The Egyptian Journal of Hospital Medicine Vol., 19: 189 – 197 June 2005
I.S.S.N: 12084
1687- 2002

Histological Study Of The Effect Of Zinc Sulphate On The Toxicity Of Aluminium Sulphate In Liver And Kidney Of Male Albino Rats

Azza M. Gawish
Department of zoology, Faculty of Science, Cairo University, Cairo, Egypt

Abstract

Aluminium (Al) is one of the most abundant and important elements in the environment. In recent years, the production of this metal and its toxicity increased with its discharge into the environment. This study was designed to evaluate the effect of Al intake toxicity in liver and kidney tissues of albino rats and the role of zinc as a protective agent against Al toxicity.

Fourty five male albino rats were divided into equal three groups. The first group of animals was considered as control. The animals in the second group were given (50mg/kg/day) of Al sulphate orally using gastric tube for 45 day. Third group were given Al sulphate (50mg/kg/day) followed by zinc sulphate (50mg/kg/day) orally as well as samples of liver and kidney tissues were obtained after 15, 30, 45 days of last doses respectively. Paraffin sections (5μm) were prepared for histological study stained with Haematoxylin & Eosin.

The obtained histological results of the histological study of the second group showed that there were congested blood sinusoids and swelling of some hepatocytes within cytoplasm in which there were vacuoles, fragmented nuclei with some cellular infiltration. Kidney tissue showed shrinkage of some glomeruli and distortion of the tubular epithelial cells. Results in the third group, where zinc sulphate was added, showed amelioration and improvement in both liver and kidney tissues.

Introduction

Aluminium is one of the most abundant metal in the earth crust. Daily human exposure of Al ranges from 1.53 to 16 mg /person/day (Domingo, 1995 and Jeffery *et al*., 1996). Al is present in water in the form of hydroxides, fluorides and sulphates (Hava *et al*., 1985, Goyer, 1996 and Alleva *et al*., 1998). Exposure to Al has recently been implicated in a number of human pathologies and symptoms of obstructive lung disease were recorded (Sjoren, 1988), samples of welders and many workers showed high deposition of Al salts in different tissues (Wennberg, 1998, Mur *et al*., 1998).

According to Massie *et al.*, (1988); accumulation of Al has been found in liver and kidney but not in brain of mice given Al chloride (McDermott *et al*., 1979 and Markesbery *et al*., 1981). Weilhelm et al., (1996) declared that the lowest level of Al occurring in cytoplasm in the subcellular fractions might lead to accumulation of Al in the liver. Other study reported that moderate toxicity of Al compounds appeared in the hepatocytes when given in amounts near to pathological values (Muller *et al.*, 1990). Van der Voet *et al*., (1992) reported that Al accumulation was correlated with the appearance of periportal giant cells due to Al administration in rabbits. Degeneration and some changes in the hepatic parenchyma were observed when male rats were given Al chloride (Ebina *et al*., 1984).

Bertholf et al., (1989) reported that high concentrations of Al caused necrosis and atrophy recorded in kidney tissue. Reduced renal functions were recorded in infants of human with high Al accumulations in their kidney tissue (Flaten *et al.*, 1996). Light and electron microscopic studies were applied on kidney tissues in which swelling in some cells of the proximal renal tubules and distortion of renal cells of the kidney were observed due to Al administration (Spencer *et al*., 1995).

Recently, the toxic effects of aluminium has been correlated with induction of free

radicals as has been demonstrated by El-Demerdash (2004) and Yossef (2004) who showed that $AlCl_3$ significantly induced free radicals (thiobarbituric acid-reactive substances) and decreased the activity of glutathione S-transferase (GST) and the levels of sulphydryl groups (SH groups) in plasma, liver, brain, testes and kidney of rat and rabbit, respectively.

Zinc, a potent antioxidant, which protects many body systems as liver kidney, brain and testes against free radicals ((Hafiez *et al.*, 1989 & 1990, Mansour *et al.* 1989, Liu & Stemmer (1990a,b), Oteiza *et al.* (1996), Noh & Koo (2001), and Ozkan *et al.* (2004). Liang *et al.*, (1999) declared that zinc enter gastrointestinal tract as a compound of metallothionine secreted by salivary gland.

Zinc administration affects the Al accumulation in brain hippocampus and liver (Liang *et al.*, 1999). Zinc supplementation may vary the effect of other metals as copper, Al in which Mendez-Alvarez *et al.*, 2002 recorded that zinc alter the toxicological effect of Al.

Material & Methods

Fourty five adult male albino rats weighing 160-170 were supplied by the breeding unit of Holding Company for Biological Product &Vaccines (VACSERA) and feed a diet and free access of tap water and kept under standard experimental conditions.

Chemicals

Chemicals used were Al sulphate ($Al_2(SO4)_3.15H2O$) and zinc sulphate ($ZnSO_4.7H2O$); both were supplied as white powder soluble in water. Aluminium sulfate and zinc sulfate were products of Sigma Co., USA. The calculated dose of Al sulphate (50mg /kg/day) was recorded according to (Domingo J. L. 1995) and Zinc sulphate (50mg/kg/day) according to (Fortes *et al.*, 1997).

Animal groups and experimental design:

The animals were divided into 3 equal groups, each of 15 rats: Group (1) control group.

Group (2): Animals of this group treated with (50mg/kg/day) of Al sulphate orally using gastric tube foe 45-day daily. Group (3): animals treated with Al sulphate (50mg/kg/day) followed by dose of zinc sulphate (50mg/kg/day) an hour later.

Rats of each group were sacrificed at 15, 30, and 45 periods. Samples of liver and kidney were fixed in aqueous Bouin solution for histological studies. 6 µm thickness paraffin sections were prepared and stained with Ehrlich Haematoxylin & Eosin according to (Bancroft & Gamble, 2002)

Results

Histological results: 1- Liver

Control liver:

Light microscope examinations of the control liver tissue showed that hepatic lobules extended in strands radiating from the central vein. These hepatic strands are separated from each other by blood sinusoids lined with endothelial cells (fig 1).

Al sulphate treated group:

The liver tissues of these animal group recorded mild histological changes, after 15 days of daily administration of with Al sulphate and some swollen cells and empty area appeared around the nucleus (fig 2); at 30 day interval inflammatory cell and some fragmentation of chromatin materials were appeared (fig 3). At the end of the experiment (45 day) empty spaces within the cytoplasm and dense bodies within the nuclei were appeared compared to the control group (fig 4).

Al suphate – zinc sulphate treated group:

Zinc sulphate supplementation after Al sulphate showed some improvement in liver tissue in which hepatocytes appeared almost and enlarged blood sinusoids normal at 15 day of treatment compared to Al treated group (fig 5). The cytoplasm was densely stai ned and little fragments were observed (fig 6). At the end of the experiment liver tissue was distorted with necrotic nuclei and zinc cannot induce clear effect in liver (fig 7).

II- Kidney

Control kidney

Control section of kidney of albino rats showed renal corpuscles, Bowman s capsule and renal tubules with their cuboidal cell lining. (Fig 8).

Aluminium sulphate treated group:

The kidney of the animals treated with Al sulphate showed various histological changes where slight shrinkage in the corpuscles were appeared within 15 day of treatment (Fig 9), At 30 day of administration the congestion of renal corpuscles were increased continuously (Fig 10), and renal tubules cells loosed their normal shape indicating distortion in their structures at 45 day of administration (Fig 11).

Aluminium sulphate – zinc sulphate treated group

Administration of Al sulphate followed by supplementation of zinc sulphate showed beginning of amelioration of kidney tissue at 15 day of treatment (Fig 12), continued with the effect of zinc, at 30 day of treatment less shrinked corpuscle was observed (Fig 13); but at the end of experiment (45day) the eff-ect of Al was still affected the renal tubules and corpuscles in which zinc cannot appear improvement on kidney tissue (Fig 14).

List of figures

Fig (1): photomicrograph of liver section of control group showing normal liver cells and hepatic sinusoids 400X.

Fig (2): photomicrograph of liver section of animal daily treated with Al sulphate for 15 day showing some granules within the cells with some vacuoles (arrow) and enlarged nuclei (400X).

Fig (3): photomicrograph of liver section of animal daily treated with Al sulphate for 30 days undefined cell membrane and granulated cytop-lasm (arrow) and dense chromatin materials were observed (400X).

