Available online at www.sciencedirect.com
ScienceDirect
Journal of Environmental Sciences 2011, 23(2) 336–345
JOURNAL OF ENVIRONMENTAL SCIENCES
ISSN 1001-0742
CN 11-2629/X
www.jesc.ac.cn

Risk assessment for the daily intake of polycyclic aromatic hydrocarbons from the ingestion of cockle (*Anadara granosa*) and exposure to contaminated water and sediments along the west coast of Peninsular Malaysia

Seiedeh Aghileh Mirsadeghi[1], Mohamad Pauzi Zakaria[1,*], Chee Kong Yap[2], Azadeh Shahbazi[1]

1. Centre of Excellence for Environmental Forensics, Faculty of Environmental Studies, University Putra Malaysia, 43400 UPM, Serdang, Selangor, Malaysia. E-mail: agh_env@yahoo.com
2. Faculty of Science, Department of Biology, University Putra Malaysia, 43400 UPM, Serdang, Selangor, Malaysia

Received 24 January 2010; revised 23 May 2010; accepted 09 July 2010

Abstract

The concentration of carcinogenic polycyclic aromatic hydrocarbons (c-PAHs) present in the sediment and water of Peninsular Malaysia as well as in the cockle *Anadara granosa* was investigated. Samples were extracted and analysed with gas chromatography-mass spectrometry. The concentrations of total carcinogenic polycyclic aromatic hydrocarbons (t-PAHs) were measured between 0.80 ± 0.04 to 162.96 ± 14.74 ng/g wet weight (ww) in sediment, between 21.85 ± 2.18 to 76.2 ± 10.82 ng/L in water samples and between 3.34 ± 0.77 to 46.85 ± 5.50 ng/g ww in the cockle tissue. The risk assessment of probable human carcinogens in the Group B2 PAHs was calculated and assessed in accordance with the standards of the United States Environmental Protection Agency (US EPA). Case I in the toxicity assessment analysed the cancer risk to consumers of Malaysian blood cockle. Case II assessed the risk of cancer from exposure to PAHs from multiple pathways. The average cancer risk of case I and case II were found to be classifiable as unsafe according to the US EPA standard. The cancer risk due to c-PAHs acquired by the ingestion of blood cockle was $(8.82 \pm 0.54) \times 10^{-6}$ to $(2.67 \pm 0.06) \times 10^{-2}$, higher than the US EPA risk management criterion. The non-cancer risks associated with multiple pathways in Kuala Gula, Kuala Juru and Kuala Perlis were higher than the US EPA safe level, but the non-cancer risk for eating blood cockle was below the level of US EPA concern.

Key words: toxicity assessment; carcinogenic polycyclic aromatic hydrocarbons; group B2 of US EPA; *Anadara granosa*

DOI: 10.1016/S1001-0742(10)60411-1

Citation: Mirsadeghi S A, Zakaria M P, Yap C K, Shahbazi A, 2011. Risk assessment for the daily intake of polycyclic aromatic hydrocarbons from the ingestion of cockle (*Anadara granosa*) and exposure to contaminated water and sediments along the west coast of Peninsular, Malaysia. Journal of Environmental Sciences, 23(2): 336–345

Introduction

Polycyclic aromatic hydrocarbons (PAHs) are persistent organic chemicals containing two or more fused aromatic benzene rings (Neff, 1979). Aquatic ecosystems are one of the major sinks of PAHs, which tend to adsorb onto suspended particulate matter (SPM) because of their lipophilic characteristics and to settle in the sediment (Shi et al., 2007). Stranded or remobilised PAHs attached to suspended sediments can become available to filter feeders, particularly the mussels, oysters and clams that inhabit the inter-tidal and shallow subtidal beds (Yender and Christine, 2002). Like other deposit/filter feeders, cockles can bioaccumulate PAHs actively and exhibit them at even higher concentrations than the background contamination in their environment (Baumard et al., 1999; Moradi, 2001; Cheevaporn and Menasveta, 2003; Froun et al., 2007; Isobe et al., 2007; Vinas et al., 2009). PAHs can also be concentrated in the human body following the consumption of contaminated shellfish and cockles. Once in the body, PAHs may pose major human health concerns (Shor et al., 2004; Nikolaou et al., 2009).

Oil tanker transportation in the Straits of Malacca, the general use of petroleum and growing industrial activities near coastal areas and along rivers have seriously increased PAH contamination in the sea and in the coastal area of Malaysia (Zakaria et al., 2001, 2002; Zakaria and Mahat, 2006; Sakari et al., 2008b; Bakhtiari et al., 2009). Contamination at high levels could affect the socio-economic and health status of the population who depend on aquaculture and coastal resources for their food and livelihoods (Ibrahim, 1995; Abdullah, 1997). Blood cockle, *Anadara granosa*, is an important protein source in Southeast Asia (Chan et al., 2002) that is commercially cultured in the tidal mudflats along the western coast of Peninsular

* Corresponding author. E-mail: mpauzi@env.upm.edu.my

Malaysia. Blood cockle is a good revenue source for the local population (Ibrahim, 1995). An investigation of global contaminant patterns in these oceanic organisms is required; contamination in this region of Malaysia, however, requires special attention.

A five-step method has been developed by the United States Environmental Protection Agency (US EPA) to assess the risk associated with hazardous compounds. Data collection, toxicity assessment, exposure assessment, risk characterisation and risk management are the major steps of this risk assessment process. According to the US EPA, contaminated environments pose both ecological and human health risks (Nikolaou et al., 2009). The US EPA risk assessment method can be applied to both carcinogens and non-carcinogens by assessing ingestion via the gastrointestinal tract, skin contact and inhalation. The overall assessment includes the quantitative evaluation of the intensity, frequency and duration of chemical contact (US EPA, 1991; Syracuse Research Corporation, 2002; ENSR Corporation, 2005; Mallika et al., 2008). The purpose of the risk characterisation is to provide quantitative estimates of the potential carcinogenic risk in humans that is associated with exposure to chemicals.

