

The Relationships between Mental Health Status, Achievement Motivation, Job Satisfaction and Oxidative Stress in Subjects Exposed to Nickel Welding Fumes

Ali Akbar Malekirad

Ph.D. in Physiology and Cognitive Neuroscience, Payame Noor University, Iran

Ahmad Goodarzi

Ph.D student in Management, University of Applied Science and Technology, Arak

Ahmad Reza Tohidnejad

BAin Professional Health, Hepco Company, Arak

Tahereh Sadeghi

MS student in Animal Physiology, Payame Noor University, Iran

Melika Heydari

MS student in Animal Physiology, Payame Noor University, Iran

Najmeh Egani

MS in Animal Physiology, Payame Noor University, Iran

Ahmad Akbari (corresponding author)

General Physician, University of Medical Science, Arak, Iran

dr.akbari49@yahoo.com

Abstract

This study was conducted on 27 male welding workers who worked in a company that manufactures road construction machinery and is located in an industrial part of Iran, in the Central province. In the serum, total antioxidant capacity (TAC), lipid peroxidation (LPO), DNA damage (8-OH-dG) was measured. Mental health status was evaluated by General Health Questionnaire (GHQ-28) and achievement motivation was evaluated by Herman's achievement motivation Test (ACMT), and job satisfaction was measured by **Dennett** Job Satisfaction Test. The results of this study showed that there was a reverse significant relationship between nickel and TAC with ACMT and job satisfaction. The oxidative stress of Ni ended up in psychological disorders. As oxidative stress causes psychological disorders, the welding workers suffered from more psychopathology and were in need of greater care.

Key Words: Nickel Welding Fumes, mental health status, achievement motivation, job satisfaction, oxidative stress

Introduction

By generating reactive oxygen and nitrogen species, metal-induced toxicity and carcinogenicity, make a lot of different changes to DNA bases, and enhance lipid peroxidation, and alter calcium and sulfhydryl homeostasis. Lipid peroxides, which are the result of the attack of radicals on polyunsaturated fatty acid residues of phospholipids, can react with redox metals which may lead into mutagenic and carcinogenic malondialdehyde, 4-hydroxynonenal and other exocyclic DNA adducts (etheno and/or propano adducts). While iron (Fe), copper (Cu), chromium (Cr), vanadium (V) and cobalt (Co) go through redox-cycling reactions, for a second group of metals like mercury (Hg), cadmium (Cd) and nickel (Ni), the primary route for their toxicity is attenuation of glutathione and bonding to sulfhydryl groups of proteins (Valko, & Cronin, 2005; Malekirad et al., 2010; Malekirad et al., 2012; Shariatzade and Malekirad, 2007; Kim and Seo, 2012). Ni and Co are heavy metals found

