



Research Article

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## ***Health Status of Workers Exposed to Copper Pollution Stress***

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### **ABSTRACT**

*The aim of the present study is to assess the health status of subjects working in a copper factory from a semi-arid region in Algeria. Workmen exposed to copper dust (working period of  $21.4 \pm 5.3$  years) were compared to the control group of administrative staff (working period of  $24 \pm 3.33$  years). However, serum aminotransferases (ASAT and ALAT), cholesterol, triglycerides, glucose, bilirubin, granulocytes, monocytes and lymphocytes levels were estimated. The obtained results have shown a remarkable decrease in glucose and triglycerides levels of the exposed individuals compared to the control. Conversely, cholesterol concentration was significantly increased in the exposed subjects. There were no significant variations concerning the level of ALAT, ASAT, bilirubin, granulocytes, monocytes and lymphocytes between the exposed workers and the administrative staff. In conclusion, within hot polluted environment, accompanied with moderately acceptable working conditions, the levels of energy-supplying molecules were remarkably depleted, which might be related to the activation of the copper-translocating ATPases within cells.*

**Keywords:** Copper, pollution, health status, hot environment, workers

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### **INTRODUCTION**

Copper is an essential trace element for different metabolic processes and enzyme activities. It is a micro-nutrient, essential for normal growth of lower organisms and higher mammals. It plays a critical role in cellular energy production [1]. The human recommended dietary allowance of copper based on age, sex and physiological state is well established [2]. However, copper excess from human activities, industrial and agricultural processes is likely to provoke toxic effects as in the case of occupational exposure in construction materials wires, tubes and electrical components [3]. Liver is the central organ for Cu homeostasis, in which it mobilized into the peripheral circulation or secreted into the bile for excretion [4]. Chronic copper toxicity is characterized by a progressive hepatic accumulation of copper that results eventually in liver damage [5]. Thus, copper is a potential oxidative stressor, where it makes inappropriate binding to macromolecules due to uncontrolled accumulation [4]. As a result, unbound copper may catalyze the formation of highly reactive hydroxyl radicals and interfere with important cellular functions. Thus, copper toxicity is affected to some extent by many factors such as the species, age, genetic, drugs, diet and duration of exposure [6].

Until now, few studies have investigated the toxic effect of trace metals in the workplace from Algeria [7; 8]. Therefore, the objectives of the present study are to assess some biochemical parameters of workers in copper-made wire factory by estimating the aminotransferases, bilirubin, triglycerides, cholesterol and glucose levels. However, our assumption is that occupational exposure to copper in this factory may lead to copper toxicity, especially at chronic long-term exposure.

## MATERIALS AND METHODS

The present study investigates the health status of the male workers from a wire factory (B07/11), which produces different types of electrical copper-based wires and tubes. Though, workers are chronically exposed to copper dust and high ambient temperature in a factory situated in a hot semi-arid region. The study was carried out in cooperation with the factory administration and occupational medicine centre. The participating subjects were informed about the nature of this investigation. Exposed and unexposed subjects were chosen to be similar in gender, age, smoking habit, residence, and socio-economic level. Most workers were recruited together during the opening of this factory and they eat a free lunch ameliorated meal supplied by the factory. Moreover, workers known to suffer from chronic diseases as diabetes, kidney failure, and copper genetic disorders were excluded from the study. Thirteen subjects were chosen from the wire unit (center of mixing), and from the unexposed administrative personnel (n=10) used as a control. The exposure period of all workers was over than 19 years. Blood was collected at the medical center of the factory by the medical staff in the morning shift. Dry test tubes were then centrifuged at 4000 rpm/min during 10 minutes. Triglycerides, cholesterol, aspartate aminotransferase (ASAT), alanine aminotransferase (ALAT), glucose and bilirubin have been measured by using commercial SPINREACT Kits (Spain), whereas granulocytes, monocytes and lymphocytes counts were obtained by "Full Automatic Blood Cell Counter, Model PCE-2 ON, ERMA INC, Tokyo". Statistical analysis was used by applying Student t-test to compare the exposed group with the control (using Minitab version 15). The significant difference at  $p < 0.05$  was considered.

## RESULTS AND DISCUSSION

Results are summarized in table 1. The ages of workmen of both groups were almost similar. In addition, the exposure period of the exposed workers was not significantly different when compared to the exposure period of the control. Concerning the enzymes, ASAT and ALAT activities were closer in the two groups. The exposure of workers to copper chronic inhalation from copper fumes has produced only small augmentation of ASAT and ALAT activities. Such results show that the degree of toxicity on the workers of wire production unit is not higher to produce a noticeable effect on the liver function indicated by these very sensitive enzymes. Accordingly, when a group of people were administered 0.14 mg Cu/kg/day during 12 consecutive weeks, no significant alterations in serum markers of liver damage, cholesterol, ASAT and ALAT was found [9]. Similarly, no variations in serum ALAT and ASAT activities were observed in infants exposed to 0.315 mg Cu/kg/day for 9 months [10], or rats exposed to 60 mg Cu/kg body weight for one month [11]. Contrary, sheep copper toxicosis has demonstrated elevated concentrations of serum liver-specific enzymes [12]. Moreover, it was reported an increased urinary ASAT activities in male and female rats exposed to 140 or 134 mg Cu/kg/day, respectively [13]. Hepatic serum enzymes were higher in rat exposed to 4 mg of copper for 30 days [14].

