

Occupational Exposure to Arsenic in Moi University and University of Eldoret Instructional Laboratories and Potential Carcinogenic Risks

 Nthenya S.^{1}, Simiyu M. G.¹ and Otieno A. C.²
¹Department of Biology and Health, School of Environmental Studies, University of Eldoret, P. O. Box 1125-30100 Eldoret, Kenya
²Department of Environmental Health, School of Public Health, Moi University, P. O. Box 3900-30100 Eldoret, Kenya

*Corresponding author's email address: <u>dnsalee@gmail.com</u>

Abstract

Virtually, all occupations to some extent have inherent risks. The objective of the study was to assess the role of arsenic (As) in universities' instructional laboratories indoor settled dust as an occupational carcinogenic risk medium. Dust samples were thus collected from Moi University and University of Eldoret within Uasin Gishu County Kenya, according to standard procedure and As analysis determined using the S1 Titan X-ray fluorescence (XRF) spectrometer. Concentrations ranged from 0.04-349.24 mg/kg while mean As concentrations ranged from 0.424-131.73 mg/kg at ETD and RMD stations, respectively. Mean As concentrations varied significantly and decreased in the order of RMD > EWW >RMR > MC > REW > MSM > EC > ECA > MMW > ETD. Comparison with acceptable ELCR (1×10^{-4} - 1×10^{-6}) levels indicated that over 50% of the stations were significantly (95 % (CI); p < 0.05) exposed to As CTE and RME carcinogenic risks for both men and women. RMD work-unit posed the highest risk for men and women, respectively. The findings indicate that employees were theoretically exposed to inherent As carcinogenic risks as a result of their work predisposition. The study recommends that appropriate biomarkers such as total urine arsenic be used to ascertain the magnitude of exposure and for occupational exposure monitoring.

Keywords: Carcinogenic risk, Arsenic, Indoor settled dust, Occupational

INTRODUCTION

Arsenic (As) is a naturally occurring element, accounting approximately 0.0002% of the earth's crust. It is ubiquitous in the environment and therefore human exposure can occur from myriad of sources. Arsenic which has been ranked number one on the Agency for Toxic Substances and Disease Registry (ATSDR) "Top 20 List" is the most known cause of acute heavy metal poisoning in adults. Inorganic arsenic has been classified as a known human carcinogen by the ATSDR (2015).

Arsenic as an element does not easily break down therefore its persistent in the environment; however, it can change from inorganic to organic forms. Besides chemical reactions such as oxidation-reduction reactions, other various natural processes affect its transport and fate in water and soil. These include bio-transformations and ligand exchange reactions. Further, it has been shown to persist in soil for over 45 years. Arsenic may also disperse in the air but will eventually settle out and deposit in outdoor soils or indoor dust (Singh *et al.*, 2015).

Arsenic has seen a wide application; inorganic arsenic for instance has been widely used in the wood industry where it is used as a preservative in form of CCA (chromated copper arsenate). It has also been used in soaps, metals semi-conductors, paints, glassware, dyes, drugs, agricultural products and applications, as well as in industrial and electrical utilities. Surface dust sampling can provide a wealth of material that can be harnessed for estimation of potential contact with any levels of contaminants of concern. Settled surface dust has been reported to often function as a reservoir of hazardous particulate contaminants including trace metals. Indoor settled dust presents as a composite of particulate matter derived from both indoor and outdoor sources (Shraim *et al.*, 2016; Mohammed and Crump, 2013).

Many past studies on arsenic exposure via indoor dust have mainly targeted household indoor dust (Middleton *et al.*, 2018; Liu *et al.*, 2016; Shraim *et al.*, 2016; Al-Madanat *et al.*, 2017). In occupational settings, surface indoor dust samples have often provided vital information in two occasions; first, hands of the employees can inadvertently come into contact with settled dust on a surface and then be subsequently orally taken up when transferred from hand-mouth; and secondly, when the contaminant on the surface can be dermally absorbed if the skin comes into contact frequently with the contaminated surface dust (Gorman *et al.*, 2014).