in land, water, and air and can be absorbed by the body chiefly through the breathing system and pile up to a dangerous toxic amount. We know that Nickel compounds are carcinogenic to both humans and animals. Co compounds make tumors in animals and may be a risk factor for cancer for humans. People who work in industries are highly in danger of being exposed to these harmful metals (Patel et al., 2012). It is widely accepted that many heavy metals, including Ni, Cd, and Cr are toxic industrial chemicals that can have hazardous outcomes, for those who are exposed to them either in their working sites or in their environment (Permenter, Lewis, & Jackson, 2011). When metal ion homeostasis is disrupted, oxidative stress may be the result which is a state in which excessive reactive oxygen species (ROS) overwhelms the body's antioxidant protection and consequently DNA damage, lipid peroxidation, protein modification and some other effects may happen. These are all symptoms of several diseases like cancer, cardiovascular diseases, diabetes, atherosclerosis, neurological disorders (Parkinson 'disease, Alzheimer's disease), and chronic inflammation (Jomova and Valko, 2011; Kubrak et al., 2012; Malekirad et al., 2010; Fazli et al., 2012; Ranjbar et al., 2007; Koedrith and Seo, 2011). Heavy metals produce free radicals and cause oxidative and nitrosative stress with depletion of antioxidants. Ni sulfate treatment causes testicular oxidative and nitrosative stress in albino rats (Jargar et al., 2012). It is thought that the ability of metals like Ni, arsenic, and Cr to produce oxidative stress is partially the cause of their carcinogenic effects. The pervasiveness of oxidative stress in biological systems has made it relatively obvious responsible for cellular damage and/or development of disease. However, the oxidative stress-induced damage is not confined to its direct effects on cellular components, like lipids, proteins, and DNA, it can extend to the potentiality to change gene expression, as well (Chervona and Costa, 2012). Besides, Ni exposure from metal inert gas (MIG) and tungsten inert gas (TIG) welding is related to work-related respiratory symptoms (Fishwick et al., 2004). Furthermore, another study has documented a high pervasiveness of symptoms of chronic bronchitis and other work-related respiratory symptoms in welders (Bradshaw et al., 1998). Also, other findings propose that limited and obstructive lung abnormalities, and airway irritation symptoms are associated with spot and arc welding exposures (Luo, Hsu & Shen, 2006). There are various elements that can influence the pathological depositions, and generally, the reason for neuronal fatality in neurological disorders seems to be multifactorial. Yet, the underlying factor in the neurological disorders is now known to be the increased oxidative stress, which is backed up by the findings that the protein side-chains are modified either directly by reactive oxygen species (ROS) and reactive nitrogen species (RNS), or indirectly, by the products of lipid peroxidation (Jomova et al., 2010). With these facts in mind, the present study was designed to examine the relationship between psychological state and oxidative markers in welding workers who are exposed to Ni.

Study population

The study was conducted on 27 male workers, with the age range of 27-50, who worked in a company located in an industrial part of Iran, in the Central province. All participants were provided with specific written specific information about the aims of the study before written consents were obtained, in accordance with ethical rules of Pharmaceutical Sciences Research Center (PSRC) of Tehran University of Medical Science where the study protocol was approved. Prior to blood collection, each individual was extensively interviewed by a specialized physician who filled in a structured questionnaire about disease and habit diet. Information on occupational history, socioeconomic status (salary, education), and lifestyle information (smoking, alcohol consumption, drug uses, consumption of vitamin or antioxidant supplements, and dietary habits) were obtained from questionnaires and interviews completed by each worker, with a trained interviewer. The Iranian version

of the General Health Questionnaire (GHQ28) was used to identify high risk subjects for mental disorder, as well as the following four symptoms: "somatic symptoms", "anxiety and insomnia", "social dysfunction", and "severe depression (Ebrahimi et al., 2008) and Iranian version of Achievement Motivation Test (ACMT) and Job satisfaction were used to evaluate achievement motivation and job satisfaction (Hoomon and Asgari, 2001; Shahrabi Farahani, Farahbakhsh & Asghari, 2012). All subjects were submitted to complete clinical examination to detect any signs or symptoms of chronic diseases such as arterial hypertension, heart failure, cancer, thyroid disturbance, asthma, diabetes, and anemia. Individuals with chronic disease, alcohol consumption, antioxidant consumption, and/or under drug treatment, or exposure to other toxic materials, radiation therapy, or substance abuse were excluded from the study. Blood samples were collected from included subjects between 7 and 8 a.m. before entering the workplace on Saturday as the beginning of the weekly work shift. Blood samples were collected into heparinized tubes and immediately centrifuged at 3000g for 10 min and the plasma was separated and frozen at -80°C for further analysis of oxidative stress markers.

