Evaluation of Occupational Exposure of Glazers of a Ceramic Industry to Cobalt Blue Dye

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Abstract

Background: Cobalt is one of the most important constituent present in ceramic industries. Glazers are the relevant workers when they are producing blue colored ceramic, causing occupational exposure to such metal. Through this study, urinary cobalt was determined in glazers in a ceramic industry when they were producing blue-colored ceramic glazes.

Methods: In this case-control study, spot urine samples were collected from 49 glazers at the start and end of work shifts (totally 98 samples) in 2011. Control group were well matched for age, height, and weight. A solid phase extraction system was used for separation and preconcentration of samples followed by analysis by inductively coupled plasma-atomic emission spectroscopy (ICP-AES).

All participants filled out a self administered questionnaire comprises questions about duration of exposure, work shift, use of mask, skin dermatitis, kind of job, ventilation system, overtime work, age, weight, and height. The lung function tests were performed on each control and cobalt exposed subjects. Analysis of covariance (ANCOVA) was used to evaluate the obtained results.

Results: Urinary levels of cobalt were significantly higher in the glazers compared to the control group. There were significant differences at urinary concentration of cobalt at the start and end of the work shift in glazers. Spirometric parameters were significantly lower in the glazers compared to the control group. Among the variables used in questionnaire the significant variables were dermatitis skin, mask, ventilation, and overtime work.

Conclusion: This study verified existence of cobalt in the urine glazers, showing lower amount than the ACGIH standard.

Keywords: Urine cobalt, Glaze, Spirometry, Inductively coupled plasma-atomic emission, Spectroscopy

Introduction

Production of blue colored ceramic in glaze industry is of important concern for structuring of floors and walls. Cobalt ion plays a main role to produce blue color in ceramic industries. This is because cobalt has mobile 3d electrons in its atomic structure (1). The main component, such as silica, present in ceramic are similar to glass, causing both favorite melting point and color (2, 3). Water is added to glaze for producing a cream suspension solution. This solution then is applied to a clay body at increasing temperature up to 1300°C. Therefore, an interface layer is made up
by glaze and clay body interaction, then, glaze is adhered to the clay (2, 3). Finally, color is occurred on glaze in result of high temperature reaction. In such process, cobalt toxicity is taken place when glazers are exposed to the cobalt in powder form. To prevent adverse effect of cobalt, such as, gamut of respiratory disorders, asthma, and also lung cancer, occupational exposures are of great concern in the ceramic industries (2, 3).

Cobalt is not a cumulative toxicant and is mainly excreted in urine to a lesser extent via faces. Cobalt in blood and urine mainly shows the exposure already occurred. The two main target organs are the skin and the respiratory tract (4). Cobalt is the critical toxic component causing hard metal lung disease (5) that is a rare form of occupational lung disease that can occur in workers engaged in the manufacture, utilization, or maintenance of tools hard metal. Cobalt may cause allergic dermatitis (6-9), cardiomyopathy (10, 11) and asthma (7,12-15). Exposure to cobalt leads to severe alterations in capillaries, edema and hemorrhage of lung in experimental animals (16).

In hard metal industries such as cobalt, occupational asthma is characterized by wheezing cough, and contact dermatitis characterized by erythematous papules. The International Agency for Research on Cancer (IARC) recently has classified cobalt and cobalt compounds as possibly carcinogenic to humans (Class 2B) (17).

The objectives of this study were to determine the cobalt level in urine of glazers of a ceramic industry and also to find out whether a relationship exists between factors including duration of exposure, work shift breathing masks, skin dermatitis, overtime work, ventilation, age, weight, and height. There were not significant differences between glazers who exposed to cobalt and the referents concerning age, weight, and height. Urine samples were taken for determination of cobalt, at the start and end of the work (totally 98 samples). The referents were examined only once ignoring the shift type (55 samples).

