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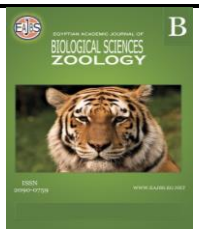

Effects of Copper Sulphate Supplementation on Some Physiological Parameters in Male Albino Rats

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ABSTRACT

Copper is an essential trace element required for different metabolic functions. The aim was to investigate the toxic effects induced by the over-exposure to copper sulphate. Materials and Methods: Rats were divided into three groups, as follows: the first group (GI) served as untreated control. The second group (GII) and the third group (GIII) were orally treated with a daily dose of copper sulphate 100 mg/kg b.wt. And 200 mg/kg b.wt., respectively for two weeks. Results: At the end of the experiment, blood samples were collected and the sera were separated for estimation the levels of total testosterone, follicule stimulating hormone (FSH), leutilizing hormone (LH) and prolactin hormone, alanine amino transferase (ALT), aspartate amino transferase (AST) and alkaline phosphatase (ALP) enzymes; urea, uric acid and creatinine. Results showed a marked reduction in the total testosterone, FSH, LH, and prolactin levels in all treated groups compared to the corresponding controls. Whereas, serum activities ALT, AST, and ALP enzymes and the urea, uric acid and creatinine levels were obviously elevated in all treated groups comparing with control group. Conclusion: The present results revealed that copper sulphate caused disruption in the sexual hormones and all biochemical parameters measured in the current study.

INTRODUCTION

Copper is an essential element for normal functioning of the organisms (Krupanidhi *et al*., 2008). It is required for normal growth, development, as a complementary of cytochrome system (Kodama and Fujisawa, 2009), and it used as food additives. Also, it is one of the key trace minerals required for an effective immune response. The functions of copper ensure electron-transfer catalysis through its two attainable oxidation states (Georgopoulos *et al*., 2001). Copper salts are used as fungicides, pesticides, and algicides (Mladenović *et al*., 2014). The exposure to copper might be through dermal contact with air, water, and soil that contains copper, inhalation, and consumption of water and food (Lee *et al*., 2016). Copper absorbed in the stomach and the upper intestine then reaches the liver in the form of a complex

with serum proteins, albumin or the amino acid histidine (Derouiche *et al*., 2013). The copper should be maintained in a complex form in the liver is to prevent the oxidative damage caused by free copper to DNA, proteins and the membrane components (Krupanidhi *et al*., 2008). When the copper intake exceeds the normal range of the biological tolerance, it may cause multiple side effects in humans and the laboratory animals, including the damage of the liver, kidney, immune system and gastrointestinal tract (ATSDR, 2004) and reduce the fertility (Pesch *et al*., 2006). In addition, the excess of copper affects the cardiovascular system, promoting atherosclerosis and causing high blood pressure (Iskra and Majewski, 2000). Also, acute consumption of copper in water may cause stomach irritation, nausea, dehydration, and loss of appetite (Araya *et al*., 2004). Copper enters in the normal component of semen, it bound to the midpiece of spermatozoa and it present also in the seminal fluid (Valsa *et al*., 1994). The high doses of copper ions have adverse side effects on the epididymis, testes, and scrotum of mammals, leading to reduction of fertility (Pesch *et al*., 2006). Copper is one of the redox-active metals that able to catalyze the oxidation process, leading to reactive oxygen species (ROS) production, that causes peroxidation of polyunsaturated fatty acids in the lipids of the membranes, leading to the damage of biomolecules (Halliwell and Gutteridge, 2007).The complication of copper sulphate poisoning is haemolytic anaemia that may cause either directly by the damage of RBCs membranes (Chuttani *et al*., 1965) or indirectly by the inactivation of enzymes that prevent oxidative stress (Mital *et al*., 1966).

In light of these facts, the main goal of the present investigation was to evaluate the toxic effects of copper sulphate on some hormones and some biochemical parameters in male rats.

MATERIALS AND METHODS

Chemicals:

Copper in the form of copper sulphate pentahydrate (Cu SO4.5H2O) crystals from El Gomhoria company, were used in this experiment.

Animals:

Eighteen male albino rats (*Rattus norvegicus*) weighing 130-140 g were used in the present study. The adult rats were purchased from the farm of National Organization For Drug Control and Research, Giza, Egypt. The adult rats were preserved under normal environmental situations with a natural light/dark cycle and free entrance to slandered diet and water *ad-libitium*.