Assay of oxidative stress markers: Ni

The activities of 8-hydroxy-2-deoxy guanosine (8-OH-dG) as a measure of deoxyribonucleic acid (DNA) damage were assayed using ELISA kits. The basis of determination of this parameter in the kits was as follows: 8-Hydroxy-2-deoxy guanosine (8-OH-dG) kit was assayed using an anti-mouse IgG-coated plate and a tracer consisting of an 8-OH-dG-enzyme conjugate and 8-OH-dG antibody recognizing both free 8-OH-dG and DNA-incorporated 8-OH-dG (Bayrami et al., 2012). To measure the rate of lipid peroxidation (LPO), thiobarbituric acid TBA method was used (Esterbauer and Cheeseman, 1990). The basis of measurement of total antioxidant capacity (TAC) was to analyze the ability of plasma in reducing Fe^{3+} to Fe^{2+} in the presence of TPTZ. Fe^{2+} -TPTZ as a blue complex is absorbed at 593 nm (Benzi and Strain, 1999). Ni in plasma samples was measured by graphite-furnace atomic absorption spectroscopy.

Statistical Analysis

Results are presented as Mean \pm SD. Statistical analyses were conducted using Stats Direct 2.7.8 software. Relationships between parameters were determined by use of canonical correlation analysis. P-value of less than 0.05 was considered statistically significant.

Results and Discussion

Table 1 shows the average levels of subject's age, duration of employment, TAC, LPO, 8-OH-dG, GHQ, ACMT and Job Satisfaction.

Table 2 presents the results of the canonical correlation analysis. This analysis was used to determine the degree to which the oxidative stress markers were related to the Psychological variable. The canonical analysis revealed that only the third root canonical correlations were statistically significant ($p < .05$). Indeed, the third canonical correlation ($R_{c1} = 0.36$) was statistically significant.

Data pertaining to the third canonical root are presented in Table 2. This table provides both standardized function coefficients and structure coefficients. The standardized canonical function coefficients revealed that, using a cutoff correlation of 0.3 recommended as an acceptable minimum coefficient value, TAC and Nickel made significant contributions to the Career motivation and Career satisfaction.

The structure coefficients (Table 2) indicated that both 8-OH-dG and TAC made important contributions to the Career motivation. The square of the structure coefficient (Table 2) indicated that TAC made a very large contribution, explaining 0.76.

Comparing the standardized and structure coefficients showed that 8-OH-dG and LPO appeared to be collinear with at least one of the other variables because their standardized coefficients were small.

Table 1. Plasma oxidative stress markers, psychological parameters and demographic data in workers

Parameters	mean	SD
TAC(mm ^{ol} mL ⁻¹)	318.45	72.81
LPO(mm ^{ol} mL ⁻¹)	18.20	2.87
8-OH-dG(Pg/mL)	393.62	82.57
GHQ28	18.18	9.74
ACMT	21.36	6.69
Job Satisfaction	161.88	45.98
Age	40.07	7.91
Worker History	12.13	7.94

Values are as Mean±SD

Table 2. Canonical solution for third function: relationship between oxidative stress markers and psychological variable

		Variable	Standardized Coefficient	Structure Coefficient
Oxidative stress markers		OHdG	-0.24	-0.40*
		LPO	0.17	0.13
		TAC	-0.95*	0.86*
		Nickel	-0.47*	-0.11
Psychological variable		GHQ	-0.18	-0.19
		Career motivation	-0.98*	-0.90*
		Career satisfaction	0.32*	-0.22
coefficients with effect sizes larger than .3 *				

As the results of present study show Ni and TAC with ACMT and job satisfaction have a reverse relationship. It seems that the piling up of Ni in the workers induces free radicals and oxidative stress and following those TAC increases because of its contrast with oxidative stress. The pres-

ence of pollution and its absorption into body was traced in the plasma of involved workers. The main reason for this increase in plasma level may be the poor and unsuitable protection tools.

When the workers were interviewed and the industry was inspected, it became clear that workers were not trained enough to use working clothes and gloves as well as wearing masks or taking regular shower. There were no proper washrooms and even standard masks. However, the workers were more willing to use clothes, gloves, and shoes.