Materials and Methods

This case-control study was carried out in glazing units of a ceramic industry where blue-colored ceramic glazes were produced. The study group was 49 glazers were working in several pottery and tile workshops. A control group consisting of 55 office workers was considered for comparison study. In glazers, age ranged from 22 to 50 years with a mean value of 30.67 years and for the control group it was 23 to 50 years with a mean value of 32.6 years. The reference group had never been occupationally exposed to cobalt. The work shift length was 8 hours, with two shifts per day. All participants filled out a self administered questionnaire comprises questions about overtime work, duration of exposure, work shift, use of mask, skin allergy, ventilation, age, weight, and height. There were not significant differences between glazers who exposed to cobalt and the referents concerning age, weight, and height. Urine samples were taken for determination of cobalt, at the start and end of the work (totally 98 samples). The referents were examined only once ignoring the shift type (55 samples).

Samples were collected before starting to work and at the end of the work shift while workers removed their work uniforms. They were advised to wash their hands before sampling in order to reduce the potential for contamination. Twenty milliliters urine was collected in acid-washed polyethylene containers and was stored at +4°C until the analyses time. A solid phase extraction procedure was used for separation and preconcentration of cobalt.

Experimental

Sample preparation

A solid phase extraction system was used for separation and preconcentration of cobalt. Cartridges were filled up with 500 mg amberlite XAD-7 resin. In order to remove organic and inorganic contaminants, amberlite XAD-7 adsorption resin was washed with ethanol, water, and 1 M HCl. 2.5 ml urine sample was adjusted at pH 9 and the cobalt present in the samples was chelated with ammonium pyrrolidine dithiocarbamate (APDC). Then,
samples were diluted to 25 ml with distilled water. The solution was then passed through the sorbent at a flow rate of 5 ml/min under gravity. The column was then washed out with water and 1 M NaOH. Then, the retained analyte was eluted by 15 ml of 2 M HNO₃.

**Analysis procedure**

The urinary cobalt was analyzed by inductively coupled plasma-atomic emission spectroscopy (ICP-AES) (SPECTRO, ARCOS, Germany). The wavelengths selected for Co was 228 nm. Standard solutions were prepared from stock solutions (1000 mg/l). The calibration of the ICP-AES was performed using standard solutions containing 200-1000 µg/l of cobalt.

The lung function tests were performed on each control and cobalt exposed subject by spirometer system (model 2120). The spirometer was calibrated daily with one liter calibration syringe and operated at temperature ranged from 20 to 25°C. Measurements were performed in a standing position. Tests were conducted according to the American Thoracic Society (ATS) recommendation. Through measurement, forced vital capacity (FVC), forced expiratory volume in one second (FEV1), forced expiratory flow FEF₂₅₋₇₅ were measured. The FEV1/FVC ratios were calculated in terms of percentage.

Statistical analysis was performed using the SPSS 11.5 software. Comparisons between control and glazers were made with independent samples t-tests. Paired t-tests were used to assess the changes between pre- and post-shift measurements for the glazers. Analysis of covariance (ANCOVA) was used to evaluate the effect of each factor on post-shift measurement after controlling for the pre-shift shift.

**Results**

Important demographic characteristics and the results of the lung function tests for glazers and their matched controls are shown in Table 1. Mean value for age, height, and weight was not significantly different between groups. Mean values of FVC, FEV1, FEV1/FVC, and FEF₂₅₋₇₅ were lower in the exposed group compared to the control group (P<0.001) (Table 1).

| Variables                  | Glazer (n = 49) | Control (n = 55) | P value |
|----------------------------|-----------------|------------------|--------|
| Age (yr)                   | 30.67 ± 5.88    | 32.6 ± 7.1       | 0.12   |
| Height (cm)                | 174.5 ± 8.6     | 176.42 ± 9.06    | 0.26   |
| Weight (kg)                | 77.44 ± 11.7    | 78.25 ± 10.6     | 0.74   |
| BMI                        | 25.59 ± 4.39    | 25.33 ± 4.29     | 0.76   |
| Overtime work (hour)       | 10.05 ± 14.9    | 0.83 ± 2.74      | <0.001 |
| Work history (month)       | 51.53 ± 33.44   | 76.32 ± 67.92    | 0.02   |
| FVC (liter)                | 4.24 ± 0.67     | 4.74 ± 0.66      | <0.001 |
| FEV1 (liter)               | 3.5 ± 0.7       | 4.25 ± 0.7       | <0.001 |
| FEV1/FVC (percent)         | 80.99 ± 8.16    | 88.85 ± 5.6      | <0.001 |
| FEF₂₅₋₇₅ (liter)           | 3.47 ± 0.8      | 4.49 ± 0.69      | <0.001 |

