Assessment of Respiratory and Renal Functions Among Gas Metal Arc Welders and Their Relations with Chromium Exposure

Afify, M.[(1)], Helal, S.F.[(2)], and Arafaa, A.M.[(3)]

Biochemistry Department, National Research Center[(1)]
33 Bohooth Street, Giza, Egypt

Industrial Medicine and Occupational Diseases Department[(2)]
NECTR[(3)]
Faculty of Medicine-Cairo University

ABSTRACT

Background: *Welding is the most common way of permanently joining metal parts. Welding, soldering, and brazing workers are often exposed to a number of hazards, including the intense light created by the arc, poisonous fumes, and very hot materials.*

Objective: *The aim of this study was to investigate some of the health hazards among gas metal arc welders including renal and pulmonary function tests and to monitor the level of chromium in blood and urine, to study its relation with the renal condition. In addition, our aim was to evaluate β_2 microglobulin in urine as a predictive marker for early renal affection.*

Patients and methodology: *This study was conducted on 18 workers in metal arc welding shops in El Maniel, and El Maadi, Cairo, Egypt. They were adult men aged between 18-42 years, working 12 hours/day with one day off per week. 20 males matched for age, sex, socio-economic status, smoking habits served as a control group. All workers were interviewed using a special questionnaire including occupational history. Ventilatory function tests were done measuring FVC, SVC, FEV1%, FEV_1/FVC, MEF 25-75% and PEF. Detection of urinary β_2 microglobulin level, chromium levels in blood and urine, blood urea, serum creatinine and fasting serum sugar levels.*

Results*: Showed statistically significant difference ($P<0.05$) between the exposed and the control groups as regards spirometric evaluation of FEV_1/FVC, PEF%, FVC%, SVC% and MEF 25-75% due to bronchospasm from chemical irritation of the lungs which was related to chromium exposure and its level in blood. It also revealed statistically significant correlation ($P<0.05$) between the level of blood chromium and renal insult represented by the affection of urinary β_2 microglobulin, urea, and creatinine among the exposed workers indicating renal tubular damage. The results showed no statistically significant correlation ($P>0.05$) between the duration of exposure to chromium in welders and all investigations done, because chromium is rapidly cleared from the blood, and measurements are related only to the recent exposure. There is a statistically significant correlation ($P<0.05$) between the levels of chromium in blood, and each of the following: spirometric measures, level of urinary chromium, urinary β_2 microglobulin, serum urea, and creatinine.*

Conclusion and recommendations: *the primary target organ for subchronic and chronic chromium toxicity is the respiratory system, with high levels of blood and urine chromium there was a great insult on the pulmonary functions. Predominant renal injury among welders is tubular, and this injury is correlated*

Afify, M.; Helal, S.F.; Arafaa, A.M. (2007) Assessment of Respiratory and Renal Functions Among Gas Metal Arc Welders and Their Relations with Chromium Exposure. In *Defence against the Effects of Chemical Hazards: Toxicology, Diagnosis and Medical Countermeasures* (pp. 22-1 – 22-12). Meeting Proceedings RTO-MP-HFM-149, Paper 22. Neuilly-sur-Seine, France: RTO. Available from: http://www.rto.nato.int.

with the blood chromium level, urinary β_2-microglobulin level could be used as early marker for renal affection. We recommend environmental monitoring in different areas in the welding shops to ensure that permissible levels of different hazardous agents are not exceeded. Pre-employment and periodic medical examinations must be performed to exclude those susceptible to lung and renal diseases. Personal protective clothes e.g. gloves and respiratory protective equipment should be used in welders. Health education programs of workers about hazards of contact with harmful agents and proper measures for protection are essential. Effective local exhaust ventilation to remove hazardous gases and vapors is essential.