Fig (4): photomicrograph of liver section of animal daily treated with Al sulphate for 45 day showing degenerated cells (400X).

Fig (5): photomicrograph of liver section of animal daily treated with Al sulphate followed by zinc sulphate for 15 day showing nearly normal appearance of hepatic cells (400X).

Fig (6): photomicrograph of liver section of animal daily treated with Al sulphate followed by zinc sulphate for 30 day revealed congestion in hepatic sinusoids and large empty spaces within the cells and few fragments inside the nuclei (400).

Fig (7): photomicrograph of liver section of animal daily treated with Al sulphate followed by zinc sulphate for 45 day showed dark dense chromatin bodies and undefined to the outer border of the cells (400).

Fig (8): photomicrograph of kidney section of control group showing normal kidney tissue and normal renal corpuscles (400X).

Fig (9): photomicrograph of kidney section of animal treated with Al sulphate for 15 day showing nearly normal kidney and some swollen glomerulus (400X).

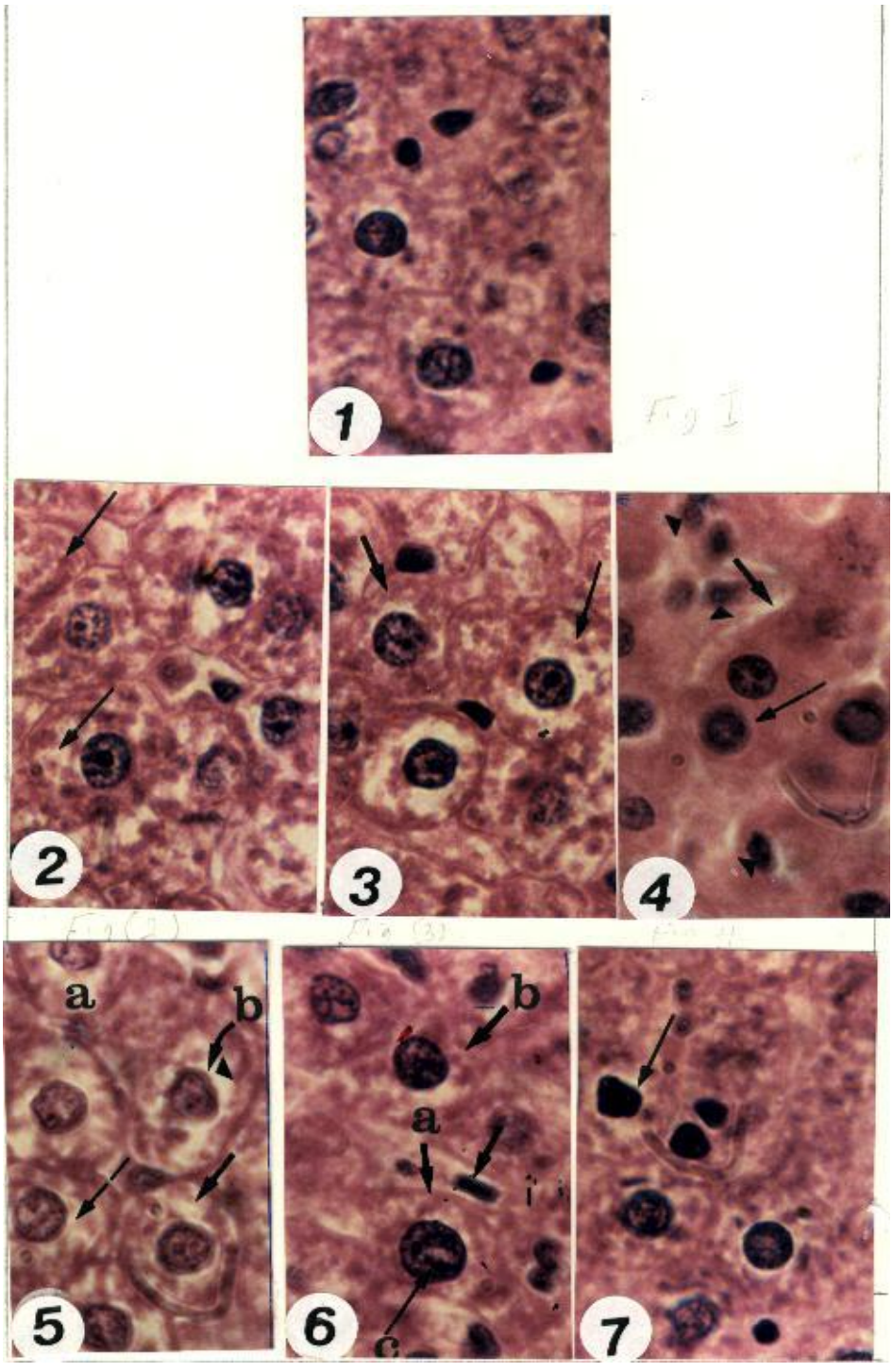
Fig (10): photomicrograph of kidney section of animal treated with Al sulphate for 30 days showed shrinkage in the corpuscles with some infiltra-tion of lymphocytes (400X).

