Effect of Cadmium Exposure among workers in Battery Industries

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ABSTRACT

The study aimed to assess the possible influence of long-term human occupational exposure to cadmium and smoking cigarettes at the time of exposure on renal and liver functions in battery manufacturing. ***Methods:*** *This cross-sectional study evaluated liver, kidney function, oxidative stress and lipid per oxidation among smoker and non smoker workers in battery manufacturing in Egypt. Multiple linear regression was conducted to investigate the association between cadmium exposure period and the serum levels of aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), gamma-glutamyl transferase (GGT), albumin, creatinine, uric acid (UA), urea, cu/zn superoxide dismutase (cu/zn SOD), glutathione reductase (GSH) in RBCs, malondialdehyde (MDA), hemoglobin and β2-microglobulin in urine, adjusting for smoking and the amount of cigarette smoked. Subjects were stratified into direct and indirect exposure according to their place of exposure.* ***Results****: our results show positive associations between exposure to cadmium and adverse effect on Hb, GSH, UA and GGT.*

Keywords: *Cadmium, Superoxide dismutase, Liver, Kidney, Toxicity, Environmental contaminant, Smoking.*

INTRODUCTION

Cadmium is a highly toxic metal with a very long half-life of 20-30 years in humans and accumulates in soft tissues, kidneys, and the liver. Specific mechanisms of cadmium toxicity are not well understood, however evidence suggests that cadmium affects DNA repair, and cell signaling and control. These effects lead to kidney damage, cancer, mutations, damage to hormone regulating mechanisms, reproductive disorders, and problems with cellular differentiation **(Rani et al., 2014).** Cadmium (Cd) is an industrial and environmental pollutant. Worker exposure to cadmium can occur in all industry sectors but mostly in manufacturing and construction. Workers may be exposed during melting and refining of metals, and manufacturing batteries, plastics, coatings, and solar panels **(Weidenhamer et al., 2011)**.

Tobacco is an important source of cadmium exposure for smokers, who have about twice as much cadmium in their bodies as nonsmokers. For nonsmokers, food is the major route of exposure to cadmium among the non-occupational population **(Barański et al., 2014)**. In the environment, Cd is dangerous because humans consume both plants and animals that absorb Cd efficiently and concentrate it within their tissues **(Stohs and Bagchi 1995)**. Cd shows different mechanisms of toxicity under different experimental conditions and in various species **(Waisberg et al., 2003)**. Cd has been demonstrated to stimulate free radical production, resulting in oxidative deterioration of lipids, proteins and DNA, and initiating various pathological conditions in humans and animals **(Waisberg et al., 2003)**. Once absorbed, Cd is rapidly cleared from the blood and concentrates in various tissues.

Chronic exposure to inorganic Cd results in accumulation of the metal mainly in the liver and kidneys, as well as in other tissues and organs causing many metabolic and histological changes, membrane damage, altered gene

expression and apoptosis **(Shaikh et al. 1999, Casalino et al. 2002, Waisberg et al., 2003)**. Both recent and long-standing studies have shown that exposure to cadmium can lead to cancers, bone problems, and neuro developmental disorders **(Barański et al., 2014)**.

Aim of the study: The study aimed to assess the possible influence of long-term human occupational exposure to cadmium and smoking cigarettes at the time of exposure on renal and liver functions in battery manufacturing.

SUBJECTS & METHODS

Materials

Chemicals and kits were purchased from Merk-schuchardt chemical company (Hohenbrunn. Germany). Commercial kits were used for assessment of biochemical parameters. All other chemicals were of analytical grade.

Subjects:

The current study was conducted on 350 workers (participants) aged 22-60 years. Workers were classified into two groups, low exposure group at office, laboratory and supervisory personnel (n=98) (indirect exposure group); and a high exposure group as production workers (direct exposure group) with long histories of work in areas with substantial airborne cadmium (n= 252). These two groups were divided into subgroups according to duration of exposure and who are smoker and those who had never smoked. This study was conducted in (Chloride and Energizer Cos. represent batteries sector), Cairo and Alexandria, Egypt.