There was also a negative significance between Ni and TAC with ACMT and job satisfaction. The oxidative stress ratio was significantly higher in the welding exposed workers of control groups (du Plessis et al., 2010; Imamoglu et al., 2008; Han et al., 2005). Also, in comparison to control, in Ni-platers the level of plasma lipid peroxidation was significantly high and erythrocyte antioxidants were significantly low. The level of plasma lipid peroxidation was positively and erythrocyte antioxidants were negatively significantly correlated with the urine Ni levels. (Kalahasthi et al., 2006). Another study reported that there were significant relationship between urine metals levels and the length of welding work; on the other hand, Ni was significantly inversely related to GSH (Luo, Hsu, & Shen, 2009). Mukherjee et al. (2004) reported that Ni, V, Cr, and Cu were significantly associated with the 8-OH-dG level (Mukherjee et al., 2004). Xing et al. (2012) reported that theory of reasoned action is suitable for welding worker occupational health related behaviors (Xing et al., 2012). Moreover, another study showed that welders and controls performed similarly on tests of verbal skills, verbal retention, and auditory span, welders performed worse than controls on tests of verbal learning, working memory, cognitive flexibility, visuomotor processing speed, and motor efficiency. Welders had poorer color vision and emotional status, and increased prevalence of illnesses and psychiatric symptoms. The increased symptoms in welders were related to decreased scores on tasks measuring verbal learning, visuomotor abilities, visuospatial abilities, and information processing, and motor efficiency (Bowler et al., 2003). Michel et al. (2012) found correlations of lower oxidative stress with antidepressant treatment and clinical outcome measures (Michel et al., 2012). Another study suggested that the increase of antioxidant defenses may be one of the mechanisms underlying the neuroprotective effects of antidepressants in the treatment of Major depressive disorder (Behr et al., 2012). Another study showed that people who suffered mild cognitive impairment (MCI) had increased brain oxidative damage before the onset of symptomatic dementia (Praticò et al., 2002). Racette et al. (2005, 2012) reported that welders have a high prevalence of Parkinsonism compared to nonwelding-exposed workers (Racette et al., 2012; Racette et al., 2005), while results from other studies do not support an association between welding occupations and death from Parkinson's disease or other neurodegenerative diseases (Stampfer, 2009; Kenborg et al., 2012; Fryzek et al., 2005; Fored et al., 2006).

Conclusion

The oxidative stress of Ni ends up in psychological disorders. As oxidative stress causes psychological disorders, the welding worker suffered from more psychopathology and were in need of greater care. However, providing workers with proper protective tools and training and encouraging them to use those tools, together with taking daily shower can definitely reduce the risk of exposure to toxic elements and may prevent them from entering the workers' bodies.

Acknowledgment

The authors would like to thank all subjects and authorities of Hepco Company for their kind co-operation. This study is funded by Payame Noor University (PNU).

Reference

- Bayrami, M., Hashemi, T., Malekirad, A.A., Ashayeri, H., Faraji, F., Abdollahi, M., 2012. Electroencephalogram, cognitive state, psychological disorders, clinical symptom, and oxidative stress in horticulture farmers exposed to organophosphate pesticides, *Toxicology and Industrial Health*, 28(1):90-96.
- Benzi, I.F. & Strain S., 1999. Ferric reducing antioxidant assay. *Methods in Enzymology*, 292: 15-27.
- Bowler, R.M., Gysens, S., Diamond, E., Booty, A., Hartney, C., & Roels, H.A., 2003. Neuropsychological sequelae of exposure to welding fumes in a group of occupationally exposed men. *International Journal of Hygiene and Environmental Health*. 206(6):517-529.
- Bradshaw, L.M., Fishwick, D., Slater, T., Pearce, N., 1998. Chronic bronchitis, work related respiratory symptoms, and pulmonary function in welders in New Zealand. *Occupational and Environmental Medicine*, 55(3):150-4.
- Calderón-Garcidueñas, L., Maronpot, R.R., Torres-Jardon, R., Henríquez-Roldán, C., Schoonhoven, R., Acuña-Ayala, H., Villarreal-Calderón, A., Nakamura, J., Fernando, R., Reed, W., Azzarelli, B. & Swenberg, J.A., 2003. DNA damage in nasal and brain tissues of canines exposed to air pollutants is associated with evidence of chronic brain inflammation and neurodegeneration. *Toxicologic Pathology*, 31(5):524-38
- Calderón-Garcidueñas, L., Serrano-Sierra, A., Torres-Jardón, R., Zhu, H., Yuan, Y., Smith, D., Delgado-Chávez, R., Cross, J.V., Medina-Cortina, H., Kavanaugh, M. & Guilarte, T.R., 2012. The impact of environmental metals in young urbanites' brains. *Experimental and Toxicologic Pathology*. 2013 Jul;65(5):503-11. doi: 10.1016/j.etp.2012.02.006. Epub 2012 Mar 19.
- Chervona, Y., & Costa, M., 2012. The control of histone methylation and gene expression by oxidative stress, hypoxia, and metals. *Free Radical Biology and Medicine*, 3(5):1041-7. doi: 10.1016.
- Chuang, H.C., Hsueh, T.W., Chang, C.C., Hwang, J.S., Chuang, K.J., Yan, Y.H. & Cheng T.J., 2012. Nickel-regulated heart rate variability: The roles of oxidative stress and inflammation. *Toxicology and Applied Pharmacology*, 266(2):298-306. doi: 10.1016.
- du Plessis, L., Laubscher, P., Jooste, J., du Plessis, J., Franken, A., van Aarde, N., Eloff, F. 2010. Flow cytometric analysis of the oxidative status in human peripheral blood mononuclear cells of workers exposed to welding fumes. *Journal of Occupational and Environmental Hygiene*. 7(6):367-74. doi: 10.1080/15459621003724108.
- Ebrahimi, A., Molavi, H., Moosavi, G., Bornamanesh, A., & Yaghoobi, M., 2008. Psychometric Properties and Factor Structure of General Health Questionnaire 28 (GHQ-28) in Iranian Psychiatric Patients. *Journal of Behavior Science*. 5 (1):1-8.
- Esterabeur, H., & Cheeseman, K., 1990. Determination of aldehyds lipid peroxidation products: malondealdehyde and 4-hydroxyl nonenal. *Methods in Enzymology*, 186:407-421.
- Fazli, D., Malekirad, A.A., Pilehvarian, A.A., Salehi, H., Zerratishe, A., Rahzani, K. & Abdollahi, M., 2012. Effects of Melissa officinalis on oxidative status and biochemical parameters in occupationally exposed workers to Aluminum: A before after clinical trial. *International Journal of Pharmacology*. 8(5):455-458.
- Fishwick, D., Bradshaw, L., Slater, T., Curran, A., & Pearce, N., 2004. Respiratory symptoms and lung function change in welders: are they associated with workplace exposures? *New Zealand Medical Journal*, 7;117(1193):U872.
- Fryzek, J.P., Hansen, J., Cohen, S., Bonde, J.P., Llabias, M.T., Kolstad, H.A., Skytthe, A., Lipworth, L., Blot, W.J., & Olsen, J.H., 2005. A cohort study of Parkinson's disease and other neuro-

degenerative disorders in Danish welders. *Journal of Occupational & Environmental Medicine*, 47(5):466-72.

Gupta ,A.D., Patil, A.M., Ambekar, J.G., Das, S.N., Dhundasi,S.A.,&Das, K.K.,2006. L-ascorbic acid protects the antioxidant defense system in nickel-exposed albino rat lung tissue. *Journal of basic and clinical physiology and pharmacology*, 17(2):87-100.

Han, S.G., Kim, Y., Kashon, M.L., Pack, D.L., Castranova, V., &Vallyathan, V.,2005.Correlates of oxidative stress and free-radical activity in serum from asymptomatic shipyard welders. *American Journal of Respiratory and Critical Care Medicine*, 15;172(12):1541-8. Epub 2005 Sep 15.