Table 1: Population characteristics of the study groups
Urinary level of cobalt at the start of the work shift was ranged from 0.01 to 5 µg/L, with a mean value of 1.73 µg/L and at the end of the work shift ranged from 0.01 to 15 µg/L, with a mean value of 3.47 µg/L. A significant difference was found between Co concentration of start and the end shift samples. There were significant differences in the mean concentration of cobalt between the exposure group and the control group (Table 2). Table 3 gives the results of biological monitoring at the start and end of the work shift.

### Table 2: Comparison of urinary cobalt between pre- and post-shift samples in the glazers and control group

| Group             | Mean ±SD  | t    | P    |
|-------------------|-----------|------|------|
| Pre-shift glazer  | 1.73±1.29 | 2.47 | 0.036|
| Control           | 1.25±0.75 |      |      |
| Post-shift glazer | 3.47±4.3  | 3.87 | <0.001|
| Control           | 1.25±0.75 |      |      |

### Table 3: Urinary cobalt measured at pre- and post-shift of the glazers in Meybood City of Iran

| Factors                  | Number | Pre-shift Mean±S.D (µg/L) | Post-shift Mean±S.D (µg/L) |
|--------------------------|--------|---------------------------|-----------------------------|
| Shift                    |        |                           |                             |
| Morning                  | 26     | 1.46±1.21                 | 3.08±3.67                   |
| Evening                  | 23     | 2.04±1.33                 | 3.87±4.93                   |
| Skin allergy             |        |                           |                             |
| No                       | 36     | 1.55±1.2                  | 2.54±3.01                   |
| Yes                      | 13     | 2.23±1.42                 | 5.7±6.05                    |
| Mask                     |        |                           |                             |
| No                       | 18     | 1.5±0.98                  | 4.58±5.3                    |
| Yes                      | 31     | 1.87±1.43                 | 2.77±3.4                    |
| Job                      |        |                           |                             |
| Pottery glazer           | 16     | 1.56±1.3                  | 3.7±4.8                     |
| Tile glazer              | 33     | 1.8±1.29                  | 3.39±4.17                   |
| Ventilation              |        |                           |                             |
| Non standard             | 35     | 1.77±1.29                 | 4.41±4.89                   |
| Standard                 | 14     | 1.64±1.34                 | 1.33±0.49                   |
| Work history             |        |                           |                             |
| ≤ 24                     | 15     | 1.53±1.19                 | 2.14±2.14                   |
| 60-25                    | 18     | 1.89±1.5                  | 3.65±4.97                   |
| 61 ≥                     | 16     | 1.75±1.18                 | 4.47±4.8                    |
| Overtime work            |        |                           |                             |
| No                       | 29     | 1.72±1.36                 | 2.75±3.28                   |
| 1-20                     | 12     | 1.67±0.72                 | 3.15±3.83                   |
| 21-60                    | 8      | 2.62±1.3                 | 7±7.9                       |
| BMI                       |        |                           |                             |
| <20                      | 6      | 1.83±0.75                  | 1.5±0.84                    |
| 25.99-20                 | 20     | 1.85±1.53                  | 2.83±4.34                   |
| 26-30.99                 | 18     | 1.44±1.25                  | 3.57±3.9                    |
| 31-40.99                 | 5      | 2.2±0.84                   | 8.75±3.24                   |
| Age                      |        |                           |                             |
| ≤ 25                     | 10     | 2.1±1.29                   | 2.5                          |
| 26-32                    | 22     | 1.45±1.22                  | 4±5.06                      |
| ≥ 32                     | 17     | 1.88±1.37                  | 3.6±3.92                    |