Experimental Design:

Study protocols were reviewed and approved by the ethic committee of National Nutrition Institute in Egypt and consents were obtained from all participants. All participants were interrogated using questionnaire in order to have detailed information about the age, medical history of (liver, renal diseases and diabetes), work period (<5 years, represent

"298 cases and >5 years, represent 52 cases) and smoking habits (smokers and amount of cigarettes smoked/day ">20or <20 cigarettes per day"). Blood pressure was measured twice in a sitting position after 5 min rest, and the average was recorded, those have blood pressure 140/100 mm Hg or more were considered hypertensive case. Random venous blood samples 10 ml, were collected from the individuals (Participants) in a tube containing anti-coagulant (EDTA). Hemoglobin (Hb) and glutathione reductase (GSH) in RBCs were determined immediately in the field using whole blood; the rest of the sample was centrifuged for 10 min. at 3000 rpm to obtain plasma. The plasma was divided in 3 ependurf tubes to estimate the liver, kidney functions and lipid peroxidation (MDA). The RBCs were washed twice by saline for cu/zn SOD determination. The plasma, washed RBCs and urine collected were stored at -40 Ċ till analysis. Urinary β2-microglobulin, liver and kidney functions were performed only for individuals who were diagnosed as hypertensive case (about 39% of participants).

Biochemical Analyses

The following parameters were determined: Hemoglobin (Hb%) according to **Hunter, (1978),** serum aspartate amino transferase (AST) and alanine amino transferase (ALT) according to **Reitman and Frankel (1957).** alkaline phosphatase (ALP) according to **Kochmar and Moss (1976),** gamma glutamyltransferase (GGT) according to (**Webster, 1974),** albumin according to **(Doumas, 1971),** blood urea, creatinine and uric acid were carried out according to **Patton and Crouch (1977), Tietz (1986)** and **Tietz (1994)** respectively.

malondialdehyde (MDA) was determined according to **Uchiyama and Mihara (1978),** Glutathione reductase activity was assayed according to **Smith et al. (1988),** erythrocyte copper and zinc supper oxide dismutase (cu/zn SOD) according to **Winterbourne et al. (1975)** and

β2-microglobulin according to **Poulik and Reisfeld (1975).**

Since, the biochemical analysis for Hb, GSH, MDA and SOD were done for all workers (direct and indirect exposure). While, the other biochemical analysis (Urinary β2-microglobulin, ALT, AST, ALP, GGT, Albumin, Urea, Creatinine and Uric acid) were done for sub-samples (136 cases ~_39%, 50 cases according to blood pressure (high blood pressure).

Statistical analysis

The results were expressed as mean ±SD. Data were analyzed by one way analysis of variance (ANOVA). The Differences between means were tested for significance using least significant difference (LSD) test at P <0.05 **(Steel and Torri, 1980).**

RESULTS

Tables (1&2) shows the results of indirect and direct exposure for (Hb%, GSH, MDA and Cu/Zn SOD), there was no significant difference in Hb for <5, >5 years of indirect exposure subjects There were significant difference for smokers <20 cigarettes between direct and indirect exposure. Also, there was significant difference between non-smokers direct and indirect exposure for >5 years.

However, there was significant difference between workers in GSH for non smokers <5, >5 years of indirect exposure subjects; there was no significance difference between smokers <5, >5 years workers on indirect exposure subjects. Also, there was significant difference in Cu/Zn SOD for non-smokers and smokers <5 years of indirect exposure.

From Table (2) it could be seen that there was significant difference in Hb levels between smokers and non-smokers <5, >5 years. However, there was significant difference in GSH between smokers and non-smokers of >5 years direct exposure, also between <5, >5 years of direct exposure for both non-smokers and smokers >20 cigarettes. On the other hand, there was significant difference in the level of MDA between smokers and non-smokers of

direct exposure. The concentration of Cu/Zn SOD was demonstrated to be significant different between non-smokers for <5, >5 years direct exposure.

Kidney functions of indirect and direct exposure were shown in tables (3&4). It could be seen from Table 4 that there was significant difference in serum creatinine for smokers and non-smokers of <5 years exposure. There were no significant difference in blood urea and β2-microglobulin in urine for smokers and non-smokers of <5 years exposure. However, significant difference in UA concentrations were observed between <5,>5 years of exposure, also between smoker and non-smoker of <5 years exposure.

Serum concentrations of liver functions of indirect and direct exposure were represented in tables (5&6). From Table 5, there was significant difference in serum albumin between smokers < 20 cigarettes and non-smokers of <5 years indirect exposure. There was significant difference in AST for smokers > 20 cigarettes of <5 years direct and indirect exposure.