The US EPA has adopted several approaches for evaluating the toxicity and cancer potency of PAHs. Substances classified into Group B2 by the US EPA include Benzo(a)pyrene (BaPy), Benzo(a)anthracene (BAnt), Chrysene (Chry), Benzo(b)fluoranthene (BbFl), Benzo(k)fluoranthene (BkFl), Dibenzo(a,h) anthracene (DAnt), and Indeno(1,2,3-cd)pyrene (IPy). There is sufficient evidence of the carcinogenicity of Group B2 substances towards animals, but there is inadequate or no evidence of their carcinogenicity toward humans (Collins et al., 1998; US EPA, 2000; Scientific Committee on Food, 2002). PAH exposure in animals is associated with such toxic effects as bone marrow toxicity, reproductive toxicity, cardiovascular toxicity, immune system suppression and liver toxicity (Collins et al., 1998). The most serious toxic effect related to exposure to PAHs in humans is cancer.

Two types of risk were evaluated in this assessment. The first was the total cancer risk associated with the constituents considered: water, sediment and blood cockle consumption. The second type of risk was the non-cancer health hazard associated with exposure to non-carcinogenic constituents through various pathways. The objectives of this research were: (1) to determine the concentrations of potentially carcinogenic PAHs (c-PAHs) (BaPy, BaAnth, Chry, BkFluo and DAnt) in the blood cockle species *A. granosa*, in the water and sediment; (2) to assess the cancer risk and the non-cancer hazard index of c-PAHs through gastrointestinal and skin exposure to sediments; and (3) to assess the cancer risk and the non-cancer hazard index from exposure to c-PAHs through the ingestion of blood cockle.

1 Materials and methods

1.1 Sample collection

Samples were collected from mudflat aquaculture plots in the Sungai Buloh (A), Pantai Remis (B), Sungai Haji Durani (C), Kuala Gula (D), Kuala Juru (E) and Kuala Perlis (F) extended mudflats located on the west coast of Peninsular Malaysia. The location of the sampling sites is shown in Fig. 1. The experimental design required three replicates from each sampling area. Then, samples from stations A, B, C were collected from 20th to 25th January 2008 and samples from stations D, E, F were collected from 23rd to 25th March 2008. Samples were stored in a cooler box with dry ice during transfer to the laboratory. The samples were then frozen at –10°C prior to further analysis (Chouksey et al., 2004). If water and sediment samples were not analysed within two hr of collection, prior to storage they were acidified to a pH < 2 with HCl or with H_2SO_4 (Tong et al., 1999) to stop the microbial degradation of the target compounds (McGroddy and Farrington, 1995). The water samples were extracted immediately after sampling.

1.2 Sediment

Surface sediment samples were taken from a depth of 20 cm using a Van Veen grab (area 0.04 m^2). Organisms and shell debris were removed by hand, and the finer particles from the top sediment layer were used for PAH analysis (Syracuse Research Corporation, 2002; Chouksey et al., 2004).

Fig. 1 Location of sampling sites along the west coast of Peninsular Malaysia.

1.3 Total soft tissue of blood cockle

Forty individual marine blood cockles of approximately the same size and age (4–6 cm in length) were randomly collected from the aqua culture areas. The blood cockles were collected once only between January 20 and March 25, 2008, from all six sites to avoid sampling during the spawning season.

1.4 Water

Water samples were collected systematically from the surface water column (0–20 cm) in a fixed location during one tidal cycle. The surface waters were sampled using a 5 l Niskin bottle (Tong et al., 1999) while the sediment and blood cockle samples were being taken.

1.5 Data analysis

Prior to extraction of the samples, sodium sulphate (Na_2SO_4) was baked in a muffle furnace at 380°C for 4 hr to remove water content and then cooled in desiccators until use for drying samples. Total soft tissues of blood cockle and the sediment samples were dried with sodium sulphate anhydrous, spiked with 50 µL of 10 µg/g deuterated surrogates (naphthalene-d_8, anthracene-d_{10}, benzo(a)anthracene-d_{12} and perylene-d_{12}) and 50 µL of a 10 µg/g internal injection standard (*p*-terphenyl-d_{14}) (Siddiqi et al., 2009). The samples were then subjected to soxhlet extraction with 250 mL dichloromethane (DCM) for 8 hr (Anyakora et al., 2005; Culotta et al., 2006; Nikolaou et al., 2009). Extracted samples were purified and fractionated into an aliphatic and an aromatic fraction through two-step silica gel column chromatography (Zakaria et al., 2001; Yim et al., 2005; Boonyatumanond et al., 2006).

Because of the hydrophobic nature of PAHs, these compounds bond to suspended particulate matter in the water column (McGroddy and Farrington, 1995). Therefore, water samples were filtered through GF/F Whatman filter paper and then the filtered suspended particulate matter (SPM) together with filter paper were dried with sodium sulphate anhydrous and extracted in the same manner as the other samples.

Samples were collected after column chromatography and concentrated to a final volume to preserve them until analysis with gas chromatography-mass spectrometry (GC-MS). The extracts were analysed in a 6890 N Agilent gas chromatograph coupled to a 5973 N Agilent mass spectrometer (Agilent Technologies, USA) with a 7683 Agilent auto sampler. The carrier gas was helium at a constant flow rate of 1 mL/min at a constant pressure of 100 kg/cm^2. Two microlitres of the sample were injected into a HP-5 fused silica capillary column (30 m × 0.25 mm i.d. and 0.25 µm microfilm thickness, Agilent Technologies) in the splitless mode at 270°C. The GC temperature program was: 70°C for 2 min then programmed to rise at 30°C/min to 150°C, then at 4°C/min to 310°C and finally held for 10 min. Measurements were performed in the selected ion monitoring (SIM) mode. The GC-MS operating condition was 70 eV ionisation potential with the source at 200°C for 1.4 scans/sec, with the electron multiplier at 2000 V and the dwell time set at 40 msec. The interface temperature was 290°C and the injector temperature was maintained at 270°C (Baumard et al., 1999; Yim et al., 2005; Sakari et al., 2008a). The identification and quantification of the PAH compounds were achieved using ChemStation® software and were based on matching their retention time with a mixture of PAH standards. All of the PAHs were reported as wet weight (ww). Total organic carbon (TOC) was measured in sediments by standard methods, using carbon determination equipment Model LECO CR-412 (LECO Corporation, USA) with infrared absorption detection method.

