetate dissolved in distilled water daily for 21 days (Batra et al., 2001). Group III was injected Ip by 0.5 mg/kg cadmium chloride dissolved in distilled water daily for 21 days (Patra et al., 1999). Group IV was injected IP by 0.5 mg cadmium chloride and 10 mg/kg lead acetate dissolved in distilled water daily for 21 days.

One day after the last injection all rats were sacrificed by decapitation. Testes and seminal vesicles were taken out, weighed and prepared for measurements of antioxidants markers and histopathological examination.

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(3) Measurement of Testes and Seminal Vesicles Weight.

The weight of testes (gm) and seminal vesicles (mg) in all treated groups were weighed and compared to control group by electronic balance (Scaltec, Germany).

(4) Measurements of Antioxidants Markers:

Approximately 0.5 gm sample of testicular tissues or seminal vesicle were removed from each animal, rinsed in ice cold saline and blotted carefully. They were placed in an ice-cold glass homogenizer containing phosphate buffer (at PH 7). After homogenization a part of homogenate was used for estimation of catalase (CAT) activity and the remaining part was used for intracellular reduced glutathione (GSH) estimation. Catalase activity was determined by its ability to decompose H2O2 according to the method of Luck (1963). Immediately before assay, 1:500 dilution of the concentrate tissue homogenates were prepared with phosphate buffer. The activities of CAT were determined using standard solutions of CAT. The levels of enzyme activities were expressed as u/mg protein. For estimation of intracellular reduced glutathione (GSH) an equal volume of 10% metaphosphoric acid was added to the homogenate and mixed by vortexing. The mixure was allowed to stand for 5 min at room temperature. After centrifugation for 5 min, the supernatant was collected carefully without disturbing the precipitate. The GSH contents of the neutralized supernatant was measured by spectrophotometer (LKB Biochrom, England) using Ellman's reagent [(5,5-dithiobis (2-nitrobenzoic (DTNB solution)] according to the method of Griffith (1980). A standard reference

curve was prepared for each assay. Results were expressed as nmol GSH/mg protein. Protein contents were determined using the method of Lowry et al., (1951).

(4) Histopathological Examination:

After sacrification of animals, samples of the testicular tissue and seminal vesicles were fixed in 10% formaline, dehydrated, cleared, embedded in paraffin, and were sectioned at 7um. Deparaffinized sections were stained with haematoxylin and eosin (H.&E. stain) for light microscopical examination (Pearse, 1985).

(5) Statistical Analysis:

Statistical analysis was done using student's t-test by SPSS computer program. The data were presented in the form of mean± standard deviation (SD). P value >0.05 was considered insignificant while P value <0.05 was considered significant. P < 0.001 was considered highly significant.

RESULTS

Table (1) shows the effect of lead, cadmium, and their combination on weight of testes and seminal vesicles. There was significant decrease in weight of testes in comparison with control group. The weight of the testes in control was (1.59 ± 0.09) and the lead acetate treated group was (1.02 ± 0.04) in cadmium chloride treated group was (1.09 ± 0.08) , and in their combination was (0.99 ± 0.09) . The

seminal vesicles weight expressed in mg showed a highly significant decrease in lead acetate treated group (686 \pm 9.37), in cadmium chloride treated group (726.85 \pm 3.88) and in their combination (716.4 \pm 7.66).

Table (2) shows the effect of lead, cadmium, and their combination on CAT activity in testes and seminal vesicles. Activity of CAT was decreased significantly in animals treated with lead acetate (10 mg/kg) to a level of (0.98 ± 0.03) and (0.62 ± 0.06) in testes and seminal vesicles respectively. Catalase activity in rats treated with cadmium chloride (0.5 mg/kg) was significantly decreased in testes to (0.86 ±0.02) and (0.58±0.03) in seminal vesicles. Co-administration of lead acetate and cadmium chloride also significantly decreased the CAT activity in both testes and seminal vesicles.

Table (3) shows the effect of lead, cadmium, and their combination on reduced GSH of testes and seminal vesicle. The intraperitoneal injection of 10 mg/kg lead acetate in rats produced significant decrease of reduced GSH in testes (24.23 \pm 2.07) and (16.2 \pm 1.05) in seminal vesicles. After injection of 0.5 mg/kg cadmium chloride the level of GSH was (27.9 \pm 2.06) and (15.4 \pm 1.46) in testes and seminal vesicle respectively. Co-administration of two metals produced also significant decrease in GSH level in both organs (23.78 \pm 2.18)

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in testes and (14.36 ± 1.23) in seminal vesicles.