From Table 6 we can see that there was significant difference in s. Albumin, AST and ALP for non-smokers between <5 and >5 years of exposure. Also, there was significant difference in AST for smokers >20 cigarettes of <5 and >5 years of exposure.

DISCUSSION

Cadmium was widely used in industry since one decade ago, where its health risks were recognized **(Eriksen, et al., 2015).** Many current observational studies reported positive associations between exposure to Cd and adverse effect on hemoglobin, study by **(Chen, et al., 2015)** revealed that the Hb of men with the highest level of blood cadmium decreased to 10.7 g/L compared to those with the lowest level of blood cadmium**,** which is consistent with the findings of the present study. Other study by **(Hounkpatin et al., 2013)** conducted on rats reported that there is a significant decrease in

the red blood cell (RBC) count & haemoglobin (Hb) concentrations, packed cell volume (PCV), mean corpuscular volume (MCV) & mean corpuscular haemoglobin (MCH), leading to anemia, on cadmium exposure.

The toxicity of Cd is associated with oxidative damage caused by the production of ROS **(Ivanina et al., 2008).** GSH is considered a primary defense mechanism against Cd, since its cysteine thiol group rapidly reduces the metal by forming a stable GS–Cd complex. Thus, the excessive consumption of GSH in metal reduction, chelation and oxidation by ROS leads to its significant depletion following exposure to high levels of Cd, compromising detoxification **(Rana et al., (2002)**. On the other hand, Study by **Sherif et al. (2010)** conducted on rats concluded that Cd exposure significantly increased the lipid peroxidation marker malondialdehyde (MDA), while the antioxidant enzyme glutathione reductase (GSH) significantly decreased. The presented results were in accordance with various previous reports suggesting that Cd can cause oxidative stress.

The results in our study show that in indirect cadmium exposure has no change in kidney functions neither among non smokers nor smokers. These results are in line with the result by **Mortada et al. (2004),** who concluded that exposure to cadmium due to cigarettes smoke is not high enough to produce nephrotoxicity. However, it may incite signs of nephrotoxicity in the presence of risk factors for kidney diseases.

The kidney is the principal organ targeted by chronic exposure to cadmium. Cadmium nephrotoxicity may follow chronic inhalation or ingestion. Data from human studies suggest a latency period of approximately 10 years before clinical onset of renal damage, depending on intensity of exposure **(Ikeda, 2005).**

The data obtained show no significant association between duration of occupational exposure to cadmium and

smoking cigarette at the time of exposure on batteries workers and urinary β2- microglobulin levels was found in our study. This could be due to the fact that the level of cadmium, which has considerable effect on tubular function, is not high enough in batteries sector. We believe more studies on urinary β2-microglobulin levels and other low molecular weight proteins are required to determine the effects of cadmium exposure in batteries sectors on renal function. Also the appropriateness of using urinary β2 microglobulin levels as a biomarker for tubular dysfunction in batteries workers deserves more research.

Our results are in line with the results by **Smith et al. (1988)** who concluded that kidney function status between the high and low exposure groups showed a significant reduction in creatinine clearance, and increased uric acid and beta2 microglobulin excretion by the high group.

The liver plays a crucial role in detoxification and excretion of many endogenous and exogenous substances, and its detoxification systems are easily overloaded. The outcome of cadmium exposure on the liver is hepatic cell changes **(Elias, 2013).** The present study reveals that an elevation in serum liver enzyme levels in direct cadmium exposure. Our results agreed with that by **kang et al. (2013)** who showed that environmental cadmium exposures are associated with an elevation in serum liver enzyme levels in Korean adults.

The significant increase in GGT between smokers and non-smokers is agreed with that of **Milnerowicz et al. (2010)** who observed that the activities of GGT were significantly increased in the group who smoked 20 or more 20 cigarettes a day in comparison to the non-smoking group. Also, **Lee and Jacobs (2009)** who reported that serum GGT within its reference range was linearly associated with important environmental pollutants, including lead and cadmium.

CONCLUSION

Adverse health effects of cadmium occur due to acute exposure and chronic exposure. Acute exposure to cadmium usually occurs when occupational workers are exposed to high doses of cadmium in industrial processes. The outcomes due to acute exposure are oxidative stress, anemia, renal and liver dysfunctions.