Hoomon, H.A.,&Asgari, A.,2001.Developing and Standardization of Achievement Motivation Test (AMT).*Psychological Research* 1-2(11):79.

Imamoglu, N., Yerer, M.B., Donmez-Altuntas, H., &Saraymen, R.,2007.Erythrocyte antioxidant enzyme activities and lipid peroxidation in the erythrocyte membrane of stainless-steel welders exposed to welding fumes and gases. *International Journal of Hygiene and Environmental Health*,211(1-2):63-8.

Jargar, J.G., Yendigeri, S.M., Hattiwale, S.H., Dhundasi,S.A.,&Das, K.K.,2012.α-Tocopherol ameliorates nickel induced testicular oxidative and nitrosative stress in albino rats. *Journal of basic and clinical physiology and pharmacology*, 23(2):77-82.

Jomova, K., &Valko., M.,2011.Advances in metal-induced oxidative stress and human disease.*Toxicology*, 10;283(2-3):65-87.

Jomova, K., Vondrakova, D., Lawson, M.,&Valko, M.,2010.Metals, oxidative stress and neurodegenerative disorders. *Mol Cell Biochem*.345(1-2):91-104. doi: 10.1007

Kalahasthi, R.B., Hirehal Raghavendra Rao, R., & Bagalur Krishna Murthy, R.,2006. Plasma lipid peroxidation and erythrocyte antioxidants status in workers exposed to nickel. *Biomarkers*,11(3):241-9.

Kenborg, L., Lassen, C.F., Hansen, J.,&Olsen, J.H.,2012. Parkinson's disease and other neurodegenerative disorders among welders: a Danish cohort study. *Movement Disorders*, 1; 27(10):1283-9.

Kim, J.Y., Mukherjee ,S., Ngo, L.C., & Christiani, D.C.,2004. Urinary 8-hydroxy-2'-deoxyguanosine as a biomarker of oxidative DNA damage in workers exposed to fine particulates. *Environmental Health Perspectives*,112(6):666-71.

Kim, H.L., &Seo, Y.R.,2012.Molecular and genomic approach for understanding the gene-environment interaction between Nrf2 deficiency and carcinogenic nickel-induced DNA damage.*Oncology Reports*, 28(6):1959-67. doi: 10.3892/or.2012.2057. Epub 2012 Sep 26.

Koedrith, P., &Seo Y.R.,2011.Advances in carcinogenic metal toxicity and potential molecular markers.*International Journal of Molecular Sciences*,12(12):9576-95. doi: 10.3390.

Kubrak, O.I., Husak, V.V., Rovenko, B.M., Poigner, H., Mazepa, M.A., Kriews, M., Abele, D., &Lushchak,V.I.,2012. Tissue specificity in nickel uptake and induction of oxidative stress in kidney and spleen of goldfish *Carassius auratus*, exposed to waterborne nickel. *Aquatic Toxicology*,15;118-119:88-96. doi: 10.1016.

Lin, T.S., Wu, C.C., Wu, J.D., &Wei, C.H.,2012.Oxidative DNA damage estimated by urinary 8-hydroxy-2'-deoxyguanosine and arsenic in glass production workers.*Toxicology&Industrial Health*, 28(6):513-21. doi: 10.1177/0748233711416945.

Luo, J.C., Hsu, K.H.,&Shen,, W.S.,2009. Inflammatory responses and oxidative stress from metal fume exposure in automobile welders. *Journal of Occupational & Environmental Medicine*, 51(1):95-103. doi: 10.1097.

Luo, J.C., Hsu, K.H., & Shen, W.S., 2006. Pulmonary function abnormalities and airway irritation symptoms of metal fumes exposure on automobile spot welders. *American Journal of Industrial Medicine*, 49(6):407-16.