The histological examination of the testicular specimens obtained from animals injected with lead acetate (10 mg/kg daily for 21 days) showed distortion of shape and size of seminiferous tubules with disorganization and vacuolation of spermatogenic cells (Plate-1, Figs. 2-4)

Cadmium chloride administration (0.5 mg/kg daily for 21 days) produced more damage in the form of atrophy of seminiferous tubules, obliteration of lumen of tubules with marked fibrosis in the intertubular spaces. Abnormal fusion between the tubules and small sized destructed tubules were also present (Plate-2, Figs. 5-7)

Co-administration of lead acetate and cadmium chloride produced degeneration, disorganization and sloughing of germinal epithelium with arrest of spermatogenesis but there was no additive or synergistic effect in this group in comparison with lead acetate or cadmium chloride administered alone (Plate-3, Figs. 8-9).

The histological examination of the seminal vesicles of rats treated with lead acetate 10 mg/kg daily for 21 days showed destruction of the epithelial lining cells with complete absence of the glandular secretion (Plate-4, Figs.11-12). Cadmium chloride administration in a dose level

0.5 mg/kg daily for 21 days produced marked reduction in the mucosal folds, with decrease in the secretion (Plate-5, Figs. 13-15). Co-administration of lead and cadmium showed changes equal to cadmium alone with no additive or synergestic effect (Plate-6, Figs.16-18).

DISCUSSION

Lead salts are among the oldest known spermicidal agents. Evidence of the deleterious effects of lead on human reproduction dates back to the ancient Roman and Greek civilization (Gilifilian, 1965).

The present study, showed significant decrease in weight of testes and seminal vesicles. Histopathological examination of testes in lead treated animals showed distortion of shape and size of seminiferrous tubules with degeneration of the germ cells. The seminal vesicles treated with lead appear with absence of glandular secretion.

Parallel to these structural effects there was significant decrease in the enzymatic (catalase) and non enzymatic (intracellular reduced glutathione) antioxidant markers in the testicular tissues.

In agreement with the present study, Wang et al., (2006) reported that mice treated with lead showed thin seminiferous tubules and disappearance of most spermatids. Leydig cells showed shrunken cells with pyknotic nuclei and evidence of destruction.

Batra et al., (1998) postulated that the testicular histoarchitecture may be due to accumulation of lead in testicular tissue.

An emerging data suggest that some of the effects of lead on testes may be due to production of reactive oxygen species (ROS) (Mariola et al., 2004). On the other hand Marchlewicz et al., (1993) reported that the lead content in testes of the animals treated with lead acetate did not differ significantly from the value of this element in gonads of control rats. Opposite to the results of the present study Murthy et al., (1991), Wenda-Rozewicka et al. (1996) and Richter et al., (1997) concluded thats no changes of testes and epididymis were detected by histological observation, in spite of marked accumulation of lead in blood and testicular tissues which leads to changes in spermatogenesis

Thoreux-Manlay et al., (1995) reported that germ cells and Sertoli cells did not appear to be the major targets of lead but the accessory sex glands are such a target. Also, Boscolo et al., (1988) stated that lead was not augmented in testes and examination by light microscopy did not reveal alterations. Ultrastructural examination of the testes with both transmission and scanning electron microscopy did not evi-

dence modifications in the external part of the seminiferous tubules in spermatogenic cells and in connective tissue including the leydig cells.

Corpas et al., (1995) reported that relative testicular weight and gross testicular structure were not altered by lead treatment. These results were opposite to the present study which showed significant decrease in testicular weight.

Similar to the results of the present study Pinon-Lataillade et al. (1993) reported that after lead acetate ingestion seminal vesicle weight dropped significantly which might suggest an alteration in the pattern of testosterone secretion.

Lahadelie, (1995) stated that lead acetate leads to atrophy of seminiferous tubules with inhibition of spermatogenesis but no change in weight of testes and seminal vesicles.

Janecki et al., (1992) reported that single injection of CdCl2 at a dose as low as 20 umol/kg caused a rapid and severe testicular oedema followed by hemorrhage and testicular necrosis. With moderate doses of CdCl2, the spermatogenic function recovered after about 1 month whereas the higher doses result in irreversible sterility.

El-Ashmawy and Youssef, (1999) re-

ported that exposure to CdCl2 significantly decreased the weight of testes, epididymis and accessory sex organs and produced degenerative changes in testes, epididymis and seminal vesicles.

The present study demonstrate that cadmium treated testes showed more damage than lead, in the form of atrophied seminiferous tubules, obliteration of lumen of tubules with marked fibrosis. These were associated with significant decrease in enzymatic (catalase) and non enzymatic (reduced glutathione) antioxidant markers and significant decrease in weight of either testes or seminal vesicle with apparent equality between the cadmium group and co-administration group. Lead produced lesser effects.

Koyuturk et al., (2006) agreed with the present results as they stated that there was serious damage in the integrity of spermatogenic cells of seminiferous tubules and also necrotic cells and debris were examined in the seminiferous tubules. Contrary to the present study Francavilla et al., (1981) showed an increase in testicular and epididymel weight due to edema. While Teiichiro et al., (2002) reported that no changes in testicular weight have been reported in some subchronic cadmium intoxications

In agreement with the present study results, Kojima et al., (1992), reported that

the testicular weight decreased after 5 days of cadmium administration which indicate that testicular damage involves the initial hemorrhagic necrosis followed by testicular atrophy. Also Yang, et al., (2003) mentioned that cadmium is directly toxic to primary cultured Leydig cell in vitro. toxicity includes reduced cell viability and testosterone secretion, increased lipid peroxidation, decreased antioxidative ability, and DNA damage. Also study of Biswas et al., (2001) showed a marked testicular hypoplasia, and reduction in testicular weight have been attributed to the necrotic and degenerative cadmium-induced changes.

The current study shows that glutathione (GSH) is most abundant cellular thiol, which serves to protect against various forms of metal toxicity as well as cadmium (Dalton et al., 2004). The activity of antioxidant enzymes such as catalase, glutathione reductase, and superoxide dismutase, was decreased in lead-exposed rats and levels of the antioxidant molecule GSH were strongly correlated with lead intoxication (Hsu, 1981). GSH is also known as free radical scavenger and potent inhibitor of LPO. It was also demonstrated that GSH levels might be direct or indirect targets of Cd exposure in testicular tissue (Arthur, 2000)

Similar to the present results, Liu et al., (2001) and Koyuturk et al., (2006) stated

that histopathologic changes in testes are associated with reducing GSH and increasing lipid peroxidation upon CdCl2 administration. The co-administration of lead and cadmium was investigated in different organs by different authors. Lead as an environmental contaminant is often combined with cadmium which has effects similar to those of lead so that their effects are additive (Liu, 2003).

Pillai and Gupta (2005) reported that various oxidative stress parameters in the liver of rats co-exposed to lead and cadmium may result from an independent effect of lead and cadmium and also from their interaction such as changes in metal accumulation and content of essential elements like Cu, Zn, and Fe. Khare et al., (1978) reported that there was no synergistic effect of lead acetate and cadmium chloride injected into the prostate.

Nampoothiri and Gupta (2006) concluded that lead and cadmium in isolation and in combination cause oxidative stress. They also stated that lead and cadmium in combination did not show additive or synergistic effect in studied organs indicating the competition between them due to similarity in electronic affinities.

Kaczmarek-Wdowiak et al., (2004) reported that single and combined exposure to small doses of lead and cadmium in rats results in the changed parameters

showing the stimulation of lipid peroxidation and antioxidant system along with interaction in their influence.

The effect of lead and cadmium on the hypothalamic pituitary axis were studied in proestrous rats by Pillai et al., (2003). They concluded that the effects produced by the combined treatment of metals are not additive.

Skoezynska et al., (1994) mentioned that changes in the copper and zinc concentrations in tissues in the combined exposure to lead and cadmium are similar to those induced by single lead (heart and brain) or single cadmium (liver and kidney) intoxication.

Molinero et al., (1999) reported that there was no any gross structural changes in testes of Pb - Cd treated groups but the total number of prospermatogonies were increased and the diameter of seminiferous tubules were decreased in treated groups. Their results suggest that the coadministration of both cations (Pb and Cd) produced significant alterations in testicular function.

Der et al., (1976) revealed that after 20 days of injection of rats with 25 ug of both lead and cadmium, testes showed absence of spermatogenesis in some seminiferous tubules indicating that injection of low levels of lead and cadmium together have

a more synergetic damaging effect on rat testes than higher levels of lead or cadmium alone.

In the present study the changes in various biochemical parameters or histopathological structure of the testes and seminal vesicles of male rats exposed to lead and cadmium might result from independent individual effect of lead or cadmium and also from their interaction.