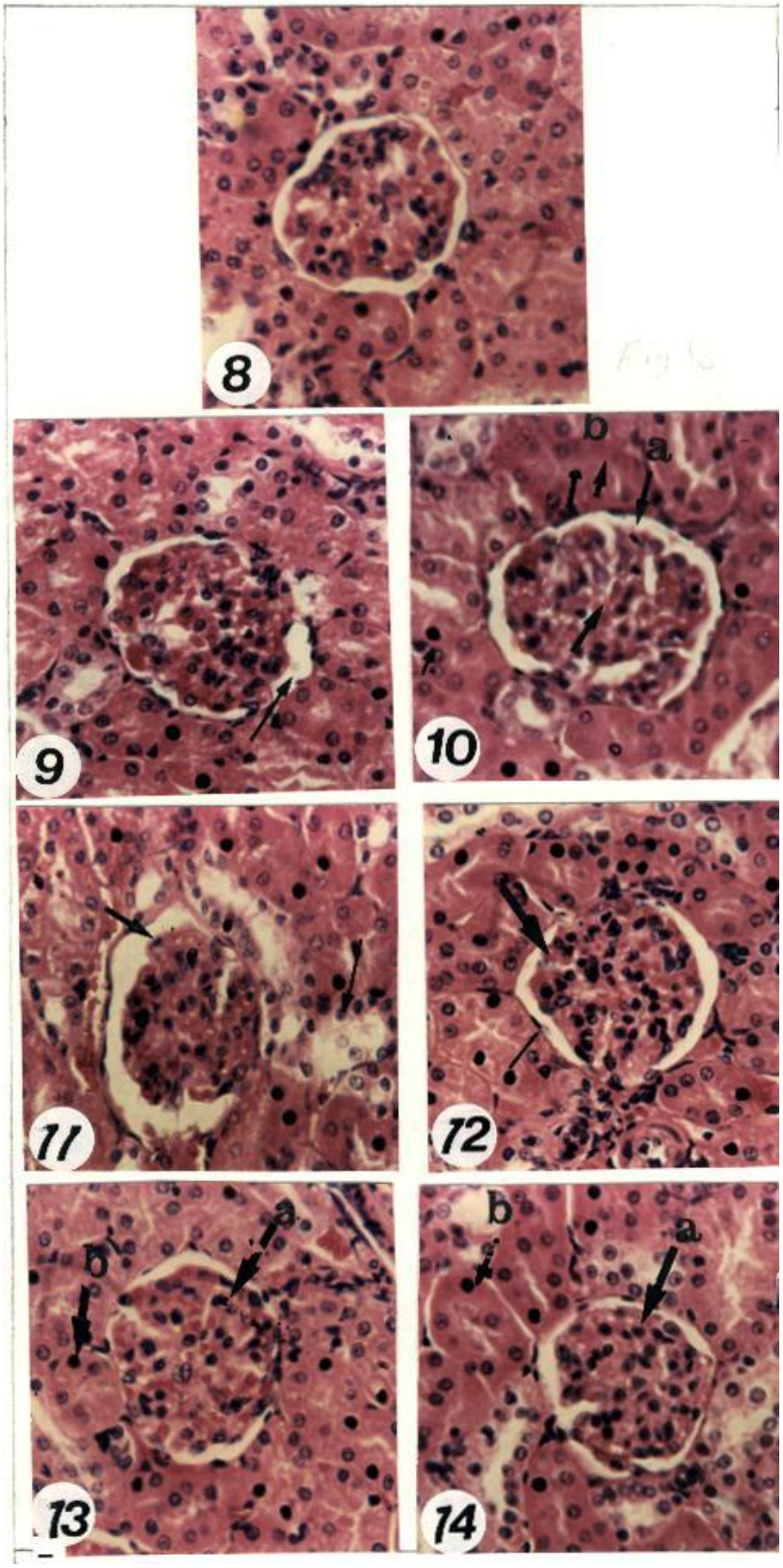
Fig (11): photomicrograph of kidney section of animal treated with Al sulphate for 45 days showed congested renal corpuscle and dead cells nuclei (400X).