The glazers were subdivided in several subgroups according to the variables. Concentrations of Co in urine increased during the shift. On the other hand, the highest values of cobalt were observed at the end of the work shift. Based on the work history, there were no significant differences in
the mean values of urine cobalt levels between glazers, however, glazers with long work history (≥61 month) had a higher urine cobalt level compared to those who had work history of 24 months (4.47 μg/L versus 2.14 μg/L) (Table 3). Glazers who were working in the evening had higher urine cobalt compared to the morning shift (3.87 μg/L versus 3.08 μg/L). ANCOVA showed that, after adjusting for pre-shift cobalt, the effect of shift on post-shift cobalt was not significant (Table 4). Cobalt concentration was at high level among the glazers, suffering dermatitis skin. The mean value of cobalt concentration in glazers with dermatitis was 5.7 μg/L versus a mean of 2.54 μg/L among glazers without skin allergy (Table 3). ANCOVA showed that, after adjusting for pre-shift cobalt, the effect of skin allergy on post-shift lead was significant (Table 4).

Tile glazers had a slightly higher urine cobalt level compared to the pottery glazers (3.39 μg/L versus 3.7 μg/L). ANCOVA showed that, after adjusting for pre-shift cobalt, the effect of job on post-shift cobalt was not significant (Table 4).

Table 4: Results obtained after ANOCOVA for post-shift urinary cobalt by independent factors controlled for pre-shift cobalt

| Source                  | F   | P_value |
|-------------------------|-----|---------|
| Shift                   | 0.05| 0.82    |
| Skin allergy            | 4.84| 0.03    |
| Mask                    | 4.26| 0.04    |
| Job                     | 0.7 | 0.4     |
| Ventilation             | 5.15| 0.03    |
| Work history (month)    | 0.5 | 0.6     |
| Overtime work (hour)    | 5.07| 0.01    |
| BMI                     | 1.21| 0.32    |
| Age (year)              | 1.88| 0.42    |

Glazers without protective masks or local exhaust ventilation during mixing or handling of glazes had significantly higher levels of cobalt compared to the glazers with protection (4.58 μg/L versus 2.77 μg/L and 4.41 μg/L versus 1.33 μg/L respectively). ANCOVA showed that, after adjusting for pre-shift cobalt, the effect of masks and ventilation on post-shift cobalt was not significant (Table 4). Glazers who had work history more than 21 hour showed higher urine cobalt levels compared to the glazers with overtime work less of 21 (7 μg/L versus 2.75 μg/L). ANCOVA showed that, after adjusting for pre-shift cobalt, the effect of overtime work on post-shift cobalt was not significant (Table 4).

**Discussion**

In this study, the mean value of the urinary concentrations of cobalt in the glazers (3.47 μg/l) was below the ACGIH biological exposure indices of 15 μg/l (26). However, cobalt is an important constituent of blue glazes, however, in time of sampling, because of ceramic characteristics, the use of this metal in glazes was somehow lower the normal. Nevertheless, the glazers had a mean urinary cobalt concentration three to four times higher compared to the controls. The subjects exposed to a soluble cobalt pigment in pottery painting had an increase in the urinary cobalt concentration (27). In contrary to the results obtained through this study, Roig-Navarro et al. (1997) have reported high urinary cobalt level in the ceramic factories (28).

Exposure to cobalt may increase the risk of lung cancer. Relationship of cobalt in the development of lung cancer has been studied among women exposed to the cobalt blue dye. Results showed the incidence of lung cancer slightly higher than the expected value (29). Moulin et al. showed simultaneous exposure to cobalt and tungsten carbide causing lung cancer (30).

At the end of the shift, urinary concentrations of cobalt were higher than start of the shift levels, thus, results indicated that, cobalt is alimented immediately via urine. This study also confirms that, urinary cobalt seems to reflect the amount of cobalt recently absorbed. Since condition of workplaces such as ventilation system and glaze constituents did not change in the shifts, there was no significant difference between groups regarding to shift work.
This study showed that, skin dermatitis can increase the cobalt exposure. Urinary cobalt levels with skin dermatitis may represent existing poor health in this subgroup. This study also confirms the role of cobalt in occupational dermatitis similar to previous studies (31,32). Exposure to cobalt can be reduced by improving skin protection and personal hygiene in workplaces (33). Hygienic behavior has an impact on the absorption of cobalt. Improperly ventilation and working without mask was associated with significantly higher levels of cobalt in the urine of glazers.