Study Protocol:

After one week of acclimatization, the rats were divided into three groups randomly (6 animals for each), as follows: Group I (control): Animals without any treatment. Group II: Animals were received orally copper sulphate daily at low dose (100 mg/kg b.wt.) for two weeks. Group III: Animals were received orally copper sulphate at high dose (200 mg/kg b.wt.) for two weeks. After the last dose of treatment, rats were fasted overnight (twenty-four hours) and then the rats in each group were anesthetized with diethyl ether. In the end, rats were sacrificed by cervical dislocation and blood samples were collected, then the sera were obtained by blood centrifugation at 4000 rpm for 20 min at 4 C and immediately stored at -20 C till time of analysis.

Hormonal Assay:

The sera levels of total testosterone, follicule stimulating hormone (FSH), luitilizing hormone (LH) and prolactin were measured by Enzyme-linked Immune

Sorbent Assay (ELISA) according to Bricaire *et al*., (1991), Urban *et al*. (1988), Levine *et al*. (1985) and Liu and Zhous (1994), respectively.

Estimation of Some Biochemical Parameters:

Urea and uric acid were estimated according to Young (2001). Creatinine was determined according to the method described by Bartels and Bohmer method (1972). The activities of serum AST and ALT were measured by the method of Reitman and Frankle (1957). Levels of serum ALP were assayed according to Englehardt (1970).

Statistical Analysis:

Data were analyzed for obtaining mean, standard error (SE) and statistical comparisons between means of different groups. The statistical analyses were done by one way ANOVA and DUNCAN test using SPSS program version 16 software. Values were considered statistically significant when P value < 0.05.

RESULTS

Table (1), shows the effect of copper sulphate supplementation on the mean concentrations of total testosterone, FSH, LH and prolactin hormones in low dose (100 mg/kg b.wt.) and high dose (200 mg/kg b.wt.). The data revealed that the treatment of rats with copper sulphate induced a significant decrease in the total testosterone, FSH, LH and prolactin hormones with low and high doses of treatment compared to the corresponding control group.

Serum urea, uric acid, and creatinine of control and treated groups are shown in table (2). The administration of copper sulphate to rats with the low and high dose exhibited a significant elevation in the urea, uric acid, and creatinine serum levels. The elevation in these parameters became more obvious by increasing the dose of copper sulphate comparing with control group.

Table (3), showed that the treatment with a low and high dose of copper sulphate revealed a significant increase in the activities of ALP, AST and ALT enzymes comparing with the control group. The recorded elevation in all enzymes activities revealed dose-dependent manner.

Table 1: Effects of low and high doses of copper sulphate on the sera levels of total testosterone, FSH, LH and prolactin hormones.

Parameters	Group I (water)	Group II (100mg/kg)	%	Group III (200mg/kg)	%	p-value
Total Testosterone (ng/ml)	**11^{a}±0.14**	**2.61^{b}±0.22**	**-76.3**	**1.4^{c}±0.31**	**-87.3**	*
FSH (mIU/ml)	**2.167^{a}±0.1**	**0.14^{b}±0.013**	**-93.54**	**0.21^{b}±0.27**	**-90.31**	*
LH (mIU/ml)	**10.72^{a}±0.2**	**0.3^{b}±0.021**	**-97.2**	**0.2^{b}±0.03**	**-98.13**	*
Prolactin (ng/ml)	**4.25^{a}±0.111**	**2.95^{b}±0.076**	**-30.6**	**2.78^{b}±0.15**	**-34.6**	*

*Values represent mean ± S.E. with the number of animals between parentheses

*% D: Percentage difference [(Treated value – Control Value) / Control Value] x 100

Different letters indicate significantly different means p-value < 0.05

* The same letters indicate non-significant changes.

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Table 2: Effects of low and high doses of copper sulphate on serum levels of urea, uric acid, and creatinine.

Parameters	Group I (water)	Group II (100mg/kg)	%	Group III (200mg/kg)	%	p-value
Urea (mg/dl)	$31.2^{a}\pm1.1$	$42.5^{b}\pm0.6$	36.21	$48.3^{c}\pm1.1$	54.81	*
Uric acid (mg/dl)	$1.8^{a}\pm0.1$	$2.3^{b}\pm0.12$	27.8	$3.23^{c}\pm0.1$	79.44	*
Creatinine (mg/dl)	$0.35^{a}\pm0.011$	$0.41^{b}\pm0.01$	17.14	$0.44^{c}\pm0.011$	25.71	*

*Values represent mean ± S.E. with the number of animals between parentheses
*% D: Percentage difference [(Treated value – Control Value) / Control Value] x 100
Different letters indicate significantly different means p-value < 0.05
* The same letters indicate non-significant changes.

Table 3: Effects of low and high doses of copper sulphate on serum activities of ALP, AST and ALT enzymes.