RECOMMENDATIONS:

Workers must be informed about the potential health effects associated with exposure to cadmium. This should include counseling on the effect of smoking on cadmium exposure.

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Table (1): Effect of cadmium exposure on (Hb GSH, MDA and Cu/Zn SOD) indirect exposure battery workers

Parameters / Exposure Period	**<5 YEARS**			**>5 YEARS**		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
NO, Case	55	16	10	5	6	6
Hb g/L	14.7 ± 1.9	14.9± 0.97[b]	14.16 ± 1.2	14.84 ±0.8[b]	14.7± 1.59	14.8 ± 0.72
GSH mg/dl	34.5 ±8.8[a]	38.8 ±7.7	38.8 ±8.5	19.34 ±3.732[a]	21.1 ±5.6	25± 6.2
MDA nmol/ml	1.8 ±0.41	1.19 ± 0. 31	1.366 ± 0.39	1.9±0.35	1.06 ±0.14	1.78 ±0.21
Cu/Zn SOD U/ml	126.5±20.4c	171.9±23.2	161.1 ±24.5c	172.2±38.23	168.2 ±25.6	184.0± 36.9

a ⟶ significant between <5,>5 years of exposure b ⟶ significant between direct and indirect exposure
c ⟶ significant between smoker and non-smoker

Table (2): Effect of cadmium exposure on (Hb, GSH, MDA and Cu/Zn SOD) direct exposure battery workers

Parameters / Exposure Period	**<5 YEARS**			**.>5 YEARS**		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
NO, Case	103	74	40	18	10	7
Hb g/L	14.9 ± 2.1[a]	14.1± 1.58[b]	14.61± 1.49[a]	15.92 ± 0.904[ab]	14.7±1.8	15.74 ± 0.84[a]
GSH mg/dl	32.56±9.09[ac]	38.64± 10.9	39.10±8.60[a c]	23.26±6.54[a]	18.3±3.9	25.65 ± 10[a]
MDA nmol/ml	1.86±0.38c	1.66±0.36	1.40 ±0.24c	1.97± 0.39	1.69±0.23	1.12 ± 0.19
Cu/Zn SOD U/ml	133.77±33.4ac	178.17±27.57	166.05±32.6c	178.82±23a	148.00±23.6	164.5±46.4

a → significant between <5,>5 years of exposure
b → significant between direct and indirect exposure
c → significant between smoker and non-smoker

Table (3): Effect of cadmium exposure on kidney functions indirect exposure battery workers.

Parameters / Exposure Period	**<5 YEARS**			**>5 YEARS**		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 **cigarettes**	>20 **cigarettes**		<20 **cigarettes**	>20 **cigarettes**
NO, Case	32	6	12	-	-	-
Creat. mg/dl	0.878 ± 0.143	0.95 ± 0.101	0.90 ± 0.282	----	----	----
Urea mg/dl	33.50± 9.112	30.66± 8.041	28.50 ± 3.45	----	----	----
UA mg/dl	4.650 ± 1.04	4.033 ±1. 09	4.550 ± 0.212	----	----	----
β2-microglobulin in urine µg/ml	0.067 ± 0.008	0.083 ± 0.001	0.02 ±0.003	----	----	----

a → significant between <5,>5 years of exposure
b → significant between direct and indirect exposure
c → significant between smoker and non-smoker

Table (4): Effect of cadmium exposure on kidney functions direct exposure battery workers.

Parameters / Exposure Period	**<5 YEARS**			**.>5 YEARS**		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 **cigarettes**	>20 **cigarettes**		<20 **cigarettes**	>20 **cigarettes**
NO, Case	39	18	16	13	-	-
Creat. mg/dl	0.848 ± 0.269[c]	1.055 ± 0.125	1.093 ± 0.154[c]	0.766 ± 0.088	----	----
Urea mg/dl	34.33 ±8.76	37.66 ±9.75	31.25 ±8.76	40.0 ±8.08	----	----
UA mg/dl	4.95± 1.06[ac]	3.98±1.05[c]	4.437±1.02	7.83 ±2.0[a]	----	----
β2-microglobulin in urine µg/ml	0.064± 0.014	0.061± 0.005	0.059±0.004	0.073 ± 0.018	----	----

a → significant between <5,>5 years of exposure
b → significant between direct and indirect exposure
c → significant between smoker and non-smoker

