Egyptian J. of Nutrition Vol. XXXIII No. 2 (2018)

Effect of Cadmium Exposure among workers in Iron-Steel Industries

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Abstract

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.**Aim of the study:**The study aimed to assess the possible influence of occupational exposure to cadmium as well as smoking cigarettes on renal and liver functions in iron steel manufacturing. This cross-sectional study evaluated liver, kidney function, oxidative stress and lipid per oxidation among smoker and non smoker workers in iron-steelmanufacturing(mean age, 35 years) in Egypt.We measured the plasma 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 (SOD), glutathione reductase (GSH), malondialdehyde (MDA), blood hemoglobin and β2-microglobulin in urine, adjusting for smokingand the amount of cigarette smoked. Subjects were stratified into direct and indirect exposure according to their place of exposure. Our results show positive associations between exposure to cadmium and adverse effect on Hb, reductase GSH, cu/zn SOD and GGT.

The outcomes due to acute and prolonged chronic cadmium exposure are oxidative stress, anemia, renal and liver dysfunctions.
Cadmium, Renal toxicity, hepatotoxicity, Smoking

Introduction

Cadmium (Cd) is considered one of the most toxic elements in the environment, with a wide range of organ toxicity and long elimination half-life ***(Patrick, 2003).*** Cadmium (Cd) is a toxic metal present in food, tobacco smoke, air, water and other media, and it can enter human bodies through inhalation, ingestion and dermal contact, Cd can accumulate over time in various organs and tissues, but mostly in kidney cortex ***(Satarug et al., 2013).*** Cadmium concentrates in the kidney, liver and various other organs and is considered more toxic than either lead or mercury. It is toxic at levels one tenth that of lead, mercury, aluminum, or nickel. Exposure to cadmium is also increasing due to its use as a coating for iron, steel and copper ***(Ray et al., 2014).***Bioavailability, retention and consequently toxicity of Cd are affected by several factors such as nutritional status (low body Fe stores) and multiple pregnancies, pre-existing health conditions or diseases ***(European Food Safety Authority, 2009).*** In humans, cadmium takes a special ionic form, which resembles ionic calcium, this enables cadmium to disrupt biologic pathways involving calcium, leading to bone and muscle issues ***(Choong et al., 2014).*** More than 90% of Cd in the surface environment is the result of industrial and agricultural processes ***(Roggeman et al., 2014).*** Environmental exposure to cadmium occurs primarily through industrial emissions, smoking, and the consumption of contaminated food and water ***(Hyder et al., 2014).***

Age and smoking status were the greatest contributors to blood and urine cadmium, older people and current smokers had the highest cadmium levels***(Garner and Levallois, 2016).***

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 iron-steel manufacturing.

Subjects *and 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 397 workers (participants) healthy male workers exposed to cadmium from iron-steel industries in Egypt, aged 22-60 years (mean age, 35 years). Two exposure groups had been selected, an indirect exposure group of office, laboratory and supervisory personnel (n=122) (indirect exposure group); and a high exposure group of production workers (direct exposure group)(n=275).These two groups were divided into subgroups according to duration of exposure and who are smoker and those who had never smoked. This study wasconducted in (Helwan for Iron-steel, Mostored for Iron-Steel and El-Nahas Egypt Cos.,) Cairo and Alexandria, Egypt.

Experimental Design

Study protocols were reviewed and approved by the ethical committee of National Nutrition Institute in Egypt and consents were obtained from all participants. A total of 397 adult males were included in this study were interrogated in order to have detailed information about the age, medical history of (liver, renal diseases and diabetes), work period (<5 years, represent "160 cases"& >5 years, represent 237 cases) and smoking habits (who is smoker and amount of cigarettes smoked/day ">or < 20 cigarettes per day"). Blood pressure was measured twice in a sitting position after 5 min rest, and the average was recorded, who blood pressure was 140/100 mm Hg or more was considered a hypertensive case. Random venous blood sample 10 ml. was collected from the individuals (Participants) in tube containing anti-coagulant (EDTA). HB% and glutathione reductase (reduced form) were determined immediately in the field from whole blood; the rest of the sample was centrifuged for 10 min. at 3000 rpm to obtain the plasma. The plasma was divided into 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 diagnosed as a hypertensive case (about 48% 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 by***(Patton and Crouch, 1977), (Tietz, 1986)*** and ***(Tietz, 1994)*** respectively. 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., 1974)*** and β2-microglobulin according to ***(Poulik and Reisfeld, 1975).***

Since, the biochemical analysis for Hb%, GSH, MDA and SOD were done for 397 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 (189 samples ~_48%, 55 samples from indirect exposure and 134 samples from direct exposure) chosen 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).*** For all tests, $p < 0.05$ was considered as statistically significant.