1.6 Quality control and quality assurance

The following quality control measures were performed: (1) the retention time and the abundance of quantification ions/confirmation ions of each individual PAH compound was compared to reliable PAH standards; (2) blanks were processed together with collected samples (Arias et al., 2009), and then analysis of a reagent blank demonstrated that the analytical system and glassware were free of contamination; (3) limits of detection (LODs) were estimated as the average signal of the blanks (n = 3) plus three times the standard deviation of the signals of the blanks (Abdullah et al., 1996; Chouksey et al., 2004; Cortazar et al., 2008); (4) quantification was performed by the internal standard method using a 16 PAH reference material mixture (16 PAHs, paratherphenyl D14 as Internal Injection Standard, and surrogates); (5) three-point internal calibration curves were built in the range of 2.0–10.0 µg/L for PAHs with correlation coefficients for calibration curves all higher than 0.993. The recovery of surrogates generally ranged from 60% to 110% of the spiked concentration. Samples with recoveries below 60% were re-analyzed. PAH concentrations were corrected using their recoveries (Cortazar et al., 2008). All samples were analyzed in triplicates, and the relative standard deviation (n =3) was less than 20%. The condition of the GC-MS was checked every day.

1.7 Risk assessment calculation

Risk assessment of exposure to the body and intake were calculated using the equations listed in Table 1 and the variables explained in Table 2. The calculations in this study were based on exposure factors listed in the literature and were supplemented by assumptions about the duration of the exposure time, intake, frequency in the sediment and water, and skin contact as there is no appropriate data in this region. The substitution values that are recommended in these formulas include 70 kg as the average body weight of an adult, 5700 cm^2 for the body surface of an adult and 2800 cm^2 for the body surface area of children (US EPA, 2009). As there is no data for children in tropical countries, the maximum consumption rate was chosen from other studies (Ministry of Health Malaysia, 2003) because children are more sensitive than adults and assessment should consider the safe range rather than maximum acceptable range. Non-cancer risk was

Table 1 Equations used for the calculation of body exposure through various routes and the ingestion of blood cockle

Parameter	Equation	Reference
Sediment absorbed dose	DAD = C × SA × EF × ED × ET × CF × AF/BW × AT	Integrated Environmental Management Inc. and Riverfront Environmental, 2007
Sediment ingestion dose	CDI = C × IR × EF × ED × FI × CF/ BW × AT	Integrated Environmental Management Inc. and Riverfront Environmental, 2007
Water absorbed dose	ADD = C × SA × PC × ET × EF × ED × AAF_d × CF/ BW × AT	US EPA, 1991
Cockle absorbed dose	CDI = C × (IR/BW) × (EF × ED/AT) × CF	Syracuse Research Corporation, 2002
Hazard Quotient of non-cancer	HQ = CDI/RfD, HQ = DAD/RfD	Syracuse Research Corporation, 2002
Hazard Index of non-cancer (HI)	∑HQs for each individual constituent	ENSR Corporation, 2005
Cancer risk	CR = 1 – exp [$-CDI_L \times (SF_{BaP} \times PEF)$]	Syracuse Research Corporation, 2002

The explanation of variables in equations are in Table 2.

Table 2 Variables of exposure assumption

Parameter	Value	Comments
Concentration (C) (ng/g)	Variable	Mean value
Ingestion rate (IR), sediment – US. standard (mg/day)	Child: 200 Adult: 100	
Conversion factor (CF)		Depend on units of equation
Ingestion rate (IR) – blood cockle only (g/day)	Peninsular Malaysia: 4.92 Malay: 5.36 Chinese: 3.10 Indian: 3.61 Child: 3.55 International adult: 8.01	IRs for Malaysians were obtained from Ministry health of Malaysia; international IRs were chosen from literature as default for countries which import Malaysian cockle as there is no established data for those countries.
Exposure frequency (EF) (day/yr)	Sediment and water: 104 Cockle: 365	EF = 0.28
Exposure duration (ED) (yr)	20 adults 6 children	Number of years using beach
Body weight (BW) (kg)	Child: 16 Adult: 70	
Averaging time (AT) (day)	25,550	70 years, Standard default for life expectancy
Skin surface area available for contact with sediment (SA) (cm^2/day)	Adult: 5700 Child: 2800	Estimating 10% of whole body
Exposure time for dermal contact with soil (ET) (hr/day)	Sediment and water: 1	
Soil to skin adherence factor (AF) (mg/cm^2)	0.07 Resident adults, 0.2 children and worker	
Absorption factor (ABS) (unitless)	chemical-specific	
Dermal permeability constant (PC) (cm/hr)		
Minimal risk level (MRL) or oral reference dose (RfD) (mg/(kg·day))	Contaminant- specific	This value for naphthalene is employed because it is the lowest oral RfD for a PAH-class (2E-02)
Dermal absorption adjustment (AAFd) (unitless)	Constituent -specific	10 for PAHs, water factor
Chronic daily intake (CDI) (mg/(kg·day))		
Dermal absorbed dose (DAD) (mg/(kg·day))		
Hazard quotient (HQ) (unitless)	Variable	HI: Sum of HQ
Oral slope factor for BaPy (SFBaPy) (mg/(kg·day))	7.3	An upper-bound estimate of the probability that individual will develop cancer over a lifetime as a consequence of exposure to a given dose of a specific carcinogen
Oral slope factor for c-PAHs (SF_x) (mg/(kg·day))	Variable	Calculated based on PEF
Potency equivalence Factor (PEF) (unitless)	Contaminant-specific	Minnesota Department of Health, 2004

This table adapted from: Syracuse Research Corporation, 2002; Ministry of Health Malaysia, 2003; ENSR Corporation, 2005; Integrated Environmental Management Inc. and Riverfront Environmental, 2007; Roberts and Goff, 2008; IRIS, 2009; US EPA, 2009 measured using a hazard quotient (HQ). An HQ is the ratio of the exposure level of a single substance over a specified time period (e.g., subchronic). The HQ is calculated with respect to a reference dose (or concentration) of a substance over a similar exposure period that is derived primarily through the use of exposure assumptions and reference doses (RfDs) provided in the US EPA guidance documents. RfDs, however, are not available for the potentially c-PAHs. Surrogate RfDs were applied based on structural similarities between the potentially c-PAHs and the non-carcinogenic PAHs (ENSR Corporation, 2005). In many risk assessments of complex pollutant mixtures, all of the c-PAHs have been considered to be as carcinogenic as BaPy. The BaPy compound was chosen as the primary representative of the carcinogenic class of PAHs because of the relatively large amount of toxicological data available and the known and frequent human exposure. When an appropriate toxicity value was not identified for a c-PAH mixture, Potency Equivalency Factors (PEFs) were used. The PEFs were calculate by adjusting the BaPy toxicity value with their relative potency to BaPy (Washington State Department of Ecology, 2007; SRP, 2009).