Parameters	Group I (water)	Group II (100mg/kg)	%	Group III (200mg/kg)	%	p-value
ALP (U/L)	158±1.93	$235.3^{b}\pm1.45$	42.94	$266^{c}\pm2.06$	64.7	*
AST (U/L)	$5^{a}\pm0.3$	$6.42^{b}\pm0.42$	28.4	$8.7^{c}\pm0.44$	74	*
ALT (U/L)	$7.42^{a}\pm0.24$	$23^{b}\pm0.73$	209.97	$25.3^{c}\pm0.6$	240.97	*

*Values represent mean ± S.E. with the number of animals between parentheses
*% D: Percentage difference [(Treated value – Control Value) / Control Value] x 100
Different letters indicate significantly different means p-value < 0.05
* The same letters indicate non-significant changes.

DISCUSSION

Copper is a necessary element for some of the biological processes, whereas prolonged exposure to high concentrations may cause toxic effects (Fuentealba and Aburto, 2003). The present work was carried out to investigate the effects of copper sulphate on sexual hormones and some biochemical parameters of rats. Noteworthy, the current study reported that rats treated with copper sulphate showed that there is a highly significant reduction in the total testosterone, FSH, LH and prolactin hormone levels as compared with control levels. The recorded changes in the measured hormones became more obvious by increasing the concentration of copper sulphate.

Normally, testosterone is controlled by a negative feedback mechanism, the decrease of testosterone hormone causes an increase in the level of LH and FSH hormones. The hypothalamic secretes gonadotropin-discharging hormone (GnRH) that follows up on the front pituitary to deliver FSH and LH (Dandona and Rosenberg, 2010). The decreasing in the level of testosterone may be attributed to either the effect on steroidogenesis enzymes in testes or its inactivation adrenergic systems involved in steroidogenesis. In the present study copper sulphate cause decrease LH may because impact to the interstitial Leydig cells of the testicles, also decrease FSH may be

through spermatogenesis, so copper sulphate effect on Sertoli cell function. This may be attributed to the anti-androgenic action and the stimulation of steroidal anti-androgen to the negative feedback inhibition of the hypothalamus that resulted in lowering the concentration of testosterone Mocktary *et al*. (2007). The present findings are in agreement with Roychoudhury *et al*. (2008) and Sakhaee *et al*. (2012), who recorded that fertility is adversely affected by copper, specifically a decline in male reproductive capacity. Several mechanisms have been proposed to explain copper-induced cellular toxicity. Copper exists either oxidized, cupric (Cu2+), or reduced, cuprous (Cu+), state. In the living cells, copper represents as catalyst in the production of superoxide radicals, hydroxyl radicals and hydrogen peroxide by the Haber-Weiss reaction (Bremner 1998 and Kadiiska *et al*.,1993), that cause oxidative damage and induce adverse side effects (Gaetke and Chow, 2003). The explanation of the disturbance in the sera levels of total testosterone, FSH, LH and prolactin hormones is that high levels of copper sulphate may be caused by a reduction in both Gonadotropin-releasing hormone (GnRH) secretion and pituitary responsiveness to GnRH. Therefore, this reduction in serum testosterone levels may be resulted from disruption of the hypothalamic-pituitary-tests regulatory axis that controls testosterone production by testicular leydig cells. Moreover, this result may be due to the significant effect of copper in treated male rats that attributed to the direct cytotoxic effect of copper on the tissue of testis of exposed animal to copper (CCOHS, 1999). Also may be due to copper has damaging effect of spermatogonial cells specially late stage of sperm maturation (Dent, 2007).

Regarding the kidney function, our results demonstrated that the oral intake of copper sulphate exhibited an increase in levels of urea, uric acid and creatinine comparing to the control group, indicating impaired renal function in copper sulphate treated groups. The progression of kidney damage is marked by the increase in two important chemical substances in the blood - creatinine, and urea whose evaluation in the serum helps to assess Glomerular Filtration Rate (GFR) followed by renal function **(**Nisha *et al*., 2017**).** These impairments could be due to the threshold of tubular reabsorption changes, renal blood flow and the rate of glomerular filtration (Zurovsky and Haber, 1995). Plasma uric acid and creatinine can be used as a rough index of the glomerular filtration rate (Hernandz and Coulson, 1967). High levels of uric acid and creatinine indicate several disturbances in the kidney (Maxine and Benjamin, 1985). The present results are consistent with the study of Sinkovic *et al*. (2008) and Akomolafe *et al*. (2014), who reported that copper exposure is associated with renal dysfunction. Also, these findings coincide with the results obtained by (Lei *et al*., 2008), who recorded that copper caused kidney damages with biochemical alterations, including increased blood urea nitrogen and creatinine. Furthermore, the uric acid related to the development of chronic kidney disease and maybe a poor factor for the progression of acute renal failure (Giordano *et al*., 2015), so the elevation of uric acid level in the serum revealed that the treatment with copper sulphate interferes with kidney function. These effects may be attributed to copper is a catalyst that leading to oxidative stress by the formation of reactive oxygen species (ROS), that cause damage for lipid peroxidation (Abuja and Albertini, 2001) and the main target organs affected by copper sulphate are liver and kidney causing hepatotoxicity and renal failure (Hassan *et al*., 2010 and Galhardi *et al*., 2004).

It is known that the activities of serum aminotransferases are considered as markers of toxicity in the liver induced by chemicals (Govindwar and Dalvi, 1990). The most sensitive biomarkers used in the diagnosis of liver diseases are AST and ALT activities (Pari and Kumar, 2002) and their levels indicate the type and extent of

damage inflicted that reflects a state of hepatocyte injury (Pari and Murugan, 2004). The present study recorded that rats consumed copper sulphate revealed a significant increase in the activities of ALT and AST enzymes in groups treated with the low and high dose of copper sulphate when compared to control rats, that used to discover of tissue damage. The increase of these enzymes clearly demonstrated that the treatment with copper sulphate disrupts the liver function, more intensely at the higher doses. The results of this study are in line with the observation of Mladenović *et al*. (2014), who reported that copper treatment caused the elevation of AST, ALT and LDH activities in the serum. Also, it was reported previously that, Cu NPs caused biochemical alterations, such as the increasing of alanine aminotransferase (ALT) and aspartate aminotransferase (AST) (Lei *et al*., 2008).

This may be attributed to the excessive hepatocelluar damage caused by the toxic effect of copper sulphate, thus caused the release of greater levels of intracellular enzymes into the blood. This damage exhibited a dose-dependent manner. Also, the elevation of serum aminotransferases activity may be due to the failure of parenchymal cells of the liver under pathological conditions to ensure the vital functions, causing distribution of metabolism. As, It was shown that copper sulphate can induce hepatic toxicity (El-Masry, 2012). Also, the present investigation revealed an increase in ALP activity in rats treated with different doses of copper sulphate. This elevation may be an indicator of obstructive damage in the liver caused by copper sulphate administration.

In conclusion, our study has demonstrated that oral administration of copper sulphate resulted in a vehement disturbance in sexual hormones including testosterone, FSH, LH, and prolactin, also it caused disturbance in all studied biochemical parameters including the activities of AST, ALT and ALP enzymes and uric acid, urea and creatinine levels. So, due to the hazardous effect of copper sulphate, it is recommended to limit and be careful when using it in food as food additives.

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ARABIC SUMMARY

تأثير المعالجة بكبريتات النحاس على بعض المعايير الفسيولوجية في ذكور الجرذان البيضاء

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يعد النحاس عنصرًا أساسيًا للتمثيل الغذائي. ويهدف هذا العمل لدراسة الآثار السامة الناتجه عن الإفراط للتعرض لكبريتات النحاس. المواد والطرق: تم تقسيم الجرذان إلى ثلاث مجموعات، على النحو التالي: المجموعة الأولى وتمثل المجموعة الظابطه ولم يتم معالجتها بشيء. وتم معالجة المجموعة الثانية والمجموعة الثالثة عن طريق الفم بجرعة يومية من كبريتات النحاس 100 مجم / كجم من وزن الجسم و 200 مجم / كجم من وزن الجسم على التوالي لمدة أسبوعين. النتائج: في نهاية التجربة، تم جمع عينات الدم وفصل الأمصال لتقدير مستوي هرمون التستوستيرون الكلي ، هرمون المحفز لحويصلات المبيضين (FSH)، هرمون اللوتيني (LH)، هرمون البرولاكتين ، ألانين أمينو ترانسفاسيز (ALT)، الأسبارتات أمينو ترانسفيز (AST) و الفوسفاتيز القلوية (ALP) ،اليوريا وحمض اليوريك والكرياتينين. وأظهرت النتائج انخفاض ملحوظ في مستوي هرمون التستوستيرون الكلي ،FSH، LH والبرولاكتين في المجموعات المعالجة بكبريتات النحاس مقارنة مع المجموعة الظابطة. بينما كان هناك زيادة ذو دلالة احصائية في نشاط ALT و AST و ALP و مستوي اليوريا وحمض اليوريك والكرياتينين في المجموعات المعالجة بكبريتات النحاس مقارنة مع المجموعة الظابطة. الاستنتاج: كشفت النتائج الحالية أن كبريتات النحاس تسببت في خلل للهرمونات الجنسية وجميع المعايير البيوالكيميائية المقاسة في هذه الدراسة.