Results

HB, GSH, MDA and Cu/Zn SOD obtained from indirect and direct Cd exposure workers were given in tables (1&2). There was significant difference in Hb concentration between non-smokers and

smokers <20 cigarettes in both <5 and >5 years of indirect exposure. However, there was significant difference in the antioxidant enzyme glutathione (GSH) (reduced form) for non smokers and smokers <20 cigarettes of >5 years indirect exposure. While, significant difference was seen in the lipid peroxidation marker malonal-dialdehyde (MDA) for non-smokers <5 and >5 years of indirect exposure subjects. On the other hand, there was significant difference in Cu/Zn SOD for non-smokers and smokers <20 cigarettes for <5 years between indirect and direct exposure, also between non-smokers and smokers for >5 years of indirect exposure.

It can be seen from Table 2 that there was significant difference in Hb concentration between non-smokers and smokers in both <5 and >5 years of direct exposure subjects. However, there was significant difference in GSH between smokers <20 cigarettes for <5 and >5 years.

On the other hand, Cu/Zn SOD shows a significant difference between non-smokers, smokers <20 cigarettes of <5 and >5 years direct exposure, also between smokers <20 cigarettes and non-smokers in both <5 and >5 years direct exposure.

Kidney functions of indirect and direct exposure were shown in tables (3&4).It can be seen from Table 3 that there was significant difference in serum creatinine between <5 and >5 years exposure for smokers and non-smokers. Also, between indirect and direct for non-smokers and smokers of <5 years exposure. There were significant difference in blood urea between smokers>20 cigarettes of >5 years indirect exposure and direct exposure. However, significant difference in UA concentrations were observed between non-

smokers <5 and >5 years of indirect exposure. On the other hand, β2-microglobulin in urine shows a significant difference between smokers <20 cigarettes of <5 direct and indirect exposure. The result in Table 4 shows significant difference in serum creatinine between smokers <20 cigarettes <5 years and non-smokers >5 years of direct exposure. Also, significant difference in blood urea between smokers <20 cigarettes <5 years and non-smokers >5 years of direct exposure.

Plasma concentrations of liver functions of indirect and direct exposure were present in tables (5&6). It can be seen from Table 5 that there was significant difference in plasma albumin between non-smokers <5 and >5 years of indirect exposure. Also, between non-smokers & smokers >20 cigarettes<5 years of direct and indirect exposure. However, there was significant difference in s. albumin between smokers <20 cigarettes <5 and >5 years of exposure. Also, between smokers >20 cigarettes<5 of direct and indirect exposure.
From Table 6 we can see that there was significant difference in ALT between smokers of <5 and >5 years of exposure. Also, there was significant difference in GGT for smokers >20 cigarettes of <5 and >5 years of exposure.

Discussion

Cadmium is an extremely toxic metal which has no known necessary function in the body. Cadmium toxicity contributes to a large number of health conditions. Cadmium concentrates in the kidney, liver and various other organs and is considered more toxic than either lead or mercury. It is toxic at levels one tenth that of lead, mercury, aluminum, or nickel. Exposure to cadmium is also

increasing due to its use as a coating for iron, steel and copper ***(Ray et al., 2014).*** The purpose of the present study to identify the adverse health effect of cadmium exposure on iron-steel male workers aged 22 to 60 years in Egypt.

The present study reveals that the workers who indirect or direct exposed to Cd showed significant reduction in Hb depending on smoking habit and duration of exposure.Our results in agreement with study by ***Fatma et al. (2004)*** conducted on rats showed that cadmium chloride(5 mg/kg BW) were orally administered caused changes in the blood indices of rats. The reduction in Hb content may be due to increased rate of destruction or reduction in the rate of formation of erythrocytes.

The present study tables (1&2) on reductase GSH and cu/zn SOD activities showed that as exposure increase in both indirect and direct exposure the activity of these antioxidant enzymes increase. The presented results were in accordance with the study by ***(Fangming et al., 2013)*** who demonstrated that the antioxidant enzyme system was initiated under the Cd exposure, i.e. almost all the activities of superoxide dismutase (SOD), peroxidase, catalase, glutathione peroxidase, and ascorbate peroxidase were elevated. Also, study by ***(Stoiber et al., 2010)*** who concluded that Cd stimulates glutathione reductase enzyme activity and increases glutathione concentrations as cells respond to Cd-induced stress by producing increased antioxidant capacity.

On the other hand, there was significant difference in Cu/Zn SOD for non-smokers and smokers <20 cigarettes for <5 years between indirect and direct exposure. This finding is in agreement

with ***(Olabsis et al., 2016)*** who reported that exposure to cadmium resulted in significant decrease in SOD activity in plasma, erythrocytes, brain and liver. Also, ***(Tobwala et al., 2014)*** who reported that administration of cadmium, both in vivo and in vitro inhibit SOD activity.

The result obtained in our present study show that significant difference in the lipid peroxidation marker malonal-dialdehyde (MDA) for non-smokers <5 and >5 years of indirect exposure subjects. This finding in line with study by***(Shi et al., 1999)*** conducted on rats concluded that Malonaldialdehyde (MDA) concentration significantly increased in the blood of Cd-treated rats which accompanied by increased formation of reactive oxygen species (ROS).

Cadmium is a nephrotoxic metal that accumulates in the proximal tubules of the kidney and caused increased urinary excretion of low molecular weight proteins due to impaired tubular reabsorption ***(Akerstrom et al., 2013).*** In our study tables (3&4) we found no association between exposure to cadmium (direct and indirect) and renal functions in iron-steel workers. Our results in line with the results by***(Omid et al., 2011)*** conducted on welders which showed no statistically difference in renal functions (β2 microglobulin and creatinine) between welders and controls. This may be due to workers (direct and indirect) exposed are not high enough to produce nephrotoxicity, and hence much work is underway to define “critical renal concentration” at which cadmium-induced renal damage occurs.

The liver is the critical organ in the case of a long-term occupational or environmental exposure to heavy metals and tobacco smoke. The obtained results demonstrated in (Tables 5&6) revealed

that, there was significant difference in plasma albumin between non-smokers <5 and >5 years of indirect exposure. Also, between non-smokers & smokers >20 cigarettes<5 years of direct and indirect exposure. Moreover, a significant difference in ALT between smokers of <5 and >5 years of exposure. Also, there was significant difference in GGT for smokers >20 cigarettes of <5 and >5 years of exposure. Similar results were recorded by ***(Milnerowicz et al., 2010)*** who observed that, the value of GGT was high in serum of smellers who smoked 20 or over 20 cigarettes per day than in non-smokers smelters. Our results are in agreement with those obtained by ***(Salim, 2013)*** who concluded that the mean activity of serum GGT in cigarette smokers was significantly higher than that of non-smoker individuals. Previous studies also has reported a significant rise in serum GGT activity despite that in other studies showed that other liver enzymes like serum ALT, AST and ALP activity were no significant elevation in moderate cigarette smokers when compared to control group ***(Abdulrazaq, 2012).*** Our results are in agreement with the results by ***(Hussien et al., 2009)*** conducted on rabbit exposed to cadmium revealed that, a significant increase in plasma ALT, AST, urea and creatinine.

Study on rats by ***(Markiewicz-Górka, et al., 2015)*** observed that combined exposure to lead, cadmium, and manganese mixtures cause AST significantly differed from control and the Pb 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 table 6 reveals that an elevation in serum liver enzyme levels in direct cadmium exposure. Our results are in

agreement with results 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 (Table 6) is in agreement with the results of ***(Milnerowicz et al., 2010)*** who observed that the activities of GGT were significant increased in group who smoked 20 or over 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, includinglead, 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 industrially processes. The outcomes due to acute and prolonged chronic exposure are oxidative stress, anemia, renal and liver dysfunctions.

Recommendation

In order to avoid harmful effect of exposure to cadmium, workers on iron- steel production lines should consume large amounts of the sources of antioxidants by eating onions, garlic, watercress,cumin, turmeric, pepper and fruit like guava and citrus fruit at breakfast. Also, workers on production lines should be using

the means of prevention, such as masks and filters, avoid smoking and awareness of smoking hazards.

- Medical surveillance involves monitoring of biological indicators of cadmium exposure and toxicity. This includes cadmium in blood (CdB), cadmium in urine (CdU), and beta-2-microglobulin levels in urine (B 2 -M).

Table (1): Mean ± S.D. of (HB, GSH, MDA and Cu/Zn SOD) in indirect cadmium exposure iron-steel workers.