2 Results and discussion

2.1 Concentration of carcinogenic-PAHs in sediment, water and total soft tissues of blood cockle

Some criteria have been established from toxicity assessment studies of PAHs based on national-level studies, but no international human criteria have been established based on the consumption of aquatic organisms, water or sediment quality (Yender and Christine, 2002). Due to the lack of regulatory criteria for the Peninsular Malaysia region, we compare the results to the national criteria from studies conducted elsewhere. To protect aquatic life from the harmful effects of PAHs that are found in sediment, the concentration of sedimentary criteria were calculated for Chry, BAnt, and BaPy among c-PAHs in marine and fresh water as recommended by O'Riordan (1993). There are no established sedimentary criteria for other c-PAHs from Group B2 in the sediment. Brackish water also does not have any c-PAHs criteria. Almost all of the SPM and sediment samples from the extended mudflats in Malaysia are brackish; neither marine nor fresh water sedimentary criteria are directly applicable.

Table 3 displays the concentrations of individual and total c-PAHs in the sediment, water (SPM) and sedimentary criteria. The concentrations of the criteria at all six stations are lower than the recommended maximum values for the three compounds in the sediment. The water samples taken from all three stations analysed in this study showed lower values compared to the values determined for the health of aquatic organisms. There are no established international water quality criteria for PAHs that can be used to protect aquatic communities. US EPA has recommended national water-quality criteria for some of the PAHs, such as BaPy, BAnt, BkFl, and DAnt, to protect against human health effects. The national recommended water-quality criterion used by the US EPA for BaPy, BAnt, BkFl and DAnt for consumption of water and organisms that live in the water is 4.4 ng/L; for organisms alone, the value is 49

Table 3 Concentrations of individual carcinogenic PAHs in sediment and blood cockle

Station	Matrix	TOC (mg/L)	Chry (ng/g)	BAnt (ng/g)	BkFl (ng/g)	BaPy (ng/g)	DAnt (ng/g)	IPy (ng/g)	BbFl (ng/g)	t-PAHs (ng/g)
Pantai Remis	Sediment	–	0.80 ± 0.04	n.d.[g]	n.d.	n.d.	n.d.	n.a. [h]	n.a.	0.80 ± 0.04
	Water	2.02 ± 0.08	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Criteria	–	F: NR M: 410	F: 410 M: NR	–	F: 120 M: 120	–	–	–	–
Sungai Haji Durani	Tissue	–	3.34 ± 0.77	n.d.	n.d.	n.d.	n.d.	n.a	n.a.	3.34 ± 0.77
	Sediment	–	0.20 ± 0.03	7.02 ± 1.16	13.75 ± 2.93	9.69 ± 1.50	10.14 ± 1.82	n.a.	n.a.	40.80 ± 2.21
	Water	0.76 ± 0.22	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Criteria	–	F: NR M: 120	F: 120 M: NR	–	F: 400 M: 400	–	–	–	–
Sungai Buloh	Tissue	–	18.02 ± 2.56	1.20 ± 0.09	8.38 ± 2.13	2.42 ± 1.40	n.d.	n.a.	n.a.	28.49 ± 3.17
	Sediment	–	39.55 ± 7.95	24.71 ± 4.99	76.52 ± 0.84	5.56 ± 0.86	16.61 ± 0.93	n.a.	n.a.	162.96 ± 14.74
	Water	1.84 ± 0.24	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.	n.a.
	Criteria	–	F: NR M: 420	F: 420 M: NR	–	F: 130 M: 130	–	–	–	–
Kuala Gula	Tissue	–	15.38 ± 3.28	14.43 ± 3.28	7.96 ± 2.30	n.d.	5.28 ± 0.47	n.a.	n.a.	43.06 ± 4.00
	Sediment	–	8.79 ± 1.88	n.d.	12.77 ± 2.62	n.d.	n.d.	n.a.	n.a.	21.57 ± 0.31
	Water	2.78 ± 0.11	28.02 ± 0.62	n.d.	18.01 ± 0.83	10.55 ± 0.87	n.d.	n.a.	n.a.	56.58 ± 0.27
	Criteria	–	F: NR M: 560	F: 560 M: NR	–	F: 170 M: 170	–	–	–	–
Kuala Perlis	Tissue	–	3.50 ± 0.87	0.98 ± 0.05	1.32 ± 0.28	1.00 ± 0.22	n.d.	n.a.	n.a.	6.85 ± 1.42
	Sediment	–	15.34 ± 2.82	n.d.	47.57 ± 9.83	1.85 ± 0.12	n.d.	n.a.	n.a.	64.76 ± 14.53
	Water	1.98 ± 0.22	3.04 ± 0.64	n.d.	18.82 ± 3.73	n.d.	n.d.	n.a.	n.a.	21.85 ± 2.18
	Criteria	–	F: NR M: 400	F: 400 M: NR	–	F: 120 M: 120	–	–	–	–
Kuala Juru	Tissue	–	5.55 ± 0.34	n.d.	n.d.	n.d.	n.d.	n.a.	n.a.	5.55 ± 0.34
	Sediment	–	27.98 ± 2.62	n.d.	16.68 ± 2.27	11.53 ± 1.82	n.d.	n.a.	n.a.	56.20 ± 5.43
	Water	1.05 ± 0.22	5.29 ± 0.33	n.d.	59.29 ± 14.48	11.64 ± 2.93	n.d.	n.a.	n.a.	76.20 ± 10.82
	Criteria	–	F: NR M: 300	F: 300 M: NR	–	F: 90 M: 90	–	–	–	–
	Tissue	–	17.80 ± 7.43	n.d.	22.39 ± 2.92	6.66 ± 0.53	n.d.	n.a.	n.a.	46.85 ± 5.50

Data are expressed as mean ± SD (n = 3). n.d.: not detected; n.a.: not analysed.
F: fresh water sedimentary criteria based on organic carbon of each station; M: marine water sedimentary criteria based on organic carbon of each station; NR: not recommended due to insufficient data.

ng/L (Yender and Christine, 2002). Water samples from the Kuala Gula and Kuala Juru stations contain BkFl and BaPy at concentrations that exceed the criteria for water quality and for the consumption of organisms. The Kuala Perlis station contained BkFl in amounts that exceeded the water and organism consumption criteria. The BkFl of the Kuala Juru station exceeded the criteria for consumption of organisms. Some investigations in the USA has sought to determine the level of BaPy in fish or shellfish tissue that would not pose a significant human health risk after consumption (Schwarzenegger et al., 2007). The PAH criteria for a safe quantity for regular weekly consumption of fish and/or shellfish (edible tissue for human consumption) varied based on consumption rate. According to these criteria, the maximum allowable concentration of BaPy in edible tissue must be 4, 2, and 1 ng/g ww for low consumption at 50 g/week, moderate consumption at 100 g/week and heavy consumption at 200 g/week, respectively (O'Riordan, 1993). Based on the BaPy criteria, blood cockle in Kuala Juru are not in the safe range, but blood cockle in Sungai Haji Durani and Kuala Gula are safe for the low and moderate consumption rates, respectively. For the other three stations the concentration of BaPy in total soft tissues was lower than the detection limit. The concentrations of individual and total carcinogenic PAHs (t-PAHs) found in the edible tissue of blood cockle are displayed in Table 3.