The present study revealed that combined exposure of male rats to lead and cadmium showed effect parallel to the effects of cadmium alone rather than lead alone at the level of histopathology of testes. The seminal vesicles showed equal structural changes in cadmium and coadministrated group while lead alone produced minimum effect. At the level of antioxidant markers there was no difference in the three examined groups either in testes or seminal vesicles. These results indicates that when cadmium and lead are present together, cadmium mediates major effects due to its more reactive nature.

CONCLUSION AND RECOMMENDATIONS

In conclusion, co-exposure to lead and cadmium produce major changes in male rat sex organs (testes and seminal vesicles) and decrease in antioxidant states (reduced glutathione and catalase). There was no synergistic or additive effect in testes and seminal vesicle as described on many other organs by different authors.

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and cadmium prevention programs. Periodic examination of workers exposed to lead and cadmium with investigation of their reproductive function are recommended.

Table (1): Effect of cadmium, lead and their combination on the weight of testes (gm) and seminal vesicles (mg) of rats.

Group Organ	Control	Pb	Cd	Cd + Pb
Testes	1.59 <u>+</u> 0.09	1.02 ± 0.04**	1.09 ± 0.08**	0.99 <u>+</u> 0.09**
Seminal vesicles	808.85 <u>+</u> 5.57	686 <u>+</u> 9.37**	726 <u>+</u> 3.88**	716.4 <u>+</u> 7.66**

The result represent mean \pm SD

Table (2): Effect of cadmium, lead and their combination on CAT activity (U/mg protein) in testes and seminal vesicles of rats.

Group Organ	Control	Pb	Cd	Cd +P b
Testes	1.86± 0.02	0.98 ± 0.03**	0.86 ± 0.02**	0.91 ± 0.05**
Seminal vesicles	0.91 ± 0.04	0.62 ± 0.06**	0.58 ± 0.03**	0.59 ± 0.02**
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The result represent mean \pm SD

Table (3): Effect of cadmium, lead and their combination on reduced GSH level (nmol/mg protein) in testes and seminal vesicle of rats.

Group Organ	Control	Pb	Cd	Cd + Pb
Testes	41.82 ± 3.62	24.23±2 .07**	27.9±2.06**	23.78±2.18**
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Seminal vesicles	20.80 ± 1.83	16.2 ± 1.05**	15.4± 1.46**	14.36±1.40**
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The result represent mean \pm SD

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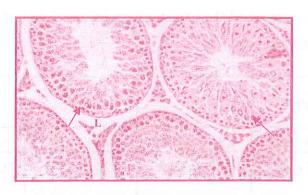


Fig (1): Control Testis

A photomicrograph of a section in the rat testis of a control group showing seminiferous tubules ST with different stages of spermatogenic cells(†) and intact leydig cells (L.C.). (H & E X 400)

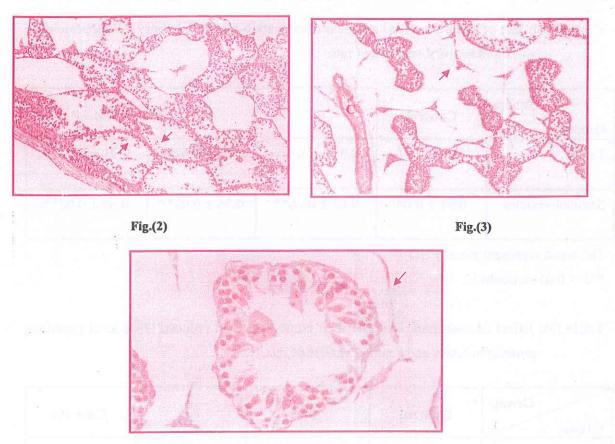


Fig.(4)

Plate-1 Figures 2-4, Photomicrographs of sections in rat testis of group (I) rats showing:

Figure (2): Degenerated spermatogenic cells (↑) with decrease in the number of germ cells in some small seminiferous tubules (ST). (H & E X 100).

Figure (3): Distorted seminiferous tubules with congested blood capillaries (C), shrunken leydig cells (↑) with dense nuclei hunged in the oedematous intertubular spaces. (H & E X 100).

Figure (4): Distortion in shape and decrease in size of seminiferous tubules, disorganized spermatogenic cells with vacuolated cytoplasm, dense nuclei and detached myoid cells (†). (H & E X 400).