Ichikawa et al. (1985 have indicated the level of cobalt in the blood and urine 2.5 and 1.8 times (respectively) lower in the workers who used respiratory protection devices compared to those who did not use respirators (34). Because of ceramic characteristics both pottery and tile glazers use glazes that generally contain the same amount of cobalt, therefore, this variable in pottery and tile glazers were not significant. In glazers with greater overtime work, due to more time exposure to cobalt, a significant increase in urinary cobalt level was observed. In the present study age, BMI, and work history were not significant variable. However, the highest values of cobalt were recorded among older workers due to the unhygienic behavior, higher BMI and work history.

In the present study, all spirometric parameters significantly decreased as compared to their controls. The decline in FEV1 and the ratio of FEV1/FVC was higher in those who have exposed to cobalt compared to those who have not exposed. Results obtained in this study showed a reduction in lung function, mainly of the obstructive syndrome type in the glazers. These findings are due to the effect of both the glaze constituents and raw material used such as silica. Bahrami et al. and Halvani et al. have reported exposure to silica related to reduction in pulmonary function and chronic obstructive pulmonary disease (COPD) (35, 36). In another study, in ceramic workers in Iran, respiratory complaints and abnormal pulmonary function were observed (37). Several studies have been indicated prevalence of chronic bronchial obstruction in relation to the cobalt exposure (38, 39).

Raffn et al. among plate painters exposed to the cobalt blue dye showed increased respiratory symptoms and airway resistance (40). Cobalt exposure is associated to a decline in FEV1 in smokers who are exposed to cobalt (41). Further studies are required to understand the mechanism of interaction between heavy metals and silica in prevalence impaired lung functions. Although cobalt poisoning occurred mainly among subjects who use cobalt for product blue colors in the ceramics, it could also occur in the general population during the normal use. Cobalt can be extracted into the food substances due to the improper formulation and firing of glazes. Acidic food and juice readily induce leaching of cobalt from glaze (42, 43). Cobalt is released by some of the dishes (44).

For reduction of cobalt exposure, all operations which disperse dust should be conducted in an isolated room. Washroom facilities and pre-employment and periodic medical test should be noticed. Workers should use respirators, not to eat or smoke at job position, separate work uniform from the other clothes to prevent the hazards of working with cobalt-containing glazes.

Conclusion

The urinary cobalt can reflect the amount of cobalt exposed by workers. Hence, biological monitoring is suggested as being useful to evaluate workers exposures. Also, hygienic behaviors, washing facilities, and using proper personal protection equipments can be considered as effective means to reduce the cobalt exposures.

Ethical considerations

Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely observed by the authors.