**Table 1.** The levels of biological markers ( $\bar{X} \pm \text{SD}$ ) in workmen from the copper wire factory

Markers	Control	Exposed
Age (y)	53.1 $\pm$ 4.3 <sup>a</sup>	49.6 $\pm$ 3.2 <sup>a</sup>
Working period (y)	24 $\pm$ 3.33 <sup>a</sup>	21.4 $\pm$ 5.3 <sup>a</sup>
ASAT (IU/l)	20.8 $\pm$ 8.91 <sup>a</sup>	28.25 $\pm$ 5.24 <sup>a</sup>
ALAT (IU/l)	21.28 $\pm$ 7.4 <sup>a</sup>	27.82 $\pm$ 5.7 <sup>a</sup>
Bilirubin (mg/l)	7.0 $\pm$ 2.16 <sup>a</sup>	09.52 $\pm$ 2.2 <sup>a</sup>
Triglycerides (g/l)	1.55 $\pm$ 0.31 <sup>a</sup>	0.77 $\pm$ 0.13 <sup>b</sup>
Total cholesterol (g/l)	0.96 $\pm$ 0.14 <sup>a</sup>	1.43 $\pm$ 0.26 <sup>b</sup>
Glucose (g/l)	1.06 $\pm$ 0.24 <sup>a</sup>	0.73 $\pm$ 0.13 <sup>b</sup>
Granulocytes ( $\mu\text{l}/10^3$ )	3.43 $\pm$ 0.72 <sup>a</sup>	4.68 $\pm$ 0.5 <sup>a</sup>

Monocytes ( $\mu\text{l}/10^3$ )	$0.46 \pm 0.24^a$	$0.33 \pm 0.1^a$
Lymphocytes ( $\mu\text{l}/10^3$ )	$2.45 \pm 0.63^a$	$2.8 \pm 0.44^a$

**a, b:** Horizontal lines sharing the same superscript letters are not significantly different.

Despite the observed augmentation of bilirubin in the exposed workers, no significant difference was found when compared to the control group. At the same manner, children serum bilirubin and copper were also unaffected by chronic Cu intoxication [10]. In Sweden, the highest concentrations of copper were found in liver, followed by brain, kidney and lung among dead workers at copper smelters [15]. Likewise, liver alteration was noticed by increased serum bilirubin level and aminotransferase activities of sublethal copper-supplemented rat during one month [16].

The concentration of triglycerides was significantly reduced in the exposed workmen compared to the control of the administrative staff, which confirms that the long working period has a great effect on workers, probably by copper accumulation in cells leading to biochemical alteration. It is known that serum triglyceride levels decrease when body consumes more energy. In addition, copper overload was found to increase lipid peroxidation [5; 14] since it acts as direct cellular toxicants resulting in cell injury and hepatocellular necrosis [17]. On the other hand, cholesterol concentration did not change in humans supplemented with copper for a period of three months [9].

The result of serum glucose is in line with those of triglycerides, in which probably an enormous oxidation of glucose has been carried out in order to produce more energy for the copper-stressed cells. Accordingly, the mechanism which could explain these findings might be resulted from increased exportation (taking out) of Cu from cells, to limit the intracellular toxicity of metal ions, by using high energy consuming pumps (copper-translocating ATPases) within cells [18]. Moreover, bean stressed by copper chloride had decreased glucose liberation from starch due to  $\alpha$ -amylase inhibition during seeds' germination [19]. At this circumstance, the expected cellular copper excess is supposed to originate from pollution within the factory. It was reported that homeostatic regulation of copper after its long-term high supplementation was not sufficient to prevent its retention in men [20].

Contrary, cholesterol level in this factory was found to be higher in the copper exposed subjects than those of the control. Such result is in line with those of [11], who reported higher concentration of cholesterol in wistar rats exposed to 60 mg Cu/kg body weight by gastric tube for one month. Similarly, daily copper oral sub lethal administration has increased cholesterol concentration in albino rats [16]. Furthermore, daily copper supplementation was seen to increase serum total cholesterol in adult men after six weeks [21]. In contrast, long-term copper supplementation to adult men has not affected serum cholesterol [9]. Hence, epidemiological studies have suggested a positive relationship between coronary heart disease and serum copper levels [22].

In this factory, the level of granulocytes, monocytes and lymphocytes seem unaffected in subjects who worked more than 19 years. Contradicting results were reported concerning white blood cells, in which they decreased significantly (monocytes and basophiles), but they increased considerably (neutrophils and eosinophils) when rabbits have had copper overfeeding [23]. Lymphocytes were reduced in fish exposed to copper [24], while in children lymphocytes were affected after long period of high copper supplementation [25]. In rats, different types of blood leucocytes have increased in response to inhaled copper oxide nanoparticles [26]. Though, pulmonary copper deposition, fibrosis, and granulomas of the lung were reported in workers after years of exposure to copper spray [27]. Finally, copper toxicity always depends on its hepatic distribution and its association with specific binding proteins, which are related to many factors such as species, age, diet, dose and duration of exposure [6]. Therefore, the results of this investigation reflect to some extent the specific factors of a semi-arid region.

## CONCLUSIONS

Long-term exposure to copper dusts within hot environment has reduced the concentrations of the energy-supplying molecules (triglycerides and glucose), although it raised the level of total cholesterol. However, the level of ASAT, ALAT, bilirubin, granulocytes, monocytes and lymphocytes were within the normal ranges. Finally, the possibility of workers' tolerance to copper dust during the long-working period is not excluded.

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