Malekirad, A.A., Mirabdollahi, M., Pilehvarian, A.A., Nassajpour, A.A., & Abdollahi M., 2013. Status of neurocognitive and oxidative stress conditions in iron-steel workers. *Toxicology Industrial Health* [Epub ahead of print]

Malekirad, A.A., Oryan, S., Fani, A., Babapor, V., Hashemi, M., Baeri, M., Bayrami, Z., & Abdollahi, M., 2010. Study on clinical and biochemical toxicity biomarkers in zinc-lead mine workers. *Toxicology and Industrial Health*, 26(6):331-337.

Mukherjee, S., Palmer, L.J., Kim, J.Y., Aeschliman, D.B., Houk, R.S., Woodin, M.A., & Christiani, D.C., 2004. Smoking status and occupational exposure affects oxidative DNA injury in boilermakers exposed to metal fume and residual oil fly ash. *Cancer Epidemiology, Biomarkers & Prevention*, 13(3):454-60.

Patel, E., Lynch, C., Ruff, V., & Reynolds, M., 2012. Co-exposure to nickel and cobalt chloride enhances cytotoxicity and oxidative stress in human lung epithelial cells. *Toxicology and Applied Pharmacology*, 1;258(3):367-75. doi: 10.1016.

Permenter, M.G., Lewis, J.A., & Jackson, D.A., 2011. Exposure to nickel, chromium, or cadmium causes distinct changes in the gene expression patterns of a rat liver derived cell line. *PLoS One*. 6(11):e27730. doi: 10.1371.

Racette, B.A., Criswell, S.R., Lundin, J.I., Hobson, A., Seixas, N., Kotzbauer, P.T., Evanoff, B.A., Perlmutter, J.S., Zhang, J., Sheppard, L., & Checkoway, H., 2012. Increased risk of parkinsonism associated with welding exposure. *Neurotoxicology*, 33(5):1356-61. doi: 10.1016/j.neuro.2012.08.011. Epub 2012 Sep 3.

Racette, B.A., Tabbal, S.D., Jennings, D., Good, L., Perlmutter, J.S., & Evanoff, B., 2005. Prevalence of parkinsonism and relationship to exposure in a large sample of Alabama welders. *Neurology*, 25;64(2):230-5.

Ranjbar, A., khani-jazani, K., Sedighi, A., Jalali- Mashayekhi, F., Ghazi-khansari, M., & Abdollahi, M., 2008. Alteration of body total antioxidant capacity and thiol molecules in human chronic exposure to aluminium. *Toxicological & Environmental Chemistry*, 90:707-713

Shahrabi Farahani, L., Farahbakhsh, K., & Asghari, M., 2012. The effectiveness of the counseling with choice theory of Glasser in women teachers successfully employment of Tehran 15 area. *Quarterly Journal of Career & Organizational Counseling*, 4(10):73-91.

Shariatzade, S.M.A., & Malekirad, A.A., 2008. Free Radicals and Antioxidants "Oxidative Stress" in Cadmium Exposure Workers. *Yakhteh Medical Journal*. 9(4): 276-279.

Stampfer, M.J., 2009. Welding occupations and mortality from Parkinson's disease and other neurodegenerative diseases among United States men, 1985-1999. *Journal of Occupational and Environmental Hygiene*, 6(5):267-72. doi: 10.1080/15459620902754703.

Sunderman, F.W. Jr., 1993. Biological monitoring of nickel in humans. *Scandinavian Journal of Work, Environment & Health*, 19 Suppl 1:34-8.

Valko, M., Morris, H., & Cronin, M.T., 2005. Metals, toxicity and oxidative stress. *Current Medicinal Chemistry*, 12(10):1161-208.

Yoshioka, N., Nakashima, H., Hosoda, K., Eitaki, Y., Shimada, N., & Omae, K., 2008. Urinary excretion of an oxidative stress marker, 8-hydroxyguanine (8-OH-Gua), among nickel-cadmium battery workers. *Journal of Occupational Health*. 50(3):229-35. Epub 2008 Apr 11.