Key words: *Metal arc welders, Chromium, Beta$_2$- microglobulin.*

INTRODUCTION

Gas Metal-Arc Welding (GMAW), also called Metal Inert Gas (MIG) welding, shields the weld zone with an external gas such as argon, helium, carbon dioxide, or gas mixtures. Deoxidizers present in the electrode can completely prevent oxidation in the weld puddle, making multiple weld layers possible at the joint. GMAW is a relatively simple, versatile, and economical welding apparatus to use. This is due to the factor of 2 welding productivity over SMAW processes. In addition, the temperatures involved in GMAW are relatively low and are therefore suitable for thin sheet and sections less than ¼ inch. GMAW may be easily automated, and lends itself readily to robotic methods. It has virtually replaced SMAW in present-day welding operations in manufacturing plants **(Philip et al., 2004)**.

In welding or cutting, the intense heat of the arc or flame vaporizes the base metal and/or the electrode coating. This vaporized metal condenses into tiny particles called fumes. These fume particles can be inhaled. Chromium fume is created by welding or cutting on stainless steel or metals that are coated with a chromium material. Welding on stainless steel without adequate control measures can lead to exposure at least several times above the legal exposure limit. http://www.bls.gov/oco/ocos226.htm

Among the short-term effects of welders' exposure is metal fume fever, symptoms of which occur 4-12 hours after exposure and include chills, fever, muscle ache, chest soreness, coughing, wheezing, fatigue, nausea, and a metallic taste. Welding smoke can irritate the eyes, nose, chest and respiratory tract and cause coughing, wheezing shortness of breath, bronchitis, pulmonary edema, and pneumonitis. Gastro-intestinal effects such as nausea, appetite loss, vomiting, cramps, and slow digestion also have been associated with welding smoke. Ultra-violet radiations given off by welding reacts with oxygen and nitrogen in the air to form ozone and nitrogen oxides, which are deadly at high doses, irritate the nose and throat, and cause serious lung diseases **(Vicki Bell, 2004)**.

Long term effects include increased risk of lung cancer and possible larynx and urinary tract cancer as welding smoke, includes cancer causing agents such as cadmium, nickel, chromium, beryllium, and arsenic. Welders also may have chronic respiratory problems. Other health problems that appear to be related to welders include heart diseases, skin diseases, hearing loss, and chronic gastritis. Studies have shown that welders, especially those who work with stainless steel, have poorer sperm quality than men in other works. Other studies reported neurodegenerative diseases that affect mental and physical ability **(Vicki 2003)**.

Glomerular injury has been noted in chromium workers, in chrome platters, in stainless steel welders. The predominant renal injury is tubular. Low doses chromium exposure typically results only in transient renal effects **(Powers et al., 1986)**.

Increased urinary levels of B_2 microglobulin are found in people with kidney damage caused by high exposure to the heavy metals. Periodic testing of workers exposed to these metals helps to detect beginning kidney damage **(Henry 1996)**.

AIM OF THE WORK

The aim of this study was to investigate some of the health hazards among gas metal arc welders including renal and pulmonary function tests and to monitor the level of chromium in blood and urine, to study its relation with the renal condition. In addition, our aim was to evaluate $ß_2$ microglobulin in urine as a predictive marker for early renal affection.

SUBJECTS AND METHODS

This study was conducted in three welder shops in El Maniel, and El Maadi, Cairo. The study was accomplished during the period from October to November 2006. The studied group comprised 18 workers in the metal arc welding shops. They were adult men aged between 18-42 years (26.5 ±7.8), working on the basis of 12 hours/day with one day off per week. None of the workers used any protective equipment during working hours. A referent group of 20 males matched for age that ranged from 21-41 yrs. (26.2±6.38), sex, socio-economic status, smoking habits selected from relatives of the Kasr El Eini hospital patients, were also enrolled in our study.

Blood sample collection

From each subject 10 cc of venous blood were taken through a vein puncture using a dry plastic disposable syringe under complete aseptic condition. Three cubic centimeters of blood were taken into a clean tube containing anticoagulant for determination of chromium level in blood. The remaining 7 cc of blood were kept in a tube and allowed to clot then centrifuged for separation of the serum for determination of the biochemical parameters using Hitashi (911) auto analyzer: i-Kidney function test as urea and creatinine, ii- Random serum sugar level. All Samples were transported to the laboratory on the same day within two hours to be analyzed.