2.2 Assessment of non-cancer hazard and cancer risk from exposure to c-PAHs

This work focused on the assessment of the non-cancer and cancer risk from exposure to c-PAHs through three routes: (1) gastrointestinal exposure from the intake of Malaysian blood cockle; (2) skin exposure while swimming or collecting blood cockle from the beach, tested for residents only; and (3) gastrointestinal exposure while swimming or collecting blood cockle, tested for residents only. The inhalation exposure of residents to c-PAHs while swimming and/or collecting blood cockle is excluded from the calculation of risk assessment in this study.

Risk assessment was calculated for two populations or cases: Case I – non-residents, i.e., the international and Malaysian consumers of Malaysian blood cockle who do not have contact with the polluted sediment and water in the study area (therefore, only cancer risk and the non-cancer hazard of ingestion of the contaminated blood cockle were considered in this group); and Case II – residents, who are most likely have frequent contact not only with blood cockles but also with the contaminated sites where they grow.

The assessment was done for the average concentration of the c-PAHs. The risk in general conditions was used to calculate the chronic daily intake (CDI), the absorbed dose (DAD, ADD), the hazard quotient of non-cancer (HQ), the hazard index (HI) and the cancer risk based on data on the Malaysian population (Ministry of Health Malaysia, 2003) and US EPA Guidance (US EPA, 1991; Syracuse Research Corporation, 2002; ENSR Corporation, 2005; Integrated Environmental Management Inc. and Riverfront Environmental, 2007) as shown in Table 1.

2.2.1 Case I

The hazard index and cancer risk of Malaysian and of international consumers of the Malaysian blood cockle, *A. granosa*, based on the ingestion rate for different populations is summarised in Table 4. The ingestion rate of cockle based on the mean value for Malay, Chinese and Indian ethnicities in Peninsular Malaysia are 5.36, 3.10 and 3.61 g/day, respectively (Ministry of Health Malaysia, 2003). For children and international consumers, the values of 3.55 and 8.01 g/day were used as defaults (US Department of Health and Human Services, 2009). As seen from the data, the non-cancer risks of eating cockle are lower than the level of concern (HQ < 1) for all of the monitoring stations (ENSR Corporation, 2005; Khairy et al., 2009). However, the cancer risk criterion indicates that eating *A. granosa* is not safe for either national or international consumers. The consumption of contaminated blood cockle increases the Malaysian cancer risk values significantly. For example, the probability of developing cancer associated with eating blood cockle from the Pantai Remis station in Chinese populations is 2.05 in 100,000, whereas the chance of developing cancer for consumers of Peninsular Malaysia by eating of blood cockle collected from Kuala Juru, is 8.87 in 1000. The risk management range of US EPA for excess cancer risk is 1×10^{-6}, which indicates the excess cancer risk equating to one excess cancer in a population of one million people. This criterion estimates that, for every 1000,000 people consuming fish or shellfish containing certain c-PAHs at a certain ingestion rate measured in g/day for 70 years, only

Table 4 Hazard quotient (HQ) and cancer risk (CR) calculated for Malaysian and international consumers of blood cockle, Case I

Station	Criteria	Sungai Buloh	Pantai Remis	Sungai Haji Durani	Kuala Gula	Kuala Juru	Kuala Perlis
EPA	HQ, adult	(1.31 ± 0.12)E-02	(1.02 ± 0.14) E-03	(8.69 ± 0.97)E-03	(2.09 ± 0.43)E-03	(1.43 ± 0.03)E-02	(1.69 ± 0.10)E-04
Peninsular Malaysia		(3.74 ± 0.25)E-02	(2.89 ± 0.15)E-03	(2.47 ± 0.18)E-02	(5.94 ± 1.23)E-03	(4.07 ± 0.83)E-02	(3.29 ± 0.20)E-03
Malay		(8.79 ± 0.52)E-03	(6.80 ± 1.61)E-04	(5.81 ± 0.35)E-03	(1.40 ± 0.29)E-03	(9.56 ± 1.94)E-03	(1.13 ± 0.07)E-03
Chinese		(5.08 ± 0.47)E-03	(3.94 ± 0.21)E-04	(3.36 ± 0.27)E-03	(8.08 ± 1.67)E-04	(5.53 ± 1.12)E-03	(6.55 ± 0.40)E-04
Indian		(5.92 ± 0.45)E-03	(4.58 ± 0.43)E-04	(3.91 ± 0.44)E-03	(9.41 ± 1.95)E-04	(6.44 ± 1.31)E-03	(7.63 ± 0.47)E-04
EPA	HQ, child	(2.42 ± 0.13)E-02	(1.88 ± 0.30)E-03	(1.60 ± 0.18)E-02	(3.86 ± 0.80)E-03	(2.64 ± 0.54)E-02	(3.12 ± 0.19)E-03
EPA	CR, adult	(1.22 ± 0.21)E-02	(5.30 ± 0.82)E-05	(3.95 ± 0.40)E-03	(2.09 ± 0.24)E-03	(1.44 ± 0.03)E-02	(8.82 ± 0.54)E-06
Peninsular Malaysia		(7.50 ± 0.50)E-03	(3.26 ± 1.73)E-05	(2.43 ± 0.35)E-03	(1.28 ± 0.15)E-03	(8.87 ± 0.20)E-03	(5.42 ± 0.33)E-05
Malay		(8.17 ± 0.76)E-03	(3.55 ± 0.88)E-05	(2.64 ± 0.69)E-03	(1.40 ± 0.16)E-03	(9.66 ± 0.22)E-03	(5.90 ± 0.36)E-05
Chinese		(4.72 ± 0.34)E-03	(2.05 ± 0.11)E-05	(1.53 ± 0.11)E-03	(8.08 ± 0.95)E-04	(5.59 ± 0.12)E-03	(3.41 ± 0.21)E-05
Indian		(5.50 ± 0.32)E-03	(2.39 ± 0.27)E-05	(1.78 ± 0.18)E-03	(9.41 ± 1.10)E-04	(6.51 ± 0.15)E-03	(3.98 ± 0.24)E-05
EPA	CR, child	(2.25 ± 0.11)E-02	(9.79 ± 1.20)E-05	(7.29 ± 0.54)E-03	(3.86 ± 0.45)E-03	(2.67 ± 0.06)E-02	(1.63 ± 0.01)E-04