Fig (12): Photomicrograph of a kidney section of animal treated daily with aluminium sulphate and supplem-ented with zinc sulfate for 15 successive days showing nearly normal appearance of the glomerulus and the tubule cells (400X).

Fig (13): Photomicrograph of a kidney section of animal treated daily with aluminium sulphate and supplem-ented with zinc sulfate for 30 successive days showing some ameliorating effect of zinc on corpuscle and renal tubules (400X).

Fig (14): Photomicrograph of a kidney section of animal treated with aluminium sulphate and supplem-ented with zinc sulphate for 45 successive days showed little effect of zinc on the toxicity of Al on kidney tissue (400X).


1
2
3
4
a
b
5
b
a
c
6
7


8
9
b
a
10
11
12
b
a
13
b
a
14

Discussion

Aluminium is one of the most abundant metal in the earth crust. Al is present in water as many complexes hydroxides, fluorides and sulphates (Goyer, 1996 and Alleva *et al.*, 1998). Exposure to Al has recently been implicated in a number of human pathologies and symptoms of obstructive lung disease were recorded (Sjoren *et al*., 1989), tissuesamples of welders and many workers showed high deposition of Al salts (Mur et al., 1998). According to Massie *et al.*, (1988); accumulation of Al has been found in liver and kidney of mice given Al chloride (McDermott *et al.*, 1979 and Markesbery et al., 1981). Zinc is a potent antioxidant, is known to play a special role in protecting the tissues as liver, kidney, brain and testis against free radicals (Hafiez *et al.*, 1989 & 1990, Mansour *et al.* 1989, Liu & Stemmer (1990a,b), Oteiza *et al.* (1996), Noh & Koo (2001), and Ozkan *et al.* (2004). This study aimed to evaluate that if zinc supplementation after any exposure to Al salts as sulphate can deduce the toxic effect of Al on liver –kidney line of rats.

The results indicated that on liver tissue daily treated of with Al sulphate, showed some swollen cells with vacuoles around the nucleus and some fragmentation of chromatin materials were appeared. Empty spaces within the cytoplasm and dense bodies within the nuclei were appeared. Zinc sulphate supplementation after Al sulphate showed some improvement in liver tissue in which hepatocytes appeared almost and enlarged blood sinusoids. At the end of the experiment liver tissue was distorted with necrotic nuclei and zinc cannot induce clear effect in liver. Kidney tissue showed slight shrinkage in the corpuscles, and renal tubules cells loosed their normal shape due to administration with Al sulphate. Zinc improved beginning of amelioration of kidney tissue, but at the end of experiment (45day) the effect of Al was still affected the renal tubules and corpuscles.

From the results, we can conclude that the effect of Al is back may be to the accumulated Al in the different tissue and kidney can not able to excrete these Al leading to aggregation in he liver tissue, Some authors agree with this opinion as Massie *et al.*, (1988); Weilhelm *et al., (*1996), declared that the lowest level of Al occurring in cytoplasm in the subcellular fractions which leading to accumulation of Al in the liver. Van der Voet *et al.*, (1992) reported that Al accumulation was correlated with the appearance of periportal giant cells in the liver tissue. Bertholf *et al.*, (1989) and Flaten *et al.*, (1996), Mahieu & Calvo, (1998) and McNall &Fosmir, (1996) reported that high concentrations of Al caused necrosis and atrophy recorded in kidney tissue. Reduced renal functions recorded in infants with high Al accumulations in their kidney tissue. Other author backed the effect of Al to the induction of free radicals as demonstrated by (El-Demerdash, 2004 and Yossef, 2004). Zinc administration affects the Al accumulation in brain hippocampus and liver (Liang *et al*., 1999) and may vary the effect of other metals as copper, Al in which Mendez-Alvarez *et al.*, 2002 recorded that zinc alter the toxicological effect of Al and also zinc acts as antioxidant reduce the effect of free radicals produced due to Al toxicity. (Hafiez *et al.*, 1989 & 1990, Mansour *et al.* 1989, Liu & Stemmer 1990(a,b), Oteiza *et al.* 1996; Noh & Koo 2001; and Ozkan *et al.* 2004).

References

1. **Abreo K.; Jangula J.; Jain S.K.; Sella M. and Glass J. (1991):** Aluminium uptake and toxicity in cultured mouse hepatocytes. J. Am. Soc. Nephrol., 1 (12): 1299 – 304.
2. **Alleva, E.; Rankin, J. and Santucci, D. (1998):** Neurobehavioral alterations in rodents following developmental exposure to aluminium. Toxicol. Ind. Health, 14 (1-2): 209 – 221.

3. **Bertholf R.L.; Herman M.M.; Savory J. Carpenter R. M.; and Wills M.R. (1989):** A long-term intravenous model of aluminium maltol in rabbits: tissue distribution, hepatic, renal, and cytoskeletal changes associated with systemic exposure. Toxicol. Appl. Pharmacol., 98 (1): 58 – 74.
4. **Dlugaszek M., Fiejka M.A., Graczyk A. Aleksadrowiez J.C. and Slowikow (2000):** Effects of various aluminium compounds given orally to mice tissue distribution and tissue concentration of essential elements. Pharmacol. Toxicol., 86 (3): 135 – 9.
5. **Domingo J. L. (1995):** Reproductive and Developmental toxicity of aluminium: A review Neuro. Teratol., 17 (4): 515-521.
6. Drury R and Wallington E., (1980): Carlton's histological techniques. Oxford University Press, New York, 5th edition. P. 138, 237 & 241.
7. **Ebina Y.; Okada S.; Hamaki S. and Midorikawa O., (1984):** Liver, kidney, and central nervous system effect due to aluminium given intraperitoneally to rats: a multiple – dose subchronic study using aluminium nitroacetate. Toxicol. Appl. Phaemacol., 75(2): 211 – 8.
8. **El-Demerdash F.M. (2004):** Antioxidant effect of vitamin E and selenium on lipid peroxidation, enzyme activities and biochemical parameters in rats exposed to aluminium. J Trace Elem Med Biol. 18(1):113-21.
9. **Flaten T.P., Alfrey A.G., Birchall J.D., Savory J. and Yokel R. A. (1996):** Status and future concerns of clinical and environmental aluminium toxicology J. Toxicol. Environ. Health, 30 (6): 527-41.
10. **Fortes, C; Agabiti, N.; Fano, V.; Pacifici, R.; Forastiere, F.; Virgili, F. and Zuccaro, P. (1997):** Zinc supplementation and plasma lipid peroxides in an elderly population. Eur. J. Clin. Nutr., 51 (2): 97-101.
11. **Goyer A.A. (1996):** Basic Science of Poisons. In Toxicology Cassaret & Doult 5th Eds. New York. Toronto London.
12. **Hafiez, A, A,, El-Kirdassy, Z.H., El-Malkh, N.M. and El-Zayat, E.M. (1990):** Role of zinc in regulating the testicular function. Part 3. Histopathological changes induced by dietary zinc deficiency in testes of male albino rats. Nahrung. 34(1): 65-73.
13. **Hafiez, A.A., El-Kirdassy, Z.H., Mansour, M.M., Sharada, H.M. and El-Zayat, E.M.. (1989):** Role of zinc in regulating the testicular function. Part 1. Effect of dietary zinc deficiency on serum levels of gonadotropins, prolactin and testosterone in male albino rats. Nahrung. 33(10): 935-40.
14. **Jeffery E. H; Abreo K.; Burgess E. Canata J and Greger J. L. (1996):** Systemic aluminium toxicity effects on bone, hematopoietic tissue, and kidney. J. Toxicol. Environ. Health, 48 (6): 649-65.
15. **Jones D. I. and Kochian L.V. (1997):** Aluminium interaction with plasma membrane lipids and enzymatic metal binding sites and its potential role of aluminium toxicity. FEBS Lett., 400 (1): 1 - 57.
16. **Liang J.Y; Liu Y.Y.; Zou J.; Franklein R. B.; Castello L.c. and Feng P. (1999):** Inhibitory effect of zinc on human prostatic carcinoma cell growth. Prostrate, 40(3): 200 – 207.
17. **Liu, J.Y. and Stemmer, K.L. (1990a):** Interaction of aluminum with zinc and copper and its effects on pituitary-testicular axis: a histological study. Biomed Environ Sci. 3(1):1-10.
18. **Liu, J.Y. and Stemmer, K.L. (1990b):** Interaction between aluminum and zinc or copper and its effects on the pituitary-testicular axis. II. Testicular enzyme and serum gonadotropin assay. Biomed Environ Sci. 3(1): 11-9.
19. **Mahieu S. and Calvo M.L. (1998):** Effect of chronic poisoning with aluminium on the renal handling of phosphate in rat. Toxicol. Lett., 94 (1): 47-56.
20. **Mansour, M.M., Hafiez, A.A., El-Kirdassy, Z.H., El-Malkh, M.N., Halawa, F.A. and El-Zayat, E.M. (1989):** Role of zinc in regulating the testicular function. Part 2. Effect of dietary zinc deficiency on gonadotropins, prolactin and testosterone levels as well as 3 beta-hydroxysteroid dehydrogenase activity in testes of male albino rats. Nahrung. 33(10): 941-7.
21. **Markesbery, W. R.; Ehmann, W. D.; Hossain, T. I. M.; Alauddin, M.; and Goodin, D. T. (1981):** Instrumental neuroactivation analysis of brain aluminium in Alzheimer's disease and aging. Ann. of Neuro. 10: 511-516.
22. **Massie, H. R.; Aiello, V. R. and Tuttle, R. S. (1988):** Aluminium in the organs and diet of aging C57BI16J mice. Mechan. of age. and Develop, 45: 145-156.

23. **McDermott, J. R.; Smith, A. I.; Iqbal, K. and Wisniewski, H. M. (1979):** Brain aluminium in aging and Alzheimer's disease. Neurology, 29: 809-814.
24. **McNall A.D. and Fosmire G.J. (1996):** Zinc status does not affect aluminium deposition in tissues of rats. Biol. Trace. Elem. Rec., 53 (1): 7-18.
25. **Mendez–Alvarez E, Soto–Otero R, Hermida–Ameijeiras A. and Lopez–Real A.M (2002):** Effects of aluminium and zinc on the oxidative stress caused by 6-hydroxydopamine autoxidation: relevance for the pathogenesis of Parkinson disease. Biochem. Biophys. Acta, 1586 (2): 155 – 68.
26. **Muller G.; Bernuzzi, V.; Desor D; Hutin M.F.; Burnel D; and Lehr P.R. (1990):** Developmental alteration in off springs of female rats orally intoxicated by aluminium acetate at different gestation periods. Teratology, 42 (2): 253-263.
27. **Mur J.M.; Wild P.; Rapp R.; Vautrin J. P. and Coulon J.P. (1998):** Demographic evaluation of the fertility of aluminium industry workers: influence of exposure to heat and static magnetic field. Hum. Reprod., 3 (7): 2016-2019.
28. **Noh, S.K. and Koo, S.I. (2001):** Feeding of a low-zinc diet lowers the tissue concentrations of alpha-tocopherol in adult rats. Biol Trace Elem Res. 81(2): 153-68.
29. **Oteiza, P. L.; Olin, K. L.; Frage, C. G. and Keen, C. L. (1996):** Oxidant defense systems in testes from zinc deficient rates. Proc. Soc. Exp. Biol. Med., 213: 85-91.
30. **Ozkan, K.U., Boran, C., Kilinc, M., Garipardic, M. and Kurutas, E.B. (2004):** The effect of zinc aspartate pretreatment on ischemia-reperfusion injury and early changes of blood and tissue antioxidant enzyme activities after unilateral testicular torsion-detorsion. J Pediatr Surg. 39(1): 91-5.
31. **Sjoren, B.; Elinder, V. G. and Lidums, V. (1988):** Uptake and urinary excretion of aluminium among welders. Intern. Arch. of Occup. and Environ. Health, 60: 77- 79.
32. **Spencer A. J.; Wood J.A; Saunder H.C.; Freeman M.S. and lote C.J. (1995):** Aluminium deposition in liver and kidney following acute intravenous administration of aluminum chloride or citrate in conscious rats. Hum. Exp. Toxicol., 14 (10): 787-794.
33. **Van der Voet G.B., Brandsma A.E., Heijink E. and Wolff F.A. (1992):** Accumulation of aluminium in rat liver: association with constituents of the cytosol. Pharmacol. Toxicol. 70 (3): 173-6.
34. **Wennberg A. (1998):** Effects on the nervous system among welders exposed to aluminum and manganese. Occup. Environ. Med., 53 (1): 32-40.
35. **Wilhelm M.; Jaeger D.E.; Schull; Cablitz, H.D.; Hofner D. and Idel. H. (1996):** Hepatic clearance and retention of aluminium: studies in the isolated perfuse rat liver. Toxicol. Lett., 31(3): 257-63.
36. **Yousef M.I. (2004):** Aluminium-induced changes in hemato-biochemical parameters, lipid peroxidation and enzyme activities of rabbits: protective role of ascorbic acid. Toxicology, 199 (1): 47-57.

دراسات هستولوجية عن تأثير كبريتات الزنك على سمية كبريتات الألومونيوم في كبد وكلى الجرذ الأمهق

عزة محمود جاويش
قسم علم الحيوان - كلية العلوم - جامعة القاهرة

يعتبر الألمونيوم من العناصر الهامة في العصر الحديث والأكثر شيوعا واستخداما في أواني الطهي وفى حياتنا اليومية وقد لوحظ آن له تأثيرا ضارا على كلى وكبد الإنسان. وقد تناولت هذه الدراسة إيضاح اثر الألمونيوم السمي ودراسة الدور الوقائي لمادة الزنك ضد هذا الأثر على نسيج الكلى والكبد وفحصها باستخدام قطاعات شمعية مصبوغة بالهيماتوكسيلين و الايوسين. وقد تم إجراء هذا البحث على عدد 70 من ذكور الجرذان المهق التي يتراوح وزنها بين 160- 170 جم وتم تقسيمها إلى ثلاث مجموعات :
المجموعة الأولى تعتبر مجموعة ضابطة والمجموعة الثانية تعاطت جرعات من كبريتات الألمونيوم (50مج/كجم/يوم) عن طريق الفم باستخدام أنبوبة معدية لمدة45 يوما متتالية. المجموعة الثالثة مدت بجرعات من الزنك (كبريتات الزنك 50مج/كجم/يوم) بعد تعاطيها نفس الجرعات المذكورة من كبريتات الألمونيوم كما في المجموعة الثانية لمدة 45 يوما أيضا.
وقد أظهرت النتائج آن للألومنيوم تأثيرات واضحة على الكبد حيث انه احدث تغيرا في الخلايا الكبدية حيث إنها فقدت الشكل المميز لها مع ظهور انويه ميتة داخلها خلال فترات التجربة مم يدل على موت الخلايا وانويتها. كما أوضحت أن كمية من النسيج الضام الليفي تركزت حول الخلايا الكبدية والوريد الدموي المركزي في الكبد. كما تغير شكل الخلايا والوحدات البولية حيث أنها ضاقت وانكمشت وصغر حجمها حتى نهاية التجربة. كما أوضحت الدراسة أن إمداد الحيوانات بالزنك أدى إلى تحسن ملحوظ في نسيج كل من الكبد والكلى امتد حتى 30 يوما من وقت التجربة ثم لم يحدث تحسن ملحوظ بعد ذلك وحتى نهاية المدة. ويتبين من نتائج هذه الدراسة أن المعالجة بالزنك تلعب دورا ولكنه محدودا في الوقاية من التأثيرات السمية الناتجة عن استعمال الألمونيوم.