Urine sample collection

A urine sample was collected from each subject, in a sterile container. All subjects washed their hands with soap and water prior to sample collection to avoid contamination.

The following investigations were performed after taking individual consent:

- All workers were interviewed using a special questionnaire including occupational history; and full clinical examination was performed.
- Ventilatory function tests were done using portable spirometry connected to a portable computer using the soft ware ZAN program, measuring FVC, SVC, FEV1%, FEV1/FVC, and PEF.
- Detection of $Beta_2$ microglobulin in urine. Random urine sample is used, volume between 1-10 ml, taken in plastic urine container (not acidified). Patients were instructed to empty their bladders then drink a large glass of water and then we collect the urine samples from them within one hour at Kasr El-Eini Hospital (Industrial and Occupational Department). The samples were kept in the refrigerator till transferred to the lab. Significant loss of B_2 microglobulin activity may occur in acidic urine (pH <or = 6), so pH of urine is adjusted to be (6-8) with 1 mol/L sodium hydroxide. Analysis of B_2 microglobulin in urine was done using immunochemiluminometric assay (ICMA). Normal range (0-160) ug/l **(Henne et al., 1997)**.
- Determination of chromium in blood and urine:
  The samples for blood chromium level were prepared by dilution of 0.5 ml of blood with 2 ml deionized water. The samples of urine were prepared by dilution of 1 ml of urine with 1ml of deionized water. The chromium in blood and urine were measured by graphite furnace atomic absorption spectrophotometer (Perkin-Elmer model 5100 PC, Norwalk, CT).

1-Sample preparation:
Three per cent butan-1-ol was added to samples and standards to match the carbon content with the aim of ensuring that the ionization efficiency of elements such as chromium is the same in all solution. TAMA 0.1% chemicals (Kwasaki city, Japan) super cleaning, (high purity surfactant), was added to maintain a stable emulsion with the diluted sample. HNO_3 0.05% was added to ensure that the trace elements are maintained in solution and to aid wash out of these elements between samples; the acid concentration was kept to a minimum, otherwise cellular component in blood sample in particular will aggregate.
2-Calibration solution preparation:
External calibrator for chromium was prepared by serial dilution of parent stock (1000 µg/ml) using the diluents as those used to dilute and prepare the sample.
3-Optimization of technique:
For reading concentration of both sample and standard (calibrator), first it was important to choose proper wave length, lamp current band pass optimization.
4-Calculation of the results:
By plotting standard curve, the reading of absorbance of sample and calibrator was plotted in semi log curve; the concentration of Cr in sample was interpreted from this standard curve.
Whole blood chromium concentrations are in the range of 10 to 30 µg/L Chromium rapidly clears from the blood, and measurements relate only to recent exposure **(Goyer and Clarkson, 2001).** Urinary chromium values are typically less than 10 µg/g creatinine (**Goyer and Clarkson, 2001).**

Statistical Methods

Statistical analysis was performed using computer statistical software package SPSS 9.02. Descriptive statistics was presented as mean $\pm$ standard deviation. Comparative analysis between different groups was applied using student's t test for parametric data and Wilcox on sum of rank for skewed data. To study the relationship between two quantitative variables Pearson's correlation coefficient (r) was calculated, P-value is consider significant if < 0.05.

RESULTS AND DISCUSSION

Chromium is a metal. It exists in several different forms: divalent, trivalent, and hexavalent. Workers in many different occupations are exposed to hexavalent chromium. Occupational exposures to hexavalent chromium occur mainly among workers who: a) handle dry chromate-containing pigments; b) spray chromate-containing paints and coatings; c) operate chrome plating baths; and d) weld or cut chromium-containing metals such as stainless steel **(Cohen and Costa 1998)**.

In welding or cutting, the intense heat of the arc or flame vaporizes the base metal and/or the electrode coating. This vaporized metal condenses into tiny particles called fumes. These fume particles can be inhaled. Chromium fume is created by welding or cutting on stainless steel or metals that are coated with a chromium material. Welding on stainless steel without adequate control measures can lead to exposure at least several times above the legal exposure limit. http://www.bls.gov/oco/ocos226.htm.