Exposure Period / Parameters	<5 YEARS			>5 YEARS		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
No. of Cases	14	14	11	46	22	15
HB (13-17 g/dL)	15.7± 1.4[a]	15.1± 1.8[a]	13.2	14.2 ± 1.7[a]	13.6± 1.4[a]	13.5± 1.1
GSHmg/dl	46.3± 13.2	42.9 ± 10.5	41.8± 9.5	43.3± 12.9c	41.1± 10.9c	39.9± 9.9
MDAnmol/ml	1.3± 0.19[a]	1.4 ± 0.32	1.45± 0.25	1.391± 0.17[a]	1.47± 0.44	1.68 ± 0.26
Cu/Zn SODU/ml →	174.6± 38.1[b]	179.9 ±29.3[b]	284.8± 42.8	128.7± 70.3	140.7± 38.6[c]	165.7± 28.1 [c]

a → significant between <5,>5 years of exposure

b → significant between direct and indirect exposure

c significant between smoker and non-smoker

Table (2): Mean ± S.D. of (HB, GSH, MDA and Cu/Zn SOD) in direct cadmium exposure iron-steel workers..

Exposure Period / Parameters	<5 YEARS			>5 YEARS		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
No. of Cases	46	59	16	89	50	15
HB(13-17 g/dL)	15.51± 1.5[a]	15.89± 1.4[a]	16.3± 2.3[a]	14.03± 1.8[a]	13.5± 1.7[a]	13.9 ± 1.97[a]
GSHmg/dl	38.01 ± 13.5	40.9± 12.4[a]	44.3 ± 16.6	38.2± 8.3	38.8± 8.9[a]	39.6± 10.7
MDAnmol/ml	1.62± 0.42	1.496± 0.38	1.77 ± 0.48	2.1± 0.4	2.1± 0.31	1.908± 0.52
Cu/Zn SODU/ml	139.6± 42.5[abc]	144.9 ± 32.8[abc]	146.9 ± 33.53	247.8± 59.1[a]	223.1± 32.1[a]	218.3± 45.1

a ⟶ significant between <5,>5 years of exposure

b ⟶ significant between direct and indirect exposure

c ⟶ significant between smoker and non-smoker

Table (3): Mean ± S.D. of kidney functions in indirect cadmium exposure iron-steel workers.

Exposure Period / Parameters	<5 YEARS			>5 YEARS		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
No. of Cases	7	6	5	20	9	8
Creat. (0.6-1.2mg/dl)	1.57 ± 0.43[ab]	1.65 ± 0.44[ab]	2.0± 0.19[ab]	0.94 ± 0.364[a]	0.922± 0.232[a]	0.9 ± 0.125[a]
Urea(10-50mg/dl)	42.57± 11.39	46.66± 7.763	55.00± 6.8	35.35± 8.73	37.66± 9.20	38.13± 2.23[b]
UA(2-7mg/dl)	4.528 ± 0.706[a]	6.133± 0.791	4.5± 0.95	5.58± 1.11[a]	5.877± 1.037	5.50± 1.541
β2-microglobulin in urine(0-0.3 µg/ml)	0.049 ± 0.033	0.064± 0.038	0.068± 0.051	0.070± 0.062	0.095± 0.074	0.107± 0.103

a → significant between <5,>5 years of exposure

b → significant between direct and indirect exposure

c → significant between smoker and non-smoker

Table (4): Mean ± S.D. of kidney functions in direct cadmium exposure iron-steel workers.

Exposure Period / Parameters	<5 YEARS			>5 YEARS		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
NO, Case	20	37	14	32	23	8
Creat.(0.6-1.2mg/dl)	0.905± 0.188[b]	1.0± 0.261[ab]	0.75 ± 0.188[b]	0.965± 0.248[a]	1.01± 0.120	0.762± 0. 226
Urea(10-50mg/dl)	38.025± 7.554	41.27± 10.06[a]	38.250 ± 6.551	35.16± 7.12[a]	35.17± 6.79	31.00± 5.13[b]
UA(2-7mg/dl)	6.510± 1.95[b]	5.80± 1.441	5.10 ± 1.651	5.815± 1.465	6.24± 1.24	5.75 ± 1.353
β2-microglobulin in urine(0-0.3 µg/ml)	0.070± 0.062	0.097± 0.116	0.12± 0.06	0.11± 0.042	0.129± 0.08	0.149± 0.015

a ⟶ significant between <5,>5 years of exposure

b ⟶ significant between direct and indirect exposure

c ⟶ significant between smoker and non-smoker

Table (5): Mean ± S.D. of Liver functions in indirect cadmium exposure iron-steel workers.

Exposure Period / Parameters	<5 YEARS			>5 YEARS		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
NO, Case	7	6	10	20	9	8
Albumin (3.5-5.2g/L)	4.23± 0.53[ab]	4.44 ± 0.44[ab]	4.60 ± 1.21	4.90 ± 0.634[a]	5.02 ± 0.551[a]	4.60 ± 0.810
AST(0-40 U/L)	26.0± 5.08	33.0± 26.94	12.00± 2.3	28.00± 3.763	41.0± 5.337	49.0± 8.99
ALT(0-30 U/L)	18.0± 4.03	13.0 ± 2.95	15.00± 1.70	17.00± 4.480	26.0 ± 8.079	30.0± 8.13
GGT(9–50 U/L)	18.0± 5.67	15.00± 3.871	17.00± 2.3	23.00± 6.281	30.0± 8.94	16.0± 5.78
ALP(50-160 U/L)	91.14 ± 11.7	83.4 ± 8.76	104.0± 11.5	91.6± 5.85	113.3± 10.81	119.25± 22.5

a ⟶ significant between <5,>5 years of exposure

b ⟶ significant between direct and indirect exposure

Table (6): Mean ± S.D. of Liver functions in direct cadmium exposure iron-steel workers..

Exposure Period / Parameters	<5 YEARS			.>5 YEARS		
	Non-Smoker	Smoker		Non-Smoker	Smoker	
		<20 cigarettes	>20 cigarettes		<20 cigarettes	>20 cigarettes
NO, Case	20	37	24	32	23	18
Albumin (3.5-5.2 g/L)	5.045 ± 0.815[b]	4.978 ± 0.920[b]	5.125 ± 1.056	4.693 ± 0.703	4.852 ± 0.661	4.750 ± 0.585
AST(0-40 U/L)	25.0± 5.43	24.0± 6.416	20.0± 3.1	26.0± 5.36	29.0± 7.384	33.50 ± 5.99
ALT(0-30 U/L)	15.0± 3.85	13.0± 2.340[a]	11.0± 1.88[a]	18.0± 5.76	22.0± 6.9[a]	25.0± 5.19[a]
GGT(9–50 U/L)	17.0± 3.76	17.0± 4.67	19.0± 5.05[a]	19.0± 3.36	21.0± 4.305	31.0± 6.94[a]
ALP(50-160 U/L)	91.11± 15.2	94.02± 24.33	94.66 ± 9.86	105.71± 21.06	102.2± 20.75	125.8± 14.1

a ⟶ significant between <5,>5 years of exposure

b ⟶ significant between direct and indirect exposure

c ⟶ significant between smoker and non-smoker

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تأثير التعرض للكادميوم علي العاملين في صناعة الحديد والصلب

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الملخص العربي

زيادة تركيز الكادميوم في جسم الإنسان تؤدي إلى أضرار كثيرة، منها الإصابة بالفشل الكلوي، إذ يخزن الجسم الغالبية العظمى من الكادميوم في الكليتين، وفي الكبد والأعضاء التناسلية أيضا، وغالبا ما تحدث أمراض الكلى عندما تصل كميات الكادميوم في الكلى بتركيزات تصل إلى ٢٠٠ جزء في المليون، ويتخلص الجسم من الكادميوم عادة ببطء، ويرتبط هذا التخلص بنوعية الغذاء. لذا تهدف هذه الدراسة لتعيين تأثير التعرض لفترات طويلة للكادميوم و التدخين أثناء فترات التعرض علي وظائف الكلي والكبد للعاملين الذين يعملون في صناعة الحديد والصلب في مصر حيث يعد الكادميوم من العناصر الرئيسية في صناعة الحديد والصلب.

ولقد أجريت هذه الدراسةعلي مجموعة من البالغين و الذين لا يعانون من أمراض 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 (ZN SOD/ CU),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.

ولقد أوضحت الدراسة أن هناك تأثير عكسي لمدة التعرض للكادميوم علي كلا من الهيموجلوبين,سوبرأكسيد داسماتيز (CU/ZN SOD) ,جليتاثيون ريداكتيز (GSH), GGTلذا يجب تقديم النصائح للعاملين في مجال صناعة الحديد والصلب علي التأثير السلبي للكادميوم علي الصحة.