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References

1. Fores A, Liuas M, Badenes JA, Calbo J, Tena MA, Monros G (2000). Cobalt minimization in willemite (CoxZn2-xSiO4) ceramic pigments. Green Chemistry, 2: 93-100.
2. Lehman RL (2002). Lead Glasses for Ceramic Foodware. 1st ed. The International lead management center. United States of America.
3. Fiedler M (2009). Singing the moly blues: The direct use of molybdenum Clusters as a precursor to the development of molybdenum blue glasses. The Undergraduate Review, 5: 37-43.
4. Lauwerys R, Lison D (1994). Health risks associated with cobalt exposure—an overview. Si Tot Environ, 150: 1-6.
5. Nemery B, Verbeken E,K, Demedts M (2001). Giant cell interstitial pneumonia (Hard Metal Lung Disease, Cobalt Lung). Semin Respir Crit Care Med, 22(4): 435-448.
6. Schwartz L, Peck SM, Blair KE, Markuson KE (1945). Allergic dermatitis due to metallic cobalt. Allergy, 16: 51-53.
7. Coates EO, Sawyer HJ, Rebuck JW, Kvale PA, Sweet LW (1973). Hypersensitivity bronchitis in tungsten carbide workers. Chest, 64: 390.
8. Kusaka Y (1983). Hard metal asthma: a case of allergic bronchial asthma and contact dermatitis due to metallic cobalt. Jpn J Thor Dis, 21: 582.
9. Uter W, Ruhl R, Peahlberg A, Geier J, Schnuch A, Gefeller O (2004). Contact Allergy in Construction Workers: Results of a Multifactorial Analysis. Am occup Hyg, 48: 21-27.
10. Barborik M, Dusek J (1972). Cardiomyopathy accompanying industrial cobalt exposure. Br Heart, 34: 113-116.
11. Linna A, Oksa P, Groundstroem K, Halkosaari M, Palmroos P, Huikko S, Uitti J (2010). Exposure to cobalt in the production of cobalt and cobalt compounds and its effect on the heart. Occup Environ Med, 61: 877–885.
12. Davison AG, Haslam PL, Corrin B, Coutts JJ, Dewar A, Riding WD, Studdy PR, New-man.
13. Van Cutsem EJ, Ceuppens JL, Lacquet LM, Demedts M (1987). Combined asthma and alveolitis induced by cobalt in a diamond polisher. Eur J Respir Dis, 70: 54-61.
14. Shirakawa T, Kusaka Y, Fujimura N, Goto S, Kato M, Heki S, et al., (1989). Occupational asthma from cobalt sensitivity in workers exposed to hard metal dust. Chest, 95: 29-37.
15. Krakowiak A, Dudek W, Tarkowski M, Swiderska-Kielbik S, Niesiernenko E, Palczynski C (2005). Occupational asthma caused by cobalt chloride in a diamond polisher after cessation of occupational exposure: A case report. Inter J Occup Med Environ Health, 18: 151-158.
16. Harding HE (1950). Notes on the toxicology of cobalt metal. Br J Ind Med, 7: 76-78.
17. Vainio H, Wilbourn J (1992). Identification of carcinogens within the IARC monograph program. Samal J Work Environ Health, 18: 64-73.
18. Shahtaheri SJ, Khadem M, Golbabaei F, Rahimi-Froushani A (2007). Optimization of sample preparation procedure for evaluation of occupational and environmental exposure to Nickel. Iranian J Publ Health, 36 (2): 73-81.
19. Shahtaheri SJ, Khademi M, Golbabaei F, Rahimi-Froushani A (2007). Solid phase extraction for evaluation of occupational exposure to Pb(II) using XAD-4 prior to atomic absorption spectroscopy. Int J Occup Safety Ergonomics (JOSE), 13(2): 137-145.
20. Shahtaheri SJ, Mesdaghinia A (2005). Stevenson D. Evaluation of factors influencing recovery of herbicide 2, 4-D from drinking water. Iran J Chem Chem Eng (IJCCE), 24(1): 33-40.
21. Shahtaheri SJ, Ibrahiimi I, Golbabaei F, Hosseini M (2007). Solid phase extraction for 1-hydroxypyrene as a biomarker of exposure to PAHs prior to high performance liquid chromatography. Iran J Chem Chem Eng (IJCCE), 26(4): 75-81.
22. Shahtaheri SJ, Abdollahi M, Golbabaei F, Rahimi-Froushani A (2008). Ghamari F. Sample preparation followed by HPLC for monitoring of mandelic acid as a biomarker of environmental and occupational exposures to styrene. Int J Environ Res, 2(2): 169-176.

Available at: http://ijph.tums.ac.ir
23. Koohpaei AR, Shahtaheri SJ, Ganjali MR, Rahimifroushani A, Golbabaei F (2008). Molecular imprinted solid phase extraction for determination of atrazine in environmental samples. *Iranian J Environ Sci Eng*, 5(4): 283-296, 2008.

24. Heidari HR, Shahtaheri SJ, Golbabaei F, Alimohammadi M, Rahimifroushani A (2009). Trace analysis of xylene in occupational exposures monitoring. *Iranian J Publ Health*, 38(1): 89-99.