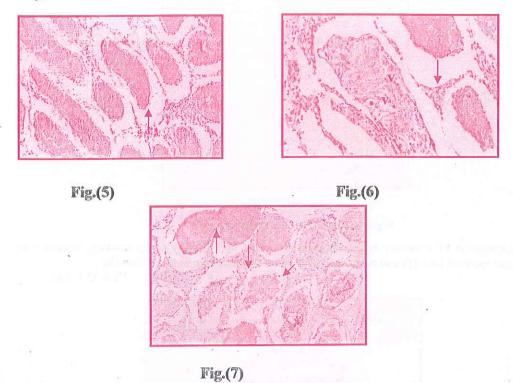


Plate-2, Figures 5-7: Photomicrographs of a sections of the testis of group II rats showing:

Figure (5): Distortion of the germinal epithelium lining the atrophic seminiferous tubules (†). (H & E X 100). Figure (6): Obliteration of the lumen of seminiferous tubules, shrunken, haphazardly arranged reduced germ cells with small sized dense nuclei and empty spaces between the degenerated cells (V). Leydig cells appeared with small dense nuclei (†). (H & E X400).

Figure (7):. Abnormal fusion between the tubules (†) and small sized destructed tubules with separation of myoid cells (†) with marked fibrosis and cellular infiltration in the intertubular spaces. (H & E X 400).

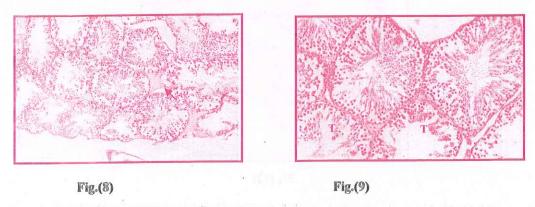


Plate-3, Figures 8-9: Photomicrographs of sections of the testis of group (III) rats showing:

Figure (8): Seminiferous tubules with advanced degeneration of the germinal epithelium. Widespread deposition of acidophilic exudates (↑) in the intertubular spaces is present. (H & E X 100).

Figure (9): Disorganization, sloughing of spermatogenic cells and their exfoliation in the lumen of the oedematous seminiferous tubules with arrest of spermatogenesis in some tubules (T). (H & E X 200).

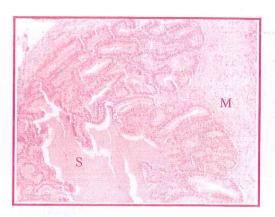


Figure (10): Control of seminal vesicle

Photomicrograph of a section in the rat seminal vesicle of a control group showing mucosa with intact mucosal folds(†) and musculosa (M). Note: the secretion in the lumen (S).

(H & E X 200)

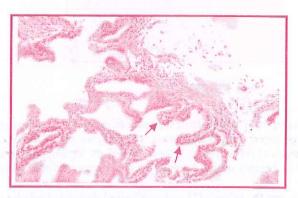


Fig.(11)



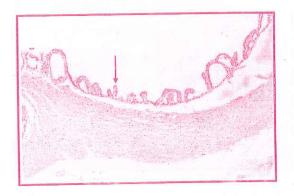
Fig.(12)

Plate-4, Figures, 11-12: Photomicrographs of a sections in the seminal vesicle of group (I) rats showing:

Figure (11): Decrease in the height of the mucosal folds (†) and absence of the glandular secretion, the epithelial cells appeared with small and dense nuclei.

(H & E X 200).

Figure (12): focal destruction in the epithelial lining cells(↑), dense, nuclei in the resting cells and foam cells (↑) and complete absence of the glandular secretion. (H & E X 400).



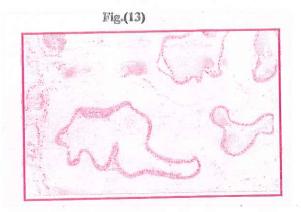


Fig.(14)

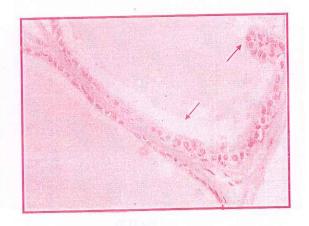


Fig.(15)

Plate-5, Figures, 13-15: Photomicrographs of a section in the seminal vesicle of group II rats showing: Figure (13): Marked reduction in the mucosal folds (↑) and absences of the secretion . (H & E X 100).

Figure (13): Marked reduction in the mucosal folds (1) and absences of the secretion. (11 to 2 × 100).