The results of this study showed a statistically significant difference between the exposed and the control groups in the spirometric parameters indicating obstructing air-ways such as FEV_1/FVC, PEF%, and MEF 25-75% as shown in (table1), and these results are in accordance with **(Frantzen 1998)** who said that the primary target organ for subchronic and chronic chromium toxicity is the respiratory system as evidenced by various signs and symptoms ranging from irritation of the respiratory tract, obstructive air ways to perforation of nasal septum**.**

Also the study illustrated that, there is a statistically significant difference between the exposed and the control groups in the levels of chromium in blood and urine even these levels did not exceed the normal

range, but still higher than the control group. There are no routine medical tests to measure the amount of hexavalent chromium that has been absorbed into the body. Excreted chromium can be measured in urine. However, this test is only useful for measuring recent exposure to stainless steel welding fumes. In most situations, air monitoring gives the best measure of worker exposure **(ATSDR, 2000)**. **Huvinen and his colleagues 1996** conducted a cross sectional study to determine whether occupational exposure to hexavalent chromium caused respiratory diseases, decreases in pulmonary function, or signs of pneumoconiosis in stainless steel production workers and they found that with high levels of blood and urine chromium there was a great insult on the pulmonary functions and these results are in agreement with our results.

This result showed no statistically significant difference between both groups as regards the blood sugar level, but there was a statistically significant difference as regards the level of beta-$_2$ microglobulin in urine. These results can be illustrated by the fact that glomerular injury has been noted in chromium workers, but the predominant renal injury is tubular, with low doses acting specifically on the proximal convoluted tubules. Low-dose, chronic chromium exposure typically results only in transient renal effects. Elevated urinary ß$_2$-microglobulin levels (an indicator of renal tubular damage) have been found in chrome platters, and higher levels have generally been observed in younger persons exposed to higher Cr (VI) concentrations **(Powers et al., 1986). Liu and his colleagues 1993** found a statistically significantly higher urinary β_2-microglobulin level in hard-chrome electroplaters exposed to 0.0042 mg chromium/m^3 for a mean of 5.8 years and these results are in accordance with our results. We also showed a statistically significant difference between the exposed and the control groups as regards urea and creatinine levels, but **(Verschoor et al., 1988)** comparing renal function tests results in chrome platters and construction workers revealed that the chrome platers had significantly ($p<0.001$) higher levels of urinary chromium and they added that there were no differences in blood urea and creatinine, and urinary β_2-microglobulin and these results do not go with our results and this may be due to the fact that the difference between the exposed and the control groups in our work which is statistically significant is still in the normal range.

Table (1) Comparison between the exposed and control groups as regards different investigations

The studied groups / Parameters	Exposed group N:18		Control group N:20		t test	P value
	Mean	±SD	Mean	±SD		
FVC% of the predicted	78.5	± 5.9	83.9	± 5.9	0.11	> 0.05
SVC% of the predicted	79.83	± 6.2	83.9	± 5.9	0.7	> 0.05
FEV_1/FVC	71.83	± 6.2	79.6	± 4.3	4.7	**< 0.05**
PEF% of the predicted	61.6	± 3.5	72.8	± 4.1	8.8	**< 0.001**
MEF 25/75% of the predicted	61.1	± 8.3	75.5	± 4.6	6.6	**< 0.05**
Chromium in blood (Up to 30 ug/L)	13.9	± 2.6	4.8	± 0.7	14.8	**< 0.001**
Chromium in urine (< 10 ug/g creatinine)	8.6	± 0.58	3.2	± 0.7	24.7	**< 0.001**
B_2 microglobulin in urine (0-160 ug/L)	145.9	± 11.7	52.9	± 18.4	18.2	**< 0.001**
Urea (10-20 mg/dl)	20.9	± 3.8	16.7	± 1.1	4.6	**< 0.05**
Creatinine (0.7-1.2 mg/dl)	1.02	± 0.12	0.8	± 6.1	6.6	**< 0.05**
Fasting blood sugar (80-120 mg/dl)	125.4	± 9.1	117.2	± 14.72	2	> 0.05

Despite the fact that there is no statistically significant difference as regards smoking habit between exposed and controls group (p>0.05) tables (2&4) shows that the mean values of FVC% of predicted, SVC% of predicted and FEV_1/FVC among the exposed group were statistically significantly lower than those of the controls (p<0.05).