25. Rahiminejad M, Shahtaheri SJ, Ganjali MR, Rahimifroushani, Koohpaei AR, Golbabaei F (2010). An experimental investigation of the molecularly imprinted polymers as tailor-made sorbents of diazinon. *Journal of Analytical Chemistry*, 65(7): 694-698.

26. American Conference of Governmental Industrial Hygienists (NIOSH) (2010). *Threshold limit values for chemical substances and physical agents and biological exposure indices*, Cincinnati, OH: US.

27. Christensen J, Mikkelsen S (1985). Cobalt concentration in whole blood from pottery plate painters exposed to cobalt paint. Int Conference on "Heavy metals in the Environment", pp:86-89.

28. Roig Navarro AF, Lopez FJ, Serrano R, Hernandez F (1997). An assessment of heavy metals and boron contamination in workplace atmospheres from ceramic factories. *Sci Tot Environ*, 201: 225-234.

29. Tuchsen F, Jensen MV, Villadsen E, Lynge E (1996). Incidence of lung cancer among cobalt-exposed women. *Scand J Work Environ Health*, 22: 444-50.

30. Moulin JJ, Wild P, Romazini S, Lasfargues G, Bozec C, et al., (1998). Lung Cancer in Hard-Metal Workers. *Am J Epidemiol*, 148(3): 241-248.

31. Athavale P, Shum KW, Chen Y, Agius R, Cherry N, Gawrrodger DJ, et al., (2007). Occupational dermatitis related to chromium and cobalt: experience of dermatologists (Epiderm) and occupational physicians (OPRA) in the U.K. over an 11 year period (1993–2004). *Br J Dermatol*, 157: 518-522.

32. Kanerva I, Jolanki R, Estlander T, Alanko K, Savela A (2000). Incidence rates of occupational allergic contact dermatitis caused by metals. *Am J Contact Dermatitis*, 11(3): 155–60.

33. Linnainmaa M, Kiihonen M (1997). Urinary cobalt as a measure of exposure in the wet sharpening of hard metal and stellite blades. *J Int Arch Occup Environ Health*, 69: 193-200.

34. Ichikawa Y, Kusaka Y, Goto S (1985). Biological monitoring of cobalt exposure, based on cobalt concentrations in blood and urine. *Int Arch Occup Environ Health*, 55(4): 269-276.

35. Bahrami AR, Mahjub H (2003). Comparative study of lung function in Iranian factory workers exposed to silica dust. *East Mediterr Health*, 9:390-8.

36. Halvani Gh, Zarei M, Halvani A, Barkhordari A (2008). Evaluation and comparison of respiratory symptoms and lung capacities in tile and ceramic factory workers. *Tanaffos*, 8: 19-25.

37. Sprince NL, Chamberlin RI, Hales CA, Weber AL, Kazemi H (1984). Respiratory disease in tungsten production workers. *Chest*, 86: 549.

38. Kusaka Y, Ichikawa Y, Shirakawa T, Goto S (1986). Effect of hard metal dust on ventilatory function. *Br J Ind Med*, 43: 486-489.

39. Raffn E, Mikkelsen S, Altman DG, Christensen JM, Groth S (1998). Health effects due to occupational exposure to cobalt blue dye among plate painters in a porcelain factory in Denmark. *Work Environ Health*, 14: 378-84.

40. Verougstraete V, Mallants A, Buchet JP, Swennen B, Lison D (2004). Lung function changes in workers exposed to cobalt compounds. *Am J Respir Crit Care Med*, 170: 162–166.

41. Omolaoye JA, Uzairu A, Gimba CE (2010). Heavy metal assessment of some ceramic products imported into Nigeria from China. *Arch Appl Sci Res*, 2: 120-125.

42. Selden AI, Norberg C, Karlson-Stiber C, Hellstrom-Lindberg E (2007). Cobalt release from glazed earthenware: Observations in a case of lead poisoning. *Environ Toxic and Pharma*, 23: 129–131.

43. Sheets RW (1998). Release of heavy metals from European and Asian porcelain dinnerware. *Sci Tot Environ*, 212: 107-113.