In a risk assessment study done in Pakistan by Subhani *et al.* (2015), total As in outdoor soil dust was found to be 2-3 times less than indoor dust and arsenic bio-accessibility ranged from 13.8% to 20.2% in outdoor soil dust while that of indoor dust ranged from 75.4% to 83.2%. Further, Gorman *et al.* (2017) recently asserted that risk assessments have greatly underestimated the role of inadvertent exposure in risk assessments. His study was in support of a positive correlation between exposures on the hands and exposure on the perioral area as found in a past study by Christopher *et al.* (2008).

The U.S. EPA (2006) policy on exposure assessment requires that a range of possible exposure scenarios be considered as opposed to a specific value. Both reasonable maximum exposure (RME) estimates or "high end" and central tendency exposure (CTE) estimates risk assessments should thus be included. The Oregon State for instance requires that both CTE and RME be considered in the risk assessments (Oregon Dept. of Environmental Quality, 2010).

Carcinogenicity for chronic arsenic exposure has been significantly associated with liver, prostrate, skin, liver, kidney and bladder cancer. Besides, recent studies have also suggested a relationship with diabetes, neurological effects, cardiac disorders and reproductive organs (Hong *et al.*, 2014; Garcia-Esquinas *et al.*, 2013; Nizam *et al.*, 2013).

Owing to the nature of instructional laboratories activities in universities, occupational exposure to heavy metals cannot be ruled out. This study therefore sought to address the potential of settled indoor dust in instructional laboratories as a medium of As heavy metal occupational carcinogenic health risk.

METHODOLOGY

Study Area

Moi University and University of Eldoret, the public universities instructional laboratories within Uasin Gishu County, were the target of this study (Fig. 1). The study area thus comprised of Moi University (MU) and University of Eldoret (UoE) located approximately

36 km southeast and 10 km to the north of Eldoret Town, in Uasin Gishu County, respectively. The study area is located in latitudes 0° 30' S and 0° 35' N and longitudes 35° 30' E and 35° 37'.



Figure 1: Map showing the study area

Out of the 20 instructional laboratories in total identified, ten (50%) were sampled for this study. practical lessons carried out in the select instructional laboratories that may expose As to the staff working in these facilities may include but not limited to pure As handling and stock/spiking spillages; analysis of pesticides; wood preservation and treatment with CCA, wood glue, wood dust and chips; welding and soldering fumes, bearings, electrotype metal cutting and soldering; repair and maintenance of electric semiconductors/devices e.g. transistors, capacitors and resistors, circuit boards, electric motors bearings and other semiconductors/devices.

Sampling, Sample Preparation and Analysis

A total of 100 composite indoor settled dust samples were collected using new pre-cleaned polyethylene brush and dustpan from the floors, corners and wiping of visible dust on raised areas such as windowsills and sash areas and equipment tops and with dry ashless filter paper (Whatman No. 42) according to standard procedures as applied by Ardashiri and Hashemi (2017). The collected dust samples were placed into sealed and well-labeled ziploc bags and transported to the laboratory for analysis.

Arsenic analysis was carried out using the S1 Titan X-ray fluorescence (XRF) Spectrometer (Bruker Model). Completely dry dust samples were sieved to $< 250 \mu m$ using standard testing sieve. Dust samples were then screened using a XRF analyzer. The XRF was calibrated using standard procedures as per the user manual prior to use. Approximately 10 g from each of the 100 previously dried and sieved dust samples was scooped into the sample cup up to $\frac{34}{4}$ full and placed in the XRF directly to the detector. The S1 Titan XRF was then mounted on a stand and interfaced with a computer and once the detection trigger was placed, the detected As levels were read directly from the interfaced computer in parts per million (mg/kg).