Table (5): Effect of cadmium exposure on liver functions in indirect exposure battery workers

Parameters / Exposure Period	**<5 YEARS**			**>5 YEARS**		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 **cigarettes**	>20 **cigarettes**		<20 **cigarettes**	>20 **cigarettes**
NO, Case	32	16	12	--	--	--
Albumin g/L	4.221± 0.659[c]	3.533± 0.445[c]	3.90±0.414	----	----	----
AST IU/L	26.0±5.50	22.0±3.84	23.00 ±5.71[b]	----	----	----
ALT IU/L	17.0±8.06	13.00 ±2.96	19.00 ± 2.77	----	----	----
GGT µkat/L	17.0±3.54	13.0±1.86	26.0±3.12	----	----	----
ALP IU/L	112.57±21.62	102.40±18. 07	126.00 ±13.00	----	----	----

a → significant between <5,>5 years of exposure
b → significant between direct and indirect exposure
c → significant between smoker and non-smoker

Table (6): Effect of cadmium exposure on liver functions in direct exposure battery workers.

Parameters / Exposure Period	**<5 YEARS**			**>5 YEARS**		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 **cigarettes**	>20 **cigarettes**		<20 **cigarettes**	>20 **cigarettes**
NO, Case	39	18	16	23	-----	14
Albumin g/L	4.43± 0.727[a]	4.05 ± 0.80	4.225 ± 0.95	3.850 ± 0.21[a]	----	4.12± 0.85
AST IU/L	23.0±5.60[a]	20.0 ±4.84	19.0 ±3.7[ab]	42.0±12.8[a]	----	42.0 ±14.4[a]
ALT IU/L	15.55±3.168	16.86 ±4.61	11.94± 2.92	15.00±3.76	----	9.0±1.2
GGT µkat/L	13.0±2.54[bc]	16.40 ±1.85	22.0 ± 2.12[c]	25.00±3.09	----	17.0±3.8
ALP IU/L	112.57 ±12.62[a]	102.40 ± 18.077	126.0 ±17.85	78.66 ± 9.237[a]	----	91.0±21.36

a → significant between <5,>5 years of exposure
b → significant between direct and indirect exposure
c → significant between smoker and non-smoker

تأثير التعرض للكادميوم في صناعة البطاريات

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الملخص العربي

يعد الكادميوم من العناصر شديدة السمية للأنسان حيث أنه يتراكم في الأنسجة الرخوة و الكلي والكبد. لذا تهدف هذه الدراسة لتعين تأثير التعرض لفترات طويلة للكادميوم و التدخين أثناء فترات التعرض علي وظائف الكلي و الكبد للعاملين الذين يعملون في صناعة البطاريات في مصر حيث يعد الكادميوم من العناصر الرئيسية في صناعة البطاريات. أجريت هذه الدراسةعلي مجموعة من البالغين و الذين لا يعانون من اماض كلي أو كبد. حيث أنه تم محاولة إيجاد علاقة بين مدي التعرض للكادميوم و قيمة كلا من اسبرتات امينوترانسفيراز (AST), ألنين أمينوترانسفيراز (ALT)، ألكلين فوسفاتيز (ALP)، جاما-جلويتاميل ترانسفيراز (GGT)، ألبيومين،كرياتينين،حمض البوليك (UA)، البولينا، مالوندالدهايد (MDA) في مصل الدم، CU/ZN سوبرأكسيد داسماتيز (CU/ZN SOD) ،جليتاثيون ريداكتيز (GSH) في كرات الدم الحمراء ، الهيموجلوبين في الدم،بيتا٢ ميكروجلبيولين في البول. مع المقارنة بين التعرض المباشر و الغير مباشر للتلوث بالكادميوم و كذلك المدخنين و عدد السجائر الذين يدخنونها في اليوم. أوضحت الدراسة أن هناك تأثير عكسي لمدة التعرض للكادميوم علي كلا من الهيموجلوبين ،GSH ،UA, GGT لذا يجب تقديم النصائح للعاملين في مجال البطاريات علي التأثير السلبي للكادميوم علي الصحة.

الكلمات المفتاحية: *الكادميوم – مضادات الاكسدة في الكبد والكلى – التسمم- ملوثات البيئة- التدخين*