Figure (14): The mucosal folds of the vesicle exhibited decrease in the height of the epithelial cells and decrease in the

secretion, with oedema around the mucosal folds.

(H & E X 100)

Figure (15): Decrease or complete loss of mucosal folding (†).

(H & E X 400).

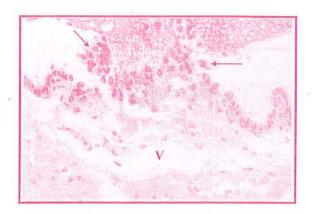


Fig.(16)

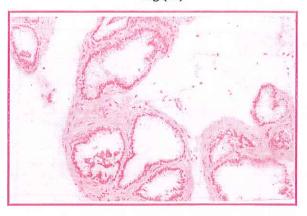


Fig.(17)



Fig.(18)

Plate-6, Figures, 16-18: photomicrographs of sections in the seminal vesicle of group III rats showing:

Figure (16) Destruction and disorganization in the lining epithelial cells (†). and decrease in the mucosal folds with vacuolation in the lamina propria (V). (H & E X 200).

Figure (17): The mucosal folds surrounded by oedema, the lining epithelial cells decreased in the height with absence of glandular secretion. (H & E X 200).

Figure (18): destruction in the epithelium in some areas, exfoliation of some cells in the lumen (†) and focal degeneration with vacuolation in the musculosa (V). (H & E X 400).

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تأثير عنصرى الرصاص والكادميوم بصورة منفردة و مجتمعين على الخصيتين والحويصلة المنوية في الجرذان

المشتركون في البحث

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أجرى هذا البحث لدراسة تأثير التعرض لمعدنى الرصاص والكادميوم كل على حدة أو مجتنعين على الخصيتين والحويصلات المنوية فى ذكور الفئران البيضاء وذلك لتعرض كثير من عمال المصانع للمعدنين مجتمعين وقد أجرى هذا البحث على أربعة وعشرون فأراً من ذكور الفئران البيضاء السليمة البالغة والتي تم تقسيمها إلى أربعة مجموعات تضم كل منها ستة فئران: المجموعة الأولى قمثل المجموعة الضابطة حيث تم حقن الفئران بواحد مللى ماء مقطر نقى في التجويف البريتوني لكل فأر يومياً لمدة ٢١ يوماً، المجموعة الثائية تم حقنها بكلوريد في الماء المقطر النقى بجرعة ١٠ مجم/كجم من وزن الفأر في التجويف البريتوني يومياً لمدة ٢١ يوماً، المجموعة الثالثة تم حقنها بكلوريد الكادميوم المذاب في الماء المقطر النقى بجرعة ٥٠ مجم / كجم بالتجويف البريتوني يومياً لمدة ٢١ يوماً.

تم ذبح الفئران بعد ٢٤ ساعة من آخر جرعة تم حقنها وتم إستنصال الخصيتين والحويصلات المنوية من الفئران بالمجموعات الأربعة وتم قياس أوزان الخصيتين والحويصلات المنوية وقياس مستوى الجلوتاثيون المختزل والكاتاليز بنسيجهما كمؤشر بيوكيميائي على مضادات الأكسدة، وكذلك تم تجهيز أنسجة الخصيتين والحويصلات المنوية للصباغة بادة هيماتوكسلين وأيوسين للفحص الهستوباثولوچي بالميكروسكوب الضوئي.

وقد أثبتت النتائج الإحصائية لهذا البحث وجود نقص ذو دلالة إحصائية في وزن الخصيتين والحويصلات المنوية في المجموعات التي تم حقنها بخلات الرصاص وكلوريد الكادميوم بصورة منفردة أو سوياً وكذلك وجود نقص ذو دلالة إحصائية في مستوى الجلوتاثيون والكاتاليز بأنسجة الخصية والحويصلات بأنسجة الخصية والحويصلات بأنسجة الخصية والحويصلات المنوية من حيث التركيب والشكل والحجم ولكن التأثير الثنائي لم يكن أكثر وضوحاً من تأثير كل منهما على حدة.

ومن خلال هذه النتائج يوصى بوضع برامج بيئية قومية للحد من تعرض الناس للتلوث بالرصاص والكادميوم في البيئة وخاصة عمال المصانع ومحطات البنزين لما في ذلك من خطورة على الإخصاب والقدرة الإنجابية للذكور ويراعي هذا التأثير عند إجراء الفحص الدوري على العمال.