Our study is more or less in consistent with the results of **Shirakawa & Morimoto., 1996;** the irritant effect of chromium compounds can explain this pulmonary dysfunction.

Table (2) Smoking habit among the examined group

Studied group	Smokers		Non smokers		Total
	N	%	N	%	
Exposed	11	61.1	7	38.9	18
Control	13	65	7	35	20
Total	24		14		38
X^2	0.06				
P value	> 0.05				

Our study illustrated a statistically significant difference between the exposed and the control groups as regards the occurrence of metal fume fever as shown in table (3). The Agency for Toxic Substances and Disease Registry **ATSDR., 2005** reported that inhaling large amounts of metal or metal oxides (as zinc dust or fumes from smelting or welding as chromium oxides) can cause a specific short-term disease called metal fume fever, which is generally reversible once exposure to these fumes ceases. The effects of inhalation exposure to chromium compounds vary somewhat with the chemical form of the chromium compound, but the majority of the effects seen will occur within the respiratory tract. The most commonly reported effect is the development of "metal fume fever. The term "metal-fume fever" MFF describes an acute industrial illness characterized by a variety of symptoms, including fever, chills, dyspnea, muscle soreness, nausea and fatigue, which occur in workers following the inhalation of finely dispersed particulate matter formed when certain metals are volatilized. The oxides of a number of metals, including zinc and chromium, can cause this acute, reversible syndrome. The incidence of MFF is in accordance with our results shown in table (3). By filling the questionnaire for the studied groups, we found a statistically significant difference between the exposed and the control groups as regards experiencing MFF as shown in table (3).

Table (3) Occurrence of metal fume fever among the examined group

Studied group	**Metal fume fever**		**No metal fume fever**		**Total**
	N	**%**	**N**	**%**	
Exposed	8	44.44	10	55.55	18
Control	0	0	20	100	20
Total	8		30		38
X^2	11.2				
P value	**< 0.05**				

The following table (4) illustrated that there was no statistically significant difference between smokers and non-smokers among the exposed group as regards blood and urine chromium levels, urea and creatinine levels, B_2 microglobulin level and fasting blood sugar level. In agreement with our results **Halasova et al., 2005** found that in exposed group to chromium there was no significant effect of smoking on blood or urine level of chromium and they concluded, that occupational exposure to chromium was identified as the main risk factor of lung cancer even overlaying effect of smoking. As regards the renal insult due to smoking, our results showed no statistically significant difference between smokers and non smokers and this also reported by **Mortada et al., 2004** who measured serum levels of creatinine, B_2 microglobulin and blood urea nitrogen (BUN) to assess glomerular filtration among smokers and non smokers and they found that the Cd level in blood and Pb level in blood and hair were significantly higher in the smokers than non-smokers. Blood levels of Cd and Pb correlated significantly with the smoking index (an indicator for the degree of smoking) in the smokers group. The studied markers of kidney damage neither elevated among the smokers nor correlated with the exposure indices of these metals. They concluded that smokers are exposed to Cd and Pb and this exposure is not high enough in smoking to produce nephrotoxicity.

We noticed also in table (4) that there was no significant difference between smokers and non-smokers as regards fasting blood sugar level. **Uchimoto and his colleagues., 1999** are in accordance with our work as they found that in their research that cigarette smoking habit is an independent risk factor for type 2 D.M and it has no role in elevating the blood sugar level.

Table (4) Effect of smoking among the exposed group on different investigations

The exposed group / Parameters	Smokers N:11		Non-Smokers N:7		t test	P value
	Mean	±SD	Mean	±SD		
FVC% of the predicted	75.18	± 4.09	83.71	± 4.3	4.1	**< 0.05**
SVC% of the predicted	76.45	± 4.08	85.14	± 5.4	3.8	**< 0.05**
FEV_1/ FVC	70.36	± 4.8	74.42	± 6.2	2.8	**< 0.05**
PEF% of the predicted	60.72	± 3.4	63.14	± 3.2	1.45	> 0.05
MEF 25/75% of the predicted	60.27	± 7.5	62.57	± 9.9	1.46	> 0.05
Chromium in blood (Up to 30 ug/L)	13.55	± 2	14.5	± 3.4	0.79	> 0.05
Chromium in urine (< 10 ug/g creatinine)	8.6	± 0.48	8.5	± 0.75	0.000	> 0.05
B_2 microglobulin in urine (0-160 ug/L)	146.14	± 14.1	145.64	± 7.4	0.086	> 0.05
Urea (10-20 mg/dl)	20.8	± 3.6	21.2	± 4.3	0.18	> 0.05
Creatinine (0.7-1.2 mg/dl)	1.04	± 0.11	1	± 0.14	0.76	> 0.05
Fasting blood sugar (80-120 mg/dl)	124.27	± 7.07	127.28	± 12.24	0.6	> 0.05

From the results of our research we can concluded that there was no statistically correlation between the duration of exposure to chromium in welding and different investigations done, but these results showed also that the more the exposure duration the more affection of the pulmonary functions even this relation does not reach the level of significance as shown in table (5). Workers exposed to mean concentrations of 0.002–0.02 mg chromium (VI)/m^3 had slight, transient decreases in forced vital capacity (FVC), forced expired volume in 1 second (FEV_1), and forced mid-expiratory flow during the workday. Workers exposed to <0.002 mg chromium (VI)/m^3 showed no effects on lung function and these effects has no relation with the duration of exposure to chromium. Exposure to vapors of chromium salts has been suspected as a cause of asthma, coughing, wheezing, and other forms of respiratory distress in ferrochromium and welders workers **Lindberg & Hedenstierna., 1983**. These above results are in accordance with our results.

Table (5) in our work showed also that there was no statistically correlation between the duration of exposure to chromium and the levels of chromium in blood and urine in the exposed group and this was

also illustrated by **Goyer and Clarkson., 2001** who found that chromium rapidly clears from the blood, and measurements relate only to recent exposure**,** and they said also that urinary chromium excretion reflects absorption over the previous 1 or 2 days only. If sufficient time has elapsed for urinary clearance, a negative biomonitoring result can occur even with injurious past exposure.

The duration of exposure to any substance should be studied in any research, here in our work table 5 illustrated positive correlation between duration of exposure to chromium and renal affection, but this relation did not reach the level of significance, and this may be explained by the affection of the kidney depends on the amount of exposure not the duration or may be explained by that the number of workers here in our study was small and the variation of the duration of exposure could not be studied well and this could be explained by the fact that chromium has no cumulative effect in the body.

In contrast to our findings **Mattamara and Leong., 2004** demonstrated that blood and urinary levels of chromium among workers were associated with increasing duration of exposure.

Table (5) Correlation coefficient between duration of exposure to chromium in welding and different investigations

Parameters	R	F	P value
FVC%	-425	0.79	> 0.05
SVC%	-0.401	0.09	> 0.05
FEV_1/FVC%	-0.09	0.97	> 0.05
PEF%	0.023	0.928	> 0.05
MEF 25-75%	-0.12	0.05	> 0.05
Chromium in blood	0.52	0.02	> 0.05
Chromium in urine	0.03	0.88	> 0.05
B_2 microglobulin in urine	0.028	0.24	> 0.05
Urea	0.173	0.49	> 0.05
Creatinine	0.27	0.26	> 0.05

Our study revealed that there was a statistically significant correlation between the level of chromium in blood and its level in urine as shown in (table 6). Our findings are in agreement with **Shouman et al., 1999,** however it didn't agree with **Kornhauser et al., 2002**, this could be due to higher exposure to chromium in our environment than in Kornhauser's study which was in tanning process. **Minoia and Cavalleri., 1988** reported also that an analysis of the urine did not detect the hexavalent form of chromium, indicating that chromium (VI) was rapidly reduced before excretion.

We found a statistically significant correlation between the level of blood chromium and renal insult represented by the affection of urinary $beta_2$ microglobulin, urea, and creatinine. **Powers and his colleagues., 1986** said that glomerular injury has been noted in chromium workers, the predominant renal injury is tubular, with low doses acting specifically on the proximal convoluted tubules and this injury is correlated with the blood chromium level as low-dose, chronic chromium exposure typically results only in transient renal effects. They also reported that elevated urinary B_2-microglobulin levels (an indicator of renal tubular damage) have been found in chrome platters, and higher levels have generally been observed in younger persons exposed to higher Cr (VI) concentrations. However, they suggested urinary threshold for nephrotoxic effects is 15-µg chromium/g Creatinine**.** Powers and his colleagues' findings are in accordance with our results obtained in table (6) except the point, that urinary threshold for nephrotoxic

effects is 15 µg chromium/g Creatinine as in our results the urinary chromium level was around 8.6 ± 0.48. This may be explained by the fact that here in Egypt, The kidneys are more susceptible to diseases than other population due to the endemic urinary Bilharziasis.

The statistically significant correlation between the level of blood chromium and the reduction of FEV_1/FVC and MEF25-75% was illustrated in our work in table (6) and these results are in accordance with the results obtained by **Nordberg., 1998** who reported that the occurrence of bronchospasm in persons working with chromates suggests chemical irritation of the lungs and this bronchospasm was related to chromium exposure and its level in blood.

Table (6) Correlation coefficient between the levels of chromium in blood and different parameters

Parameters	R	F	P value
FEV_1/FVC%	-0.488	0.002	**<0.05**
MEF 25-75%	-0.764	0.00	**<0.001**
Chromium in urine	0.910	0.00	**<0.001**
B_2 microglobulin in urine	0.867	0.00	**<0.001**
Urea	0.77	0.00	**<0.001**
Creatinine	0.671	0.00	**<0.05**

CONCLUSION & RECOMMENDATIONS

From the present study, the primary target organ for subchronic and chronic chromium toxicity is the respiratory system, with high levels of blood and urine chromium there was a great insult on the pulmonary functions. Predominant renal injury among welders is tubular, and this injury is correlated with the blood chromium level, urinary ß$_2$-microglobulin level could be used as early marker for renal affection. We recommend environmental monitoring in different areas in the welding shops to ensure that permissible levels of different hazardous agents are not exceeded. Pre-employment and periodic medical examinations must be performed to exclude those susceptible to lung and renal diseases. Personal protective clothes e.g. gloves and respiratory protective equipment should be used in welders. Health education programs of workers about hazards of contact with harmful agents and proper measures for protection are essential. Effective local exhaust ventilation to remove hazardous gases and vapors is essential.

REFERENCES

Agency for Toxic Substances and Disease Registry (ATSDR)(2005): Toxicological Profile for Zinc (Update). Atlanta, GA: U.S. Department of Public Health and Human Services, Public Health Service.

ATSDR (2000): Cases studies in Environmental Medicine. Chromium toxicity. Course: SS3048. *http://www.atsdr.cdc.gov/HEC/CSEM/chromium.*

Bureau of Labor Statistics, U.S. Department of Labor, *Occupational Outlook Handbook, 2006-07 Edition*, Welding, Soldering, and Brazing Workers, on the Internet at http://www.bls.gov/oco/ocos226.htm.

Cohen MD and Costa M (1998): Chromium Compounds, Chapter (74), in: William N Rom ed. Environmental and Occupational Medicine, 3rd edition. Lippincott Raven Publishers, Philadelphia. p. 1045-55.

Frantzen K (1998): Chromium, Chapter (12), in: Harbison RD, ed. Hamilton &Hardy´s Industrial Toxicology, 5th ed. New York, London Sydney, Tokyo, Toronto. p. 51-54.

Goyer RA and Clarkson TW (2001): Chromium. Toxic Effects of Metals, Chapter (23), in: Klaassen CD, ed. Casarett and Doull´s: The Basic Science of Poisons, 6th edition. McGraw-Hill. New York, London Sydney, Tokyo, Toronto. p. 826-27.

Halasova E, Baska T, Kukura F,Mazurova D, Bukovska E, Dobrota D, Poliacek I, and Halasa M (2005): Lung cancer in relation to occupational and environmental chromium exposure and smoking. In Neoplasm; 52 (4):287-91.

HenneV, Frei P, and Burgisser P L(1997):-"$Beta_2$-microglobulin-A Rapid and Automated Determination for a Broad Range of Clinical Applications." Anticancer Research: 2915-2918.

Huvinen M, Uitti J and Zitting A (1996): Respiratory health of workers exposed to low levels of chromium in stainless steel production. Occup. Environ. Med. 53:741-747.

Henry J B (1996):- Clinical diagnosis and management by laboratory methods 19th ed. Philadelphia: W.B Saunders CO.

Kornhauser C, Wrobel K, Malacara JM, Nava LE, Gomez L and Gonzalez R (2002): Possible adverse effects of chromium in occupational exposure of tannery workers. Ind. Health. 40: 207-13.

Liu CS, Kuo HW and Lai JS (1993): Urinary N-acetyl-B-glucosaminidase as an indicator of renal dysfunction in electroplating workers. Int. Arch. Occup. Environ. Health 71:348-352.

Minoia C and Cavalleri A (1988): Chromium in urine, serum and red blood cells in the biological monitoring of workers exposed to different chromium valency states. Sci. Total Environ. 71:323-327.

Mortada WI, **Sobh MA**, and **El-Defrawy MM** (2004): The exposure of cadmium, lead and mercury from smoking and its impact on renal integrity. In: Med. Sci. Monit. ; 10 (3): CR 112-6. Urology and Nephrology Center, Mansoura University, Mansoura, Egypt.

Muttamara S, and Leong ST (2004): Health implication among occupational exposed workers in a chromium alloy factory, Thailand. In: J. Environ. Sci. (China) ; 16 (2): 181-6.

Nordberg G (1998): Chromium. Metals: Chemical properties and toxicity. In: Stellman J Mager Ed. Encyclopadedia of Occupational Health and Safety. 4th edition. Vol. (3) International Labour Office. Geneva. p.63.11-14.

Philip, Platcow G S and Lyndon (2004) : Welding and thermal cutting. This article is a revision of the 3rd edition of the Encyclopedia of Occupational Health and Safety article “Welding and thermal cutting” by G.S. Lyndon.

Powers WJ, Gad SC, Siino KM and Pechman JC (1986): Effects of therapeutic agents on chromium-induced acute nephrotoxicity, in: Serrone DM, ed. Chromium symposium: an update. Pittsburgh (PA): Industrial Health Foundation, Inc. p. 79-86.

Shirakawa T and Morimoto K (1996): Brief reversible bronchospasm resulting from bichromate exposure. Arch. Environ. Health 51(3): 221-25.

Shouman AE, Abdel Karim AH, Gadallah MA, Al- Ansary MS and Kalil SB (1999): Health hazards among workers of tanning. Egypt. J. Comm. Med. 17 (1):17-23.

Uchimoto S, **Tsumura K**, **Hayashi T**, **Suematsu C**, **Endo G**, **Fujii S**, and **Okada K** (1999): Impact of cigarette smoking on the incidence of type 2 diabetes mellitus in middle- aged Japanese men : the Osaka Health Survey . In: Diabet. Med. ; 16 (11):951-5. Second Department of Internal Medicine, Osaka City University Medical School, Osaka, Japan.

Verschoor MA, Bragt PC and Herber RF (1988): Renal function of chrome-plating workers and welders. Int. Arch. Occup. Environ. Health 60:67-70.

Vicki B (2003): Welding fume health hazards in Journal of Critical Reviews in Toxicology, 33 (1): 61-103.