Quality Control

To ensure accuracy of the data obtained, at each sampling station, dust samples were collected using pre-cleaned brush and dust pan and samples placed into new sealed and well-labeled ziploc bags. Cross contamination of samples during XRF analysis was minimized by pre-cleaning the scooper and sampling cup before analyzing each sample.

Potential Occupational Carcinogenic Risk Assessment

The oral and dermal routes of exposure assessment including CTE and RME risks were estimated by determining their respective carcinogenic Lifetime Average Daily Intake (LADI) in mg/kg/bw using U.S. EPA (2011) models.

Carcinogenic oral lifetime average daily intake (LADIing) was determined as;

$$LADIng = \frac{EPC_S \times CR \times EF \times ED}{BW \times AT_C \times UCF}$$

Carcinogenic LADI via dermal contact (LADIder) was determined using the equation:

$$LADIder = \frac{EPC_{S} \times SAd \times CF \times AF \times EF \times ED \times ABSder}{BW \times AT_{C} \times UCF}$$

LADI for dermal contact and subsequent incidental ingestion (LADIder/ing) was calculated as:

$$LADIder/ing = \frac{EPC_S \times SAi \times CF \times ABSder \times fdo \times fgi \times EF \times ED}{BW \times AT_C \times UCF}$$

Whereby: EPCs is As exposure point concentration (mg/kg); BW is the body weight (70 kg for men: 60 kg for women); EF is exposure frequency (days/yr); ED is exposure duration (yrs); CF is the contact frequency; CR is contact rate (mg/day); SAd is skin surface area for dermal route (cm²); SAi is skin surface area for ingestion route (cm²); ABSder is dermal absorption fraction; fdo is dermal-oral fraction transfer; fgi is fraction GI absorption; AF is the skin adherence factor (mg/cm²); AT_c is the averaging time for cancer (25,550) and UCF is the unit conversion factor (10⁻⁶).

Risk characterization was estimated as the incremental probability of an individual developing cancer over a lifetime as a result of exposure to the potential carcinogen as:

$Risk = LADI \times SF$

Where risk is unit less and CSF is the cancer slope factor which is chemical specific (for instance, the oral cancer slope factor for As is 1.5 mg/kg/day). The total excess lifetime cancer risk (ELCR) was finally calculated as:

Risk (total) = Risk (der/ing) + Risk (der) + Risk (ing)

Where Risk (der/ing), Risk (ing) and Risk (der) were the extrapolated risks through dermal and subsequent ingestion, ingestion and dermal pathways respectively. Calculated risks were then compared with U.S. EPA's acceptable excess lifetime cancer risk (ELCR) of $1x10^{-6}$ - $1x10^{-4}$ risk levels (or one individual in 1,000,000 – one individual in 10,000 persons developing cancer).

RESULTS AND DISCUSSION

Concentration of Arsenic Heavy Metal in settled indoor dust

Arsenic concentrations ranged from 0.04 mg/kg to 349.24 mg/kg while mean As concentrations ranged from 0.424 mg/kg -131.73 mg/kg in samples from ETD and RMD, respectively (Figure 2). Mean As concentrations in the sampling sites decreased in the order of RMD > EWW > RMR > MC > REW > MSM > EC > ECA > MMW > ETD. Mean dust levels were significantly lower (p > 0.05) in most (80%) sampling stations than EU and FAO/WHO (20 mg/kg) while 60% of the stations significantly surpassed (p < 0.05) U.S. EPA (7 mg/kg) recommended standards in uncontaminated dust (WHO, 2010).

Elevated As levels at EWW sampling station could be attributed to the use of treated wood in the facility which is commonly used for practical lessons by the Wood Science and Technology students. Arsenic is a component of wood preservatives such as chromated copper arsenate (CCA) as reported in a study by Kwon *et al.* (2004) on As contamination arising from use of CCA as a wood preservative. Further, Zartarian (2006), reported higher mean As concentrations derived from CCA playgrounds as compared to those obtained for the non-CCA playgrounds with these results relatively lower compared to the findings for this study.

Similar studies of examining As metal in university's instructional laboratories indoor settled dust were not found in the literature as a means for comparison. Although, As levels from the findings of this study were less when compared to results from other studies in different environmental scenarios (Kamunda *et al.*, 2016; Liu *et al.*, 2010; Kar *et al.*, 2013; Huang *et al.*, 2017), they were higher than those of a study in nursery schools by Lu *et al.*, (2014). These findings suggest the importance of universities instructional laboratories as environmental scenarios to occupational heavy metal exposure risks.



Figure 2: Arsenic Concentrations in the Sampling Stations.

EWW (Wood science workshop); ECA (Chemistry Lab 1); EC (Chem Lab 3); ETD (Technology Education workshop); MC (Chemistry Lab); MMW (Welding shop); MSM (Sheet metal shop); RE.

Occupational Arsenic Carcinogenic Risk Characterization

Calculated mean As risks for the various sampling stations were as presented in (Table 1). The RMD sampling station had the highest risk at 13.9584×10^{-4} and 16.2847×10^{-4} while ETD recorded the least carcinogenic risk at 0.00689×10^{-4} and 0.00813×10^{-4} for men and women respectively.

One sample t-test results 95% (CI) for comparison of As carcinogenic risk from the considered pathways for both men and women in the sampling stations with acceptable ELCR values were also as presented in Table 1. The results indicate that all the sampling stations had significantly higher risk levels than the lower bound ELCR value of 1 x 10^{-6} value (p < 0.05) except for men at ETD station (p = 0.0977). However, comparison with upper bound acceptable ELCR (1 x 10^{-4}) levels indicated that 50% of the stations (EWW, MC, REW, RMR and RMD) had significantly higher risks (p < 0.05) for both men and women.

One-way ANOVA analysis indicated there was significant variation for As carcinogenic risk in men (p = 0.0021) and women (p = 0.0096) between the sampling stations. Further, Tukey's HSD post hoc analysis found there was significant variation in men and women As risk between sampling stations (p < 0.05). Significant variations (p < 0.05) were found to be due to variations between RMD and all the other sampling stations in both men and women respectively. This could be attributed to the very high As levels recorded in this particular station as compared to the rest.

	Mean Ris	k l	P value (1-tailed)		P value (1-tailed)		P value
	(×10 ⁻⁴)		(µ=1 x 10 ⁻⁴)		(µ=1 x 10 ⁻⁶)	(2-tailed)
			-		-		M and W
S.S	М	W	Μ	W	М	W	
EWW	2.7071	3.1582	0.0113	0.0099	0.0437	0.0437	0.8156
EC	0.4989	0.5819	0.1166	0.3083	0.0127	0.0125	0.7158
ECA	0.2682	0.3129	0.0853	0.0836	0.0043	0.0095	0.4937
ETD	0.0069	0.0081	0.7905	0.3160	0.0977	0.0589	0.4917
MC	1.4159	1.6519	0.0134	0.0081	0.0062	0.0062	0.6563
MMW	0.1518	0.1771	0.6455	0.1450	0.0321	0.0370	0.8282
MSM	0.9543	1.1134	0.4617	0.3513	0.0172	0.0179	0.7132
REW	1.1549	1.3475	0.0263	0.0134	0.0050	0.0043	0.6600
RMR	1.6446	1.9186	0.0139	0.0076	0.0006	0.0005	0.3955
RMD	13.9584	16.2847	0.0064	0.0081	0.0055	0.0073	0.7040
S.S-Sampling Station M-Men				W-Women			

Table 1: One Sample t-test p Values for Arsenic Carcinogenic Risk Characterization

Arsenic theoretical carcinogenic risks were therefore anticipated in 50% of the sampling stations. Notably was the RMD work-unit which posed very high As carcinogenic risk at 1.39584E-03 (1 in every 716 individuals) and 1.62847E-03 (1 in every 614 individuals) for men and women respectively which is regarded as "moderate" increased risk hence unacceptable risk. Elevated risk recorded at the EWW station which recorded the second highest risk could be attributed to the use of CCA treated wood in its operations. Arsenic carcinogenic risk characterization at ETD for instance was considered "extremely low" increased risk at 0.813E-06 (one in every 1,230,012 individuals) in men.

Arsenic CTE and RME Risks

Arsenic CTE risks ranged from 0.225×10^{-4} (one in every 44,444 individuals) – 37.0062×10⁻⁴ (one in every 270 individuals) and 0.2621×10^{-4} (one in every 38,153 individuals) – 43.1739×10⁻⁴ (one in every 232 individuals) for men and women, respectively. On the other hand, RME risks ranged from 0.273×10^{-4} (one in every 36, 630 individuals) – 84.045×10⁻⁴ (one in every 118 individuals) and 0.291×10^{-4} one in every 34,364 individuals) – 98.053×10⁻⁴ (one in every 101 individuals) for men and women, respectively. While RMD station posed the highest risks in all cases, ETD posed the least CTE risk while MMW posed the least RME risk (Table 2).

S.S	M/W	RME RISK	RME <i>p</i> values	CTE RISK	CTE <i>p</i> values
		(×10 ⁻⁴)	L.	(×10 ⁻⁴)	Å
EWW	М	5.844	0.004	2.707	0.001
	W	6.818	0.004	3.158	0.001
EC	М	3.403	0.048	0.499	0.219
	W	3.970	0.038	0.582	0.321
ECA	М	0.756	0.089	0.268	0.094
	W	0.882	0.924	0.313	0.293
ETD	М	0.642	0.658	0.225	0.976
	W	0.675	0.827	0.262	0.872
MC	М	3.955	0.032	1.416	0.050
	W	4.614	0.001	1.652	0.006
MMW	М	0.273	0.481	0.759	0.295
	W	0.291	0.458	0.885	0.119
MSM	Μ	2.796	0.539	0.954	0.046
	W	3.280	0.071	1.052	0.049
REW	М	3.097	0.439	1.155	0.042
	W	3.613	0.044	1.348	0.024
RMR	М	2.641	0.012	6.100	0.010
	W	3.209	0.036	7.081	0.003
RMD	М	84.045	0.038	37.006	0.000
	W	98.053	0.038	43.174	0.000

Table 2: One Sample t-test p Values for Arsenic RME and CTE Risks

At 95% (CI) one sample t-test comparison of extrapolated As CTE and RME risk results with upper bound ELCR (1×10^{-4} or one in 10,000 individuals) found that 40% (EC, ECA, ETD and MMW) and 30% (ECA, ETD and MMW) were theoretically significantly safe from As carcinogenic risks. Therefore, whereas centrally exposed staffs from ECA work station were theoretically safe, "high end" exposed staff from the same station were at risk to As carcinogenic risk. These findings therefore exemplify the importance of considering a range of possible exposure scenarios in human health risk assessments in order to increase certainty.

The findings of the study were, however, way below those for arsenic CTE cancer health risk for Tamso (approximately 10 out of 100 individuals) and Prestea (approximately 1 out of 100 individuals) areas in Ghana as reported by Obiri *et al.* (2006), but higher than those reported by US Dept. of Health and Human Services (2014) though for different environmental scenarios. Further, RME results for both Pb and As in this study were all below those reported by Ted (2014) though for a different environmental scenario. Long-term exposure to As also known as arseniasis can cause skin cancer, carcinoma, cancers in lungs, liver, urinary bladder, kidney and colon (Baker *et al.*, 2018; Armah, 2012).

The study concluded that instructional laboratories staff was not entirely safe from As carcinogenic risks. RMD sampling station was found to be the most exposed with "moderate" increased risk for As carcinogenic risk which should be of concern. There was no documented evidence of prior risk assessments in the study area; therefore these findings may be useful as a screening study. Due to the presence of and elevated As levels recorded in the sampling stations, the study recommends that appropriate biomarkers such as total

urine arsenic be used for occupational exposure monitoring within the exposed facilities besides workers using appropriate personal protective equipment at all times.

REFERENCES

- Agency for Toxic Substances and Diseases Registry (ATSDR) (2015). *Toxicological Profile for Arsenic*. <u>http://www.atsdr.cdc.gov/toxprofiles/tp2.pdf</u>. Accessed on Feb 26, 2018.
- Al-Madanat, O., Jiries, A., Bartarseh, M., Al-Nasir, F. (2017). Indoor and Outdoor Pollution with Heavy Metals in Al-Krak City, Jordan. J. Int. Environmental Application and science, 12(2): 131-139.
- Ardashiri, S., Hashemi, S. E. (2017). Health risk assessment of heavy metals in indoor dust from Bushehr, Iran. Iranian Journal of Health, Safety & Environment, 5 (2), 966 – 971.
- Armah, F. A., Markku, K., Luginaah, I., Mkandawire, P. (2012). Non Occupational Health Risk Assessment from Exposure to Chemical Contaminants in the Gold Mining Environment of Tarkwa, Ghana. *Trends in Applied Sciences Research*, 7, 181 - 195.
- Baker, B. A., Cassano, V. A. and Murray, C. (2018). Arsenic Exposure, Assessment, Toxicity, Diagnosis and Management. JOEM, 60(12):e634-e639.
- Christopher, Y. (2008). Inadvertent ingestion exposure to hazardous substances in the workplace. PhD Thesis, Aberdeen: University of Aberdeen.
- Garcia-Esquinas, E., Pollan, M., Umans, J. G., Goessler, W., Guallar, E., Howard, B., Farley, J., Yeh, J., Best, L. G., Navas-Acien, A. (2013). Arsenic exposure and cancer mortality in a US-based prospective cohort: the strong heart study. *Cancer Epidemiol Biomarkers Prev*, 22(11):1944-1953.
- Gorman, M. N., Davis, A., Tongeren, M., Cowie, H., Semple. S. (2014). Inadvertent Ingestion Exposure: hand and object-to-mouth behavior among workers. J. Sci Environ Epidemiol 26(1).
- Gorman, M. N., MacCalman, L., Semple, S., Tongeren. M., (2017). Field Measurements of Indvertent Ingestion Exposure to Metals. Annals of Work Exposures and Health, 61 (9), 1097–1107.
- Hong, Y., Song, K. and Chung, J. (2014). Health Effects of Chronic Arsenic Exposure. J Prev Med Public Health, 47(5):245-252.
- Huang, S. H., Li, Q., Yang, Y., Yuan, C. Y., Ouyang, K. and You, P. (2017). Risk Assessment of Heavy Metals in Soils of a Lead-Zinc Mining Area in Hunan Province (China). *Kem. Ind.* 66 (3-4), 173–178.
- Kamunda, C., Mathuthu, M., Madhuku, M. (2016). Health Risk Assessment of Heavy Metals in Soils from Witwatersrand Gold Mining Basin, South Africa. Int. J. Environ. Res. Public Health, 13(7), 663.
- Kar, S., Das, S., Jean, J., Chakraborty, S., Liu, C. (2013). Arsenic in the water-soil-plant system and the potential health risks in the coastal part of Chianan Plain, Southwestern Taiwan. J Asian Earth Sci, 77, 295 - 302.
- Kwon, E., Zhang, H., Wang, Z., Jhangri, G., Lu, X., Fok, N., Gabos, S., Li, X., Le, X. (2004). Arsenic on the Hands of Children after Playing in Playgrounds. *Environmental Health Perspectives*, 112, (14), 1375-1380.
- Liu, Y., Junwei M., Hongxia, Y., Yuqing, R. (2016). Bioacesssibility and health risk assessment of arsenic in soil and indoor dust in rural and urban areas of Hubei province, China. *Ecotoxicology and environmental* safety, 126:14-22.
- Lu, X., Zhang, X., Li, L.Y. and Chen, H. (2014). Assessment of metals pollution and health risk in dust from nursery schools in Xi'an, China. *Environ. Res.* 128: 27–34.
- Middleton, D. R. S., Watts, M. J., Hamilton, E. M., Coe, J. D., Fletcher, T., Crabbe, H., Close, R., Leonardi, G. S., Polya, D. A. (2018). Surface wipe and bulk sampling of household dust: arsenic exposure in Cornwall, UK. *Environ. Sci.: Processes Impacts*, 20, 505 512.
- Mohammed, F. S. and Crump, D. (2013). Characterization of Indoor/Outdoor Settled Dust and Air Pollutants in Damaturu, Nigeria. IACSIT International Journal of Engineering and Technology, 5(1):104-108.
- Nizam, S., Kato, M., Yatsuya, H., Khalequzzaman, M., Ohnuma, S., Naito, H., & Nakajima, T. (2013). Differences in urinary arsenic metabolites between diabetic and non-diabetic subjects in Bangladesh. *International journal of environmental research and public health*, 10(3), 1006-1019.
- Obiri, S., Dodoo, D. K., Okai-Sam, F., Essuman, D. K., Adjorlolo-Gasokpoh, A. (2006). Cancer an non-cancer health risk from eating cassava grown in some mining communities from Ghana. *Environ Monit Assess*. 118(1-3), 37 49.
- Oregon Department of Environmental Quality (2010). Human Health risk Assessment Guidance Environmental Cleanup Program, State of Oregon.
- Shraim, A.M., Alenazi, D.A., Abdel-Salam, A. G., Kumar, P. (2016). Loading Rates of Dust and Metals in Residential Houses of Arid and Dry Climatic Regions. *Aerosol and Air Quality Research*, 166: 2462-2473.
- Nizam, S., Kato, M., Yatsuya, H., Khalequzzaman, M., Ohnuma, S., Naito, H., & Nakajima, T. (2013). Differences in urinary arsenic metabolites between diabetic and non-diabetic subjects in Bangladesh. *International journal of environmental research and public health*, 10(3), 1006-1019.

African Journal of Education, Science and Technology, December, 2019, Vol 5, No. 3

- Subhani, M., Mustafa, I., Alamdar A., Katsoyiannis, I. A., Ali, N. and Huang, Q. (2015). Arsenic levels from different land-use settings in Pakistan: Bio-accumulation and estimation of potential human health risk via dust exposure, *Ecotoxicol Environ Safety*, 115:187-194.
- Ted, S. (2014). Environmental Risk Assessment; A Toxicological Approach, Taylor and Francis Group, CRC press, New York.
- World Health Organization. (2010). Human Health Risk Assessment Toolkit: Chemical Hazards. IPCS harmonization project document, no. 8. WHO Press, Geneva, Switzerland.
- U.S. Dept. of Health and Human Services (2014). *Health consultation soil data review for properties near the former John T. Lewis and Brothers Site*. Philadelphia, Pennsylvania. Agency for Toxic Substances and Disease Registry, Division of Community Health Investigation. June, 2014.
- U.S. EPA (2006). Guidelines for Exposure Assessment. U.S. Environmental Protection Agency, Risk Assessment Forum, Washington, DC, 600Z-92/001.
- U.S. EPA (2011). *Exposure Factors Handbook: 2011 Edition*. EPA/600/R-09/052F. Washington, D.C.: United States Environmental Protection Agency, Office of Research and Development, National Center for Environmental Assessment.
- Zartarian, V. G., Xue, J., Ozkaynak, H. Dang, W., Glen, G., Smith, L., Stallings, C. (2006). A probabilistic arsenic exposure assessment for children who contact CCA-treated playsets and decks, part 1: model methodology, variability results, and model evaluation. *Risk Anal*, 26:515 – 31.