Data are expressed as mean± STD of three replicates one additional case of cancer would be expected. This is a theoretical estimate that is based on very conservative mathematical calculations. The true risk could be much lower, even zero (Schwarzenegger et al., 2007; Verbrugge, 2008; US Department of Health and Human Services, 2009).

Children could be at greater risk than adults because children more often engage in hand-to-mouth behaviours. A lower body weight and a higher intake rate in children results in a greater dose of hazardous substances per unit of body mass (Verbrugge, 2008). The chance of developing cancer through the ingestion of blood cockle varies between $(9.79 \pm 1.20) \times 10^{-5}$ to $(2.06 \pm 0.06) \times 10^{-4}$ at the Pantai Remis and Kuala Juru stations, respectively.

The contributions to the cancer risk associated with the ingestion of blood cockle contaminated with PAHs varied across stations. For instance, the blood cockle samples from Pantai Remis and Kuala Perlis contained only one of the B2 group compounds, chrysene. In Kuala Gula, Kuala Juru and Sungai Haji Durani, BaPy found in the blood cockle soft tissue was the main cancer developer among the c-PAH compounds.

In blood cockle collected at Sungai Buloh, the cancer risk associated with DAnt increased more than any other c-PAH. Figure 2 summarises the contributions of cancer risk from c-PAHs associated with the ingestion of blood cockle tissue collected from mudflats on the west coast of Peninsular Malaysia.

2.2.2 Case II

The total HI of the non-cancer quotient is calculated for each exposure pathway by summing the HQs for each individual constituent. If the total HI becomes greater than the US EPA target HI for any receptor, potential non-carcinogenic effects based on specific health end points might begin to appear. Excessive cancer risks are summed across all of the c-PAHs and all of the exposure pathways that contribute to the exposure of an individual in a given population (ENSR Corporation, 2005). A summary of all of the HIs and cancer risks for each receptor group is presented in Table 5. Due to a lack of data, skin exposure to water was excluded from the calculation of the HQ and CR in Sungai Haji Durani, Sungai Buloh, and Pantai Remis. As explained before, the high level of c-PAHs in

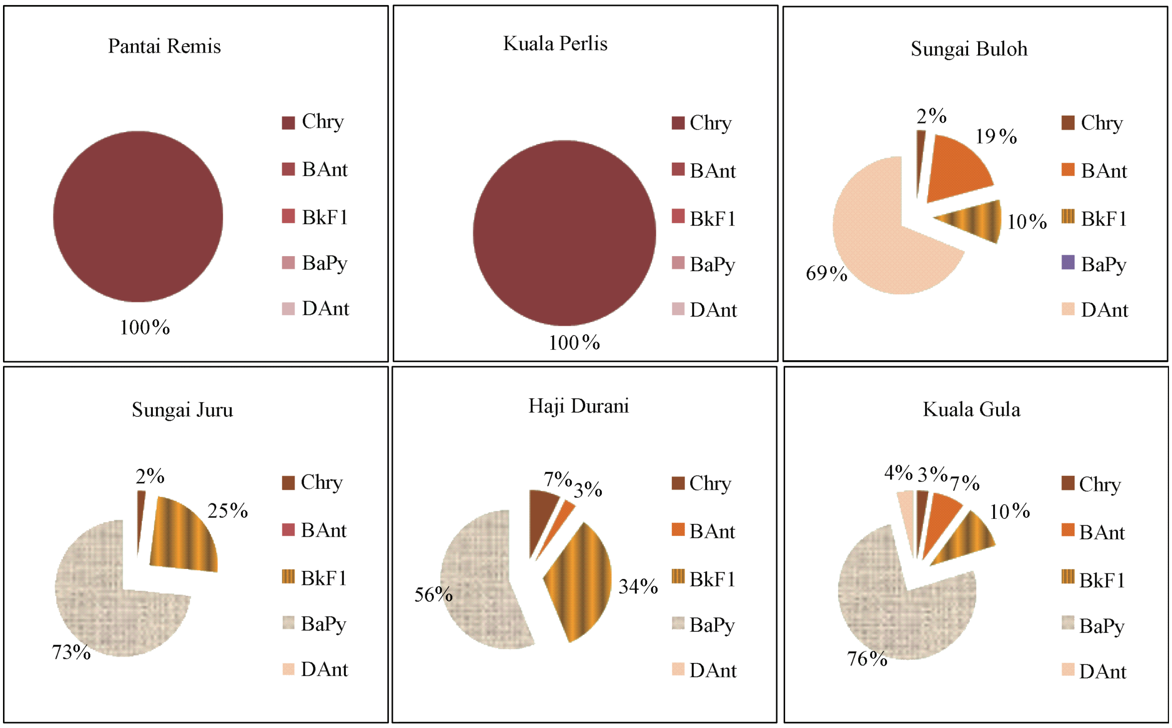

Fig. 2 Contributions to cancer risk of PAHs from ingestion of blood cockle, calculated from the average concentrations at different stations.

Table 5 Hazard index (HI) and total cancer risk calculated for residents from different sources, case II

Station	Criteria	Sungai Buloh	Pantai Remis	Sungai Haji Durani	Kuala Gula	Kuala Juru	Kuala Perlis
Peninsular Malaysia	HI, adult	(3.74 ± 0.69)E-02	(2.89 ± 0.37)E-03	(2.47 ± 0.55)E-02	3.04 ± 0.37	4.63 ± 1.09	1.11 ± 0.12
Malay		(8.79 ± 1.63)E-03	(6.80 ± 1.23)E-04	(5.81 ± 1.29)E-03	3.03 ± 0.37	4.60 ± 0.89	1.12 ± 0.70
Chinese		(5.09 ± 0.95)E-03	(3.94 ± 0.18)E-04	(3.36 ± 0.75)E-03	3.03 ± 0.21	4.59 ± 1.11	1.10 ± 0.21
Indian		(5.92 ± 1.10)E-03	(4.58 ± 1.87)E-04	(3.92 ± 0.87)E-03	3.03 ± 0.70	4.59 ± 1.41	1.10 ± 0.31
EPA	HI, child	(2.43 ± 0.45)E-02	(1.88 ± 0.19)E-03	(1.60 ± 0.26)E-02	3.05 ± 0.02	46.10 ± 8.81	110.00 ± 45.30
Peninsular Malaysia	∑CR, adult	(7.50 ± 1.40)E-03	(3.26 ± 0.35)E-05	(2.43 ± 0.49)E-03	(4.10 ± 1.00)E-02	(5.48 ± 1.81)E-02	(4.68 ± 0.31)E-03
Malay		(8.17 ± 1.53)E-03	(3.55 ± 0.37)E-05	(2.64 ± 0.54)E-03	(4.11 ± 1.27)E-02	(5.56 ± 1.01)E-02	(4.69 ± 0.33)E-03
Chinese		(4.72 ± 0.88)E-03	(2.05 ± 0.22)E-05	(1.53 ± 0.31)E-03	(4.05 ± 1.11)E-02	(5.15 ± 1.11)E-02	(4.66 ± 0.36)E-03
Indian		(5.50 ± 1.03)E-03	(2.39 ± 0.15)E-05	(1.78 ± 0.26)E-03	(4.06 ± 0.97)E-02	(5.24 ± 1.01)E-02	(4.67 ± 0.38)E-03
EPA	∑CR, child	(2.25 ± 0.42)E-02	(9.79 ± 1.04)E-05	(7.29 ± 1.48)E-03	(2.12 ± 0.15)E-02	(4.67 ± 1.39)E-02	(2.17 ± 0.27)E-03

Data are expressed as mean ± STD of three replicates.

Malaysian blood cockle on west coast of Malaysia has been increasing the cancer risk of blood cockle consumers. The food pathway is of the greatest concern for c-PAH intake across a wide range of consumers, but other pathways are also important, particularly for local residents, based on the accumulative nature of c-PAHs. Additional intake of c-PAHs for residents and workers in these polluted areas is another possible and serious concern. Cumulative amounts of dermal intake and the ingestion of sediments by persons who come into direct contact with the sediment at a site (e.g., by ingesting it or through small amounts of sediment that adhere to hands during outdoor activity) potentially raise cancer risks for workers and other residents in polluted areas.

In this study, the HI measures for Kuala Juru, Kuala Perlis and Kuala Gula were found to be higher than the US EPA criteria. The HI at these stations were calculated for the pathways that include transmission through the skin and gastrointestinal water, the sediment alone and the gastrointestinal transmission associated with blood cockle. For the other three stations, c-PAHs of water were not analysed. Water samples taken from Malaysia's extended mudflats are heavily muddy, which causes the remobilisation of contaminants to increase the level and bioavailability of contaminants. The high levels of c-PAHs in study areas along the west coast of Peninsular Malaysia increased the population-level cancer risk significantly. According to the US EPA, an excess cancer risk above 1×10^{-4} is not safe (ENSR Corporation, 2005). Based on the findings from case II, the cancer risk is not in the US EPA's safe range at any of the studied sites, except for the Pantai Remis station, where the cancer risk was calculated without the risk from skin absorption of water because of the lack of data and therefore might well be as unsafe as the other sites studied. Data demonstrated that all of the populations studied in case II showed a risk in excess of US EPA risk management criteria 1×10^{-6}.

2.3 Risk management

In general, the US EPA considers excess cancer risks that are below 1×10^{-6} to be so small that can be negligible; risks above 1×10^{-4} are sufficiently large and require some sort of intervention or remediation. Excess cancer risks that range between 1×10^{-4} and 1×10^{-6} are generally not considered large enough to warrant action under superfund guidelines (ENSR Corporation, 2005). Based on reported data standards, all of the study areas in the west coast of Peninsular Malaysia need intervention, remediation or further serious action to reduce the amount of toxic compounds.

It is advised that workers and residents of the Malaysian extended mudflats avoid contact with the water and the sediment. Residents should clean their body properly after contact with the water and sediment and should avoid hand-to-mouth actions to avoid ingestion of contaminated sediment. Consumers of any age should not eat Malaysian blood cockle collected from Kuala Gula, Kuala Juru, Sungai Haji Durani, and Sungai Buloh.

2.4 Uncertainty of risk assessment results

The uncertainty of risk assessment was summarised according to reported data (US EPA, 2000, 2009; ENSR Corporation, 2005) and this case study. The cancer risk assessment is calculated over a lifetime, meaning that exposure characteristics to PAHs may change for an individual. Risk assessment based on the US EPA criteria presents a worst-case scenario; this research assessed the risk for the highest exposure time from the average concentration and average ingestion, intake and uptake rates. The methods used also assessed the risk from the toxicity of each compound and then combined all of the risks. In reality, the sediment and blood cockle consist of many carcinogens, such as organochlorines and dioxins, but the risk assessment in this research focussed on c-PAHs only. Therefore, the total cancer risk could well be higher than the values estimated in this article due to exposure to other carcinogens. There is currently no investigated surrogate or real RfD for c-PAHs, therefore, the RfD of naphthalene was used as it is the lowest oral RfD for a PAH-class compound (Integrated Environmental Management Inc. and Riverfront Environmental, 2007; IRIS, 2009). In this case, the estimation of the non-cancer risk might be overestimated. The Surrogate Oral Slop Factor (SF) of BaPy was used for humans, assuming that humans and rodents have the same quantitative probability to BaPy's cancer action (Department for Environment, 2002), an assumption which again causes uncertainty in the calculation.

3 Conclusions

The c-PAH data from the present study conducted in Peninsular Malaysia highlighted major problems related to the effects of c-PAH contamination and at the same time to the lack of registered guidelines in Malaysia and in the Straits of Malacca.

This study has demonstrated that hazard indexes from multiple sources based on population-specific ingestion rates and contact rates with polluted water and sediment are higher than the US EPA's target for HI consumption. However, the HIs for consumption of total soft tissues of blood cockle are under the target value of US EPA.

According to ingestion rate of population, cancer risk of consumption of cockle ranged from $(8.82 \pm 0.54) \times 10^{-6}$ to $(2.67 \pm 0.06) \times 10^{-2}$. The calculations of cancer risk at all of the stations for all consumers exceed the risk management range by US EPA.

The present findings suggest that strict regulations and improvements in the existing environmental legislation should be made to avoid adverse effects to public health both within and outside of the country. Based on these findings, consumers should be advised not eat Malaysian blood cockle.

Acknowledgments

This work was supported by the MOSTI Science Funding Project (No. 5450100). Special thanks to Associate Professor Dr. Aziz Arshad and Dr. Mahyar Sakari for their assistance in this study.

References

Abdullah A R, Woon W C, Bakar R A, 1996. Distribution of oil and grease and petroleum hydrocarbons in the straits of Johor, Peninsular Malaysia. *Bulletin of Environmental Contamination and Toxicology*, 57(1): 155–162.

Abdullah M P, 1997. Hydrocarbon pollution in the sediment of some Malaysian coastal areas. *Environmental Monitoring and Assessment*, 44: 443–454.

Anyakora C, Ogbeche A, Palmer P, Coker H, Ukpo G, Ogah C, 2005. GC/MS analysis of polynuclear aromatic hydrocarbons in sediment samples from the Niger Delta Region. *Chemosphere*, 60: 990–997.

Arias A H, Spetter C V, Freije R N H, Marcovecchio J E, 2009. Polycyclic aromatic hydrocarbons in water, mussels (*Brachidontes* sp., *Tagelus* sp.) and fish (*Odontesthes* sp.) from Bahía Blanca Estuary, Argentina. *Estuarine, Coastal and Shelf Science*, 85: 67–81.

Bakhtiari A R, Zakaria M P, Yaziz M I, Lajis M N H, Bi X, 2009. Polycyclic aromatic hydrocarbons and *n*-alkanes in suspended particulate matter and sediments from the Langat River, Peninsular Malaysia. *Environment Asia*, 2: 1–10.

Baumard P, Budzinski H, Garrigues P, Narbonne J F, Burgeot T, Michel X et al., 1999. Polycyclic aromatic hydrocarbon (PAH) burden of mussels (*Mytilus* sp.) in different marine environments in relation with sediment PAH contamination, and bioavailability. *Marine Environmental Research*, 47: 415–439.

Boonyatumanon R, Wattayakorn G, Togo A, Takada H, 2006. Distribution and origins of polycyclic aromatic hydrocarbons (PAHs) in riverine, estuarine, and marine sediments in Thailand. *Marin Pollution Bulletin*, 52: 942–956.

Chan M K, Othman R, Zubir D, Salmijah S, 2002. Induction of a putative metallothionein gene in the blood cockle, *Anadara granosa*, exposed to cadmium. *Comparative Biochemistry and Physiology Part C*, 131: 123–132.

Cheevaporn V, Menasveta P, 2003. Water pollution and habitat degradation in the Gulf of Thailand. *Marine Pollution Bulletin*, 47: 43–51.

Chouksey M K, Kadamb A N, Zingdeb M D, 2004. Petroleum hydrocarbon residues in the marine environment of Bassein-Mumbai. *Marine Pollution Bulletin*, 49(7-8): 637–647.

Collins J F, Brown J P, Alexeeff G V, Salmon A G, 1998. Potency equivalency factors for some polycyclic aromatic hydrocarbons and polycyclic aromatic hydrocarbon derivatives. *Regulatory Toxicology and Pharmacology*, 28: 45–54.

Cortazar E, Bartolom L, Arrasate S, Usobiaga A, Raposo J C, Zuloaga O et al., 2008. Distribution and bioaccumulation of PAHs in the UNESCO protected natural reserve of Urdaibai, Bay of Biscay. *Chemosphere*, 72: 1467–1474.

Culotta L, Stefano C D, Gianguzza A, Mannino M R, Orecchio S, 2006. The PAH composition of surface sediments from stagnone coastal lagoon, Marsala (Italy). *Marine Chemistry*, 99: 117–127.

Department for Environment, Food and Rural Affairs and the Environment Agency, 2002. Contaminants in soil collation of toxicological data and intake valaues for humans, Benzo[a]Pyrene. Research Report of Environment Agency.

ENSR Corporation, 2005. Remedial investigation/feasibility study work plan: human health risk assessment work plan pines area of investigation AOC II Docket. In: Research Report No. V-W-'04-C-784 of Human Health Risk Assessment Work Plan.

Froun H, Polletin J, Fournier M, Pelletier E, Richard P, Pichaud N et al., 2007. Physiological effects of polycyclic aromatic hydrocarbons on soft-shell clam Mya Arenaria. *Aquatic Toxicology*, 82: 120– 134

Ibrahim N, 1995. Trace element content of Malaysian cockles (*Anadara granosa*). *Food Chemistry*, 54: 133–135.

Integrated Environmental Management Inc., Riverfront environmental, 2007. Baseline human health risk assessment for the Westinghouse Hematite site. In: Research Report No. EO-05-003.

IRIS (Integrated Risk Information System). 2009. A-Z list of substances. Retrieved December. http://www.epa.gov/NCEA/iris/subst/.

Isobe T, Takada H, Kanai M, Tsumi S, Isobe K O, Boonyatumanond R et al., 2007. Distribution of polycyclic aromatic hydrocarbons (PAHs) and phenolic endocrine disruption chemicals in South and Southeast Asian mussels. *Environmental Monitoring and Assessment*, 135(1-3): 423–440.

Khairy M A, Kol M, Mostafa A R, EL-Fiky A, Bahadir M, 2009. Risk assessment of polycyclic aromatic hydrocarbons in a Mediterranean semi-enclosed basin affected by human activities (Abu Qir Bay, Egypt). *Hazardous Materials*, 170: 389–397.

Mallika P, Sarisak S, Pongsri P, 2008. Cancer risk assessment from exposure to trihalomethanes in tap water and swimming pool water. *Journal of Environmental Sciences*, 20(3): 372–378.

McGroddy S E, Farrington J W, 1995. Sediment porewater partitioning of polycyclic aromatic hydrocarbons in three cores from Boston Harbor, Massachusetts. *Environmental Science and Technology*, 29: 1542–1550.

Ministry of Health Malaysia, 2003. Food consumption statistics of Malaysia 2003 for adult population aged 18 to 59 years. In: Research Report No. KKM/2000/2006 of Family Health Development Division and Food Safety and Quality Division, Putrajaya, Malaysia.

Minnesota Department of Health, 2004. Polycyclic aromatic hydrocarