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Cadmium (Cd) Exposure among Waste Collector in Urban Area, Malaysia

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ABSTRACT

Introduction: Cadmium (Cd) in urine and inhaled dust of the municipal waste operators was assessed. **Methods:** Urine spot samples were collected and analysed for Cd and creatinine of 60 municipal waste operators between April to June 2013. Respirable dust was collected using personal air sampling pump GilAir-3 and GilAir-5 for 8 working hours. Cd in urine and dust were analysed using the Flame Atomic Absorption Spectrometer (Perkin Elmer A Analyst 800) while urinary creatinine was measured using Reflotron® Plus creatinine. **Results:** The mean and standard deviation (SD) of Cd in the respirable dust (0.59 ± 50.27 µg/m3) was within the permissible exposure limit (PEL). The level of Cd in urine (0.015 ± 0.0097 µg/g Cr) was lower than the safe limit of 5 µg/g. The creatinine level (173.59 ± 50.27 mg/dl) was within the normal range (20 to 350 mg/dl). The multiple regression model shows smoking and years of smoking were the significant predictors for the Cd in the urine (R = 0.216 F(3,56) = 5.150, p < 0.05). **Conclusion:** Municipal waste operators were exposed to minimal Cd exposure while handling waste and the accumulation of this metal urine was correlated with smoking habit.

Keywords: Cadmium, Biological monitoring, Waste operator, Dust, Health risk

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INTRODUCTION

Municipal waste collectors are hired by the local authority to collect waste from residential areas, commercial, industrial and other collection site for disposal. They are exposed to one or more workplace hazards while loading the waste into the garbage truck to be transported to the disposal sites (i.e. landfill) (1, 2). Among the common types of hazards involved are physical (i.e. broken glasses), biological (i.e. germ, bacteria, viruses), chemicals (i.e. chemical liquid, detergent), and ergonomic hazard (i.e. musculoskeletal disorder from waste loading onto trucks, falling) (1). These workers are vulnerable to diseases caused by inhaling dust, smoke, fume and bad odours in the landfill. They also may be involved in traffic accidents while at work (1).

Landfilling is the only waste disposal method that can treat all materials in the solid waste stream (1, 2). Other disposal options are incineration or thermal treatments which also produce ashes that need to be landfilled. In the most basic form of landfill, waste is usually dumped in a large hole or on the land's surface described as open dumping. This is an uncontrolled deposition of waste material and there is no distinction between household or hazardous materials and is often open burning to reduce volume. Open dumping is also an unsatisfactory method of final disposal as it is not based on engineering design (2). As waste disposal technology has evolved, open dumping is no longer acceptable, and sanitary landfill has been introduced in order to replace this most basic method.

Studies have identified more than 400 hazardous compounds including organic and metal organic compound and inorganic elements were detected in landfill leachate (3). Compounds detected include heavy metals, halogenated aliphatic compounds, benzene and alkylated benzenes, phenol and alkylated phenols, ethoxylates, polycyclic aromatic compounds, PCBs, chlorinated dioxins and chlorinated furans, pesticides, organic tin, and methyl mercury.

Heavy metals are defined as those elements with specific density of more than 5 g/cm3 (4). Among the most heavy metals with human health concern are Cadmium (Cd), Copper (Cu), Arsenic (As), Mercury (Hg), Chromium (Cr) and lead (Pb). Heavy metals occur naturally through volcanic activity, weathering process, and mobilisation deposited in soils (5, 6).

It also may arise through anthropogenic resources such as municipal solid waste (MSW) disposal (5 – 7), atmospheric deposition (8), fertilizer and pesticide use (9), waste water (10), industrial process (11) and mining activities (12). Consumer products such as plastic and pigments dispose in the landfill also may contain heavy metals (13).

Exposure to heavy metals can occur through inhalation of dust, fume or vapour, ingested through food and drink and absorption through skin (15 - 17). These elements are distributed in tissues and organs through absorption and excretion typically occurs primarily through kidneys and digestive tract (16). It has a tendency to persist in some storage sites, such as liver, bones, and kidneys, for long time (17). The International Agency for Research on Cancer (IARC) has classified Cd and its compound as Group 1 which is carcinogenic to human (17). Inhalation to Cd within 8 to 24 hours can cause acute chemical pneumonitis and accidental Cd ingestion can cause gastrointestinal tract irritation. Other chronic effects include renal dysfunction, kidney stones, emphysema, bone pain and anosmia (18, 19).

Atmospheric Cd compounds are transported and deposited onto surface soils and water with minimal transformation in the atmosphere (15). The total global anthropogenic Cd emissions in the mid-1990s were estimated at 3,000 tonnes and decreased about half in Europe and two-thirds in Canada between 1990 –2003 (13). The mean of Cd concentrations in air from the northern Europe in 1980–88 were reported as 0.1 ng/m3 in the remote area, 0.1 to 0.5 ng/m3 in the rural area, 1 to 10 ng/m3 in the urban area and 1-20 ng/m3 in the industrial areas (13).

Exposure to organic dust was reported probable underlies the inflammation mediated by neutrophils that result in increased upper airway inflammation and respiratory symptoms among waste collectors in Netherlands (16). The urban population in the East and South-East Asia were reported exposed to high level of Pb and Cd (21). Several epidemiology studies also had determined the relationship between exposure to Cd and cancer (22 – 23). The assessment done in 1990 to 1993 had estimated 207,350 of the European Union (EU) workers in various occupational setting such as construction, manufacture, metal industries, services, and machinery industries were exposed to Cd (23).

A study on the mortality of a cohort of 926 male workers from a factory engaged in the manufacture of nickel-cadmium batteries in the West Midlands of England has reported significant increase of mortality rate for cancer. The mortality rate was associated with the cancer of pharynx, non-malignant diseases of the respiratory system, and non-malignant diseases of the genitourinary system (24). However, non-significantly increased mortality rate shown for lung and prostate cancer (24). Cd exposure also was reported among tin smelter in United Kingdom (25) and among welders and vehicle operators in China (Cd in blood of welders ranged between 0.2 – 12.5 ug/L) (26). Workers involved with mechanical plating, production of Cd, pigment and battery manufacturing, zinc smelting and refining were also exposed to Cd (19).

Limited studies on Cd exposure among waste collectors were found, because the risk usually being ignored. This study was aimed to fill in this gap by measuring the Cd exposure among municipal solid waste operators through the inhaled dust and the excretion of Cd in human body through urine as the biomarker. Findings of this study provide baseline information on the level of Cd exposure among the waste collectors and the potential health risk.

MATERIALS AND METHODS

Study area and population

The study was conducted in an urban area of Selangor, Malaysia. Approximately 5.9 million people live in this area and previous researches highlighted that major proportion of municipal solid waste in Malaysia is generated by Selangor (27, 28). Landfilling is the major waste disposal method which serves 60–90% of the area. Waste operators hired by the local authority work for eight hours a day and six days per week. Their task is to load the waste into the garbage truck while the truck driver navigate through the streets and operate the hydraulic lift (if any) and transport the waste for final disposal to landfill. Waste compactors and bulldozers were used to spread and compact the waste in the landfill before covered the waste with soil or alternative materials daily to extend the lifespan of the landfill.

Study design and recruitment of respondents

This is a cross sectional study design. Sixty (60) male waste collectors aged between 20 to 50 years old were recruited in this study. The study was conducted between April to June 2013. Most of the workers are unskilled foreign workers and of low socio-economic status. Study objectives, procedures, and possible risks associated with participating in the study were explained to the respondents prior to recruitment and written informed consent was obtained. A face to face interview using a structured questionnaire were performed and respondents socio-demographic background, working information (i.e. duration of work, working hour, previous history of Cd exposure, health symptoms and lifestyle (i.e. smoking habit, alcohol consumption) were recorded. This research has obtained ethical approval from the Medical Research Ethic Committee, Faculty of Medicine and Health Sciences, Universiti Putra Malaysia (UPM/FPSK/100-9/2-JKEUPM (JKPP(U)_Oct(12)34).

The questionnaire was adopted and modified from the Workplace Health and Safety Queensland, Department of Justice and Attorney-General Cadmium health monitoring forms (PN10449 Version 2). Smoking habit, canned food and alcohol consumption contributes to the accumulation of heavy metals in human body (20, 21), thus this information were recorded in the questionnaire. The history of Cd exposure from previous workplace and the employment duration also reflect the Cd level in the urine. Long term exposure to Cd lead to high accumulations in the kidneys (29, 30). For instant, workers exposed to Cd for 250 days and more reflects the current exposure of the pollutant (31).

Urine and respirable dust sample collection

Respondents were given a clean, empty disposable polypropylene containers into which they urinated and collected the urine themselves early in the morning as spot samples. Urine samples were preserved with 5 mL concentrated HNO3 and packed in an insulated container at 4°C for heavy metal and creatinine analysis. The inhaled dust was sampled using air sampling pump (GilAir-3 and GilAir-5) (Model: Gilian). The pump was attached to respondents for 8 hours of their working shift. The air sampling pump was sealed into a sling bag for the comfort of respondents. The cellulose ester membrane filter paper (37 mm in diameter and 0.8um pore) was sealed in the air filter cassette and clipped to the workers shirt within their breathing zone.

Laboratory analysis of cadmium in urine and inhaled dust

Urine samples were filtrated through a filter paper (Whatman no. 1). An aliquot of 5 ml urine was taken in 30-ml flask and diluted five times with 2 % HNO3 and transferred to pre-cleaned polyethylene bottles and kept at 4 °C for Cd analysis (32). The Urinary Creatinine (UACr) was analysed by using Reflotron® Plus creatinine. Measurement of creatinine levels is used as one indicator of kidney function (33).

NIOSH Method 7048 (1994) was used to prepare the sample. The filter was transferred to clean beaker and cut into small pieces. 2 mL of concentrated HNO3 was added to the sample and heated on hotplate (140 °C) until the volume reduced to 0.5 mL. Then, 2 mL of HCl was added and samples were heated on hotplate (400 °C) until the volume reduced to 0.5 mL. The solution was set to cool and 10 mL distilled water was added before transferred to a volumetric flask and dilute to volume with distilled water.

Quantification of Cd in the urine and inhaled dust was carried out using Flame Atomic Absorption Spectrometer (Perkin Elmer AAnalyst 800). Instrument was calibrated by the standards. All the results after calculation of dilution factor were presented in µg/g Cr for urine and mg/m3 for inhaled dust.

Quality assurance and control (QA & QC)

All the containers and glassware were soaked in 10 % HNO3 and washed with distilled water. All samples were analysed in triplicate to minimise the potential error in the analysis. All machines were calibrated followed the standard protocols. Pre-test of the questionnaire was performed to 10% of the total respondents with similar socio-demographic background and the Cronbach's Alpha value was used to measure the reliability of the questionnaire in this study.

Statistical analysis

Data were analyzed using IBM Statistical Package for Social Science (SPSS) version 22.0. Descriptive analysis was used to report the socio-demographic background of respondents and perceived health symptoms. Mann-Whitney and Kruskal Wallis Test were performed to compare the Cd and creatinine level in urine samples by variables. Bivariate analysis was used to determine the correlation between Cd concentration in the inhalation dust with urine and creatinine level. Multiple linear regression model was used to determine the predictor for Cd in urine.

RESULTS

Respondent's background

Table I shows the socio demographic background, respondents lifestyle and occupational exposure. Majority of the respondents aged between 21 to 30 years old (N = 31, 51.7%). They are mainly foreigners from Bangladesh (N = 35, 59%) and Indonesian (N = 20, 33%) and have no formal education (N = 43, 71.67%). Majority of the workers have normal Body Mass Index (BMI) (N = 49, 81.7%) with the average ± standard deviation of 61.75 ± 8.20 kg weight and 1.66 ± 0.49 m height.

Majority of these workers are active smokers (N = 37, 61.7%) and they have been smoking more than 6 years (N = 18, 48.6%) with an average 11 to 15 pieces of cigarette per day (N = 20, 54.1%). One quarter of the workers consumed alcohol (N = 15, 25%) and canned food (N = 14, 23.3%). Majority of them have worked as waste operators for 1 to 5 years (N = 40, 66.7%). They are working for 8 hours per day and 6 days per week. Only 10% (N = 6) of the respondents were exposed to heavy metals in their previous job. Half of the workers (N = 33, 55%) used personal protective equipment (PPE) (safety boots) at work. None of the workers used mask or respirator to protect them from the waste dust.

Perceived health symptoms

The most common perceived health symptom reported by respondents was headache (N=19, 31.7%), excessive sweating (N=11, 18.3%), fever (N=7, 11.7%), coughing (N=6, 10%) and muscle pain (N=5, 8.3%) (Table II). Only one worker has the symptom of irritation nose and throat, shivering muscles, chest and stomach pain. None of the workers have felt the symptom of restlessness, vomiting, nausea, anosmia, anaemia and hyperuricemia.

Table I: The socio-demographic background, lifestyle and workplace exposure history of respondents

	Variables	Total (n = 60) N (%)	Mean (SD)[a]
Age (years)	21 -30	31 (51.7)	
	31 – 40	23 (38.3)	
	41 – 50	6 (10)	
Ethnicity	Malay	3 (5)	
	India	2 (3.3)	
	Bangladesh	35 (59)	
	Indonesia	20 (33)	
Formal education	No education	43 (71.67)	
	Primary	5 (8.3)	
	Lower secondary	6 (10)	
	Upper secondary	6 (10)	
Mean Weight (kg)			61.75 (SD 8.20)
Mean Height (m)			1.66 (SD 0.49)
Mean BMI (kg/m^2)			22.27 (SD 2.55)
BMI (kg/m^2)	Underweight (< 18.5)	4 (6.7)	
	Normal (18.5 – 24.99)	49 (81.7)	
	Overweight (> 25)	7 (11.7)	
Smoking habit	Active smoker	37 (61.7)	
	Never	23 (38.3)	
Years of smoking	1 – 5 years	14 (37.8)	
	6 – 10 years	18 (48.6)	
	>11 years	5 (13.6)	
Number of cigarette per day	<5	4 (10.8)	
	6 - 10	10 (27.0)	
	11 - 15	20 (54.1)	
	>16	3 (8.1)	
Alcohol consumption	Yes	15 (25)	
Canned food consumption	Yes	14 (23.3)	
Length of employment (years)	< 6 months	8 (13.3)	
	6 months - 1 year	9 (15.0)	
	1 – 5 years	40 (66.7)	
	6 – 10 years	3 (5.0)	
Exposure to Cd in previous job	Yes	6 (10)	
Use PPE	Yes	33 (55%)	

[a] SD = Standard deviation

(N=6, 10%) and muscle pain (N=5, 8.3%) (Table II). Only one worker has the symptom of irritation nose and throat, shivering muscles, chest and stomach pain. None of the workers have felt the symptom of restlessness, vomiting, nausea, anosmia, anaemia and hyperuricemia.

Urine-Cd, inhaled-Cd and creatinine level

The mean ± standard deviation (SD) of Cd in inhaled dust was 0.59 ± 50.27 μg/m3 and ranged between 0.42 to 1.03 μg/m3. The value was within the PEL 8hr TWA of Cd compound in respirable fraction standard by DOSH (19). The mean ± SD of Cd in urine (0.015 ± 0.0097 μg/g Cr) was lower than the safe limit of 5 μg/g (19) and ranged between 0.0015 to 0.532 μg/g Cr. The mean ± SD of creatinine level was 173.59 ± 50.27 mg/dl and ranged between 38.72 to 210.0 mg/dl. This value is within the standard range of 20 to 350 mg/dl provided by the WHO (20). The correlation test found no significant relationship between Cd concentration in the inhalation dust and in urine (r = -0.043, p = 0.746) and creatinine level (r = 0.070, p = 0.595). Significant negative association was determined between Cd in urine and creatinine level (r = -0.778, p <0.001), indicate that high level of Cd in urine resulted in low

Table II: The perceived health symptoms associated with Cd exposure

Variables	Waste operators (n=51) N (%)
	Yes
Cough	6 (10)
Irritation of nose and throat	1(1.7)
Headache	19(31.7)
Excessive sweating	11(18.3)
Fever	7(11.7)
Shivering muscle	1(1.7)
Restlessness	-
Vomit/nausea	-
Chest pain	1(1.7)
Stomach pain	1(1.7)
Muscle pain	5(8.3)
Anosmia	-
Anaemia	-
Hyperucemia	-

Note: 51 respondents (85.1%) answer yes to the symptoms, while 9 (14.9%) respondents answer no.

level of creatinine level.

The distribution of Urine-Cd and creatinine level by variables

Table III shows the comparison of Cd and creatinine level in urine samples by variables. The Cd level in urine was significantly difference by years of smoking ($Z = 8.168$, $p = 0.006$), number of cigarette per day ($Z = 4.54$, $p = 0.04$) and type of canned food ($X2 = 4.436$, $p = 0.007$). The creatinine level was significantly difference by years of smoking ($Z = 5.399$, $p = 0.02$) and type of canned food consumption ($X2 = 2.890$, $p = 0.043$).

Multivariate analysis of Cd in urine

Multiple regression analyses were conducted to examine the relationship between Cd in urine and various potential predictors. Table IV summarizes the analysis results. The multiple regression model with all three predictors produced R = 0.216 $F(3,56) = 5.150$, $p < 0.05$. The result shows smoking and years of smoking are positive significant predictors for the Cd in urine. The result indicates that smokers and years of smoking tend to have higher Cd in their urine.

Table III: Cd and creatinine level in urine [a]

	Variables	Mean Cd in urine	Z (p-value)	Mean Creatinine	Z (p-value)
Age (years)	Young adult (20-40)	0.0154	0.026 (0.873)	175.00	0.423 (0.518)
	Middle adult (40 – 60)	0.0148		160.87	
Ethnicity [b]	Malay (Local)	0.0102	1.337 (0.271)	210.0	1.052 (0.377)
	India (Local)	0.0159		157.5	
	Bangladesh	0.0174		166.29	
	Indonesia	0.0126		182.51	
BMI (kg/m^2)	Normal BMI	0.0161	2.264 (0.138)	171.21	1.022 (0.316)
	Overweight	0.0102		191.64	
Smoking	Yes	0.0149	0.253 (0.617)	177.18	0.488 (0.488)
	No	0.0162		167.81	
Years of smoking	< 5 years	0.0215	8.168 (0.006)*	147.22	5.399 (0.024)*
	> 5 years	0.0135		181.62	
Number of cigarette per day	< 5	0.0251	4.540 (0.04)*	130.18	3.322 (0.074)
	6 -15	0.0147		176.69	
Alcohol consumption	Yes	0.0122	2.177 (0.146)	183.89	0.837 (0.364)
	No	0.0164		170.16	
Canned food consumption	Yes	0.0161	0.112 (0.739)	166.63	0.346 (0.558)
	No	0.0151		175.71	
Types of canned food [b]	Meat / fish	0.0143	4.436 (0.007)*	164.94	2.890 (0.043)*
	Drinks	0.0132		186.34	
	Milk / sauces	0.0476		138.72	
Length of employment (years)	< 1 yr	0.0151	0.008 (0.927)	171.39	0.017 (0.896)
	> 1 yr	0.0154		173.93	
Exposure to Cd in previous job	Yes	0.0176	0.355 (0.554)	167.32	0.102 (0.750)
	No	0.0151		174.28	

Note: Mean Cd in urine (µg/g Cr); Mean Creatinine (mg/dl); *significant difference with $p < 0.05$, [a] Mann-Whitney test, [b] Kruskal Wallis Test, reporting X^2 value

Table IV: Multiple linear regression model of predictor for Cd in urine

Model	Coefficient	Std. Error	t	p-value
Intercept	.039	.009	4.217	.000
Smoking	.006	.003	2.172	.034
Number of cigarette / day	-.008	.005	-1.555	.126
Years of smoking	-.009	.003	-3.023	.004
R2 = ….R2 = ….				

Note: Predictors : Cd urine

DISCUSSION

This study measure Cd exposure among municipal solid waste collectors through inhaled dust and urine assessment. According to the Occupational safety and Health (Use and Standard of Exposure of Chemicals Hazardous to Health) (USECHH) Regulations, (2000), exposure to Cd in workplace is required for a medical surveillance (19). The permissible exposure limit (PEL) of 8-hour time-weighted average (TWA) for elemental Cd is 0.01 mg/m^3 while the respirable Cd compound is 0.002 mg/m3. A medical surveillance program is needed for employees who are exposed at or above > 50% of PEL or where there is significant risk of absorbing Cd (19). The biological exposure Indices (BEI) for Cd in urine is 5 µg/g Cr.

Results of this study show that Cd concentration in the inhaled dust was within the Permissible Exposure Limit (PEL) (0.002 mg/m3) of USECHH Regulations, (2000) but slightly higher than generally reported in the ambient air of rural (between 0.1 to 5 ng/m^3) and urban areas (2 to 15 ng/m^3) (20). Previous study also had reported high Cd in industrial areas within a range of 15 to 150 ng/m^3 (34). Furthermore, Cd concentration in urine was also below than the PEL value of USECHH Regulations, (2000) which medical surveillance program does not required for these workers.

Many of the previous studies have found that Cd exposure has a significant effect on the kidney (4, 21, 35). It can cause disturbance in calcium metabolism and the formation of kidney stones (20). The first sign of renal abnormalities occur at 2 µg/g creatinine. At urinary Cd levels of 4 µg/g Cr, enzymes such as N-acetyl-B-glucosaminidase (NAG) are elevated in urine and signs of glomerular damage including increased albumin in the urine and decrease in glomerular filtration rate can be seen. In the final stages of Cd nephropathy, glycosuria, wasting of calcium and phosphate, and altered calcium metabolism with secondary effects on the skeleton of osteoporosis and osteomalacia can be seen (4). The threshold value of renal damage occurred at urinary Cd levels of 2-4 nmol/mmol Cr (36). Urine Cd level of 1 nmol/mmol Cr had a threefold risk of increased ά-1 microglobulin (37). However, low level of Cd exposure is yet to be scientifically proven associated with the early subclinical changes in kidney biomarkers (38).

The level or urinary creatinine in this study was within the normal range of 20-350 mg/dl. The analysis of urinary creatinine is an indicator of kidney function and renal health. The urinary creatinine level was related to large muscle mass among waste collector. Creatinine is a muscle breakdown product, and people with large muscle mass excrete more creatinine in urine and creatinine less than 20 mg/dl is considered as diluted (39). Increased level of creatinine signifies the problem of poor performing kidneys (40). This possibly related to Cd accumulation in the kidneys that induce kidney damage (37). The risk of kidney disease among worker will increase with the exposure to the airborne Cd greater than 300 mg/m3, urine Cd more than 10 µg/g Cr and Cd in the renal cortex of 200 ppm (41).

The correlation between Cd concentration and the creatinine level in this study shows a negative association, which indicate high level of Cd in urine resulted in low creatinine level. This result highlights the potentially kidney outcomes between urine biomarkers and kidney function when urine creatinine is used. However, this study has limited ability to explain the finding as other factors such as age, race, gender and body size of the population may also influence the result. Further assessment using glomerular filtration rate (GFR) perhaps can be used to estimates the kidney health range which is relevant for most occupational populations.

The waste operators in this study have minimal health symptoms. The general perceived health symptoms reported was headache, excessive sweating, fever and coughing. The health effects are generally associated with the exposure routes, the pollutant concentration, the duration or frequency of exposure and how the person's body responds (17). Exposure to Cd for 14 days or less may produce acute effect, while exposure to 365 days and more may create chronic health effects. The intermediate effects may occur at the exposure between 15 to 365 days (15). Acute exposure to Cd through inhalation may induce symptoms such as flu with chills, fever and muscle pain, and at the later stage can cause lung damage, shortness of breath, chest pain and cough (17). Meanwhile, long term exposure to Cd may cause kidney disease and weak bones (42). There is no definite connection between these symptoms with exposure to Cd or vice versa. Therefore, a more in-depth study in the future needs to be carried out to prove the probability of the relationship.

Correlation test in this study was unable to detect significant relationship between the concentrations of Cd in the respirable dust and in urine. This is probably because; the accumulation of Cd in urine was determined by many of other factors such as age, smoking, body mass and canned food consumption (30). Previous studies also had reported the half-life of Cd in blood and urine is between 10 to 30 years (43) in which 5 to 15 years in liver, 10 to 30 years in kidney and 30 years in muscle (44).

The regression results in this study show the influence of smoking behaviour with the level of Cd in urine and creatinine level. Cigarette smoking increased blood Cd levels and the total Cd body burden (45). Smoker had approximately 1 to 5 times of Cd concentration in urine and blood higher than non-smoker. Previous research has reported, a single cigarette was determined to have 1-2 µg of Cd and 10% of inhaled Cd is absorbed by the lungs (46). If the Cd urine level reaches 10 µg/g Cr, Cd in the renal cortex is considered high and will reach a critical level (200 µg/g) (46). Cigarette is a significant source of Cd exposure as the tobacco leaves naturally accumulate large amounts of Cd. It was estimated that smokers are exposed to 1.7 µg Cd per cigarette and 10% of this concentration is inhaled through smoke (47).

This study fills in the research gap about the association of Cd exposure through inhalation and its accumulation in human body among waste operators through biomarkers assessment. The strength of this study is the use of urine as biological sample in the assessment. Urine and blood sample are non-invasive biological sample that frequently used to estimate the recent and past exposure of toxic element (48). Previous studies have shown a relationship between urinary cadmium excretion and the total body burden. Urinary Cd is considered to be mainly in equilibrium with body burden before renal damage occurs. It is a good indicator of body burden for occupation-exposed people than other biomarkers such as blood, hair or nails (26). Urinary Cd has been shown to accurately reflect the amount of Cd in the body from recent and past exposure (45). This is because, Cd excretion with the urine is dependent on the existing body burden, exposure situation and degree of kidney damage (20). A level of 200 µg/g is considered to be the critical Cd concentration in the renal cortex and corresponds to Cd excretion with the urine of about 5 to 10 µg/g creatinine. Daily excretion of Cd is about 0.005 % to 0.02 % of the total body burden and this is corresponds to a biological half-life of 10 to 20 years. Human body can change most of the Cd to a less toxic form, but too much Cd can overload the ability of liver and kidney (20).

This study has observed several limitations which need improvement in the future research. Small sample size in this study may reduce the ability to observe significant cause-effect relationship. Small sample size in this study was due to lack of cooperation from the workers. Due to time constraint, the duration for respondent's recruitment cannot be extended for a better response rate. Therefore, it was recommended for future study to consider the extension of sampling duration to get a better sample size. This study only analysed one heavy metals which is Cd. A single element analysis may not adequately represent the mix exposures of metal from different types. Therefore, future study was recommended to consider multi elements analysis to have a better discussion of the results.

CONCLUSION

The municipal waste operators were exposed to low Cd concentration through inhalation. The concentration of Cd in the respirable dust (0.59 ± 50.27 µg/m3) and urine (0.015 ± 0.0097 µg/g Cr) were within the Permissible Exposure Limit (PEL) with normal urinary creatinine level (173.59 ± 50.27 mg/dl). Medical health surveillance was not required for these workers since the exposure to Cd was within an acceptable range. There is no significant relationship was obtained between concentrations of Cd in respirable dust and urine. Smoking is the main predictor of urine-Cd concentration in this study ($R = 0.216$ $F(3,56) = 5.150$, $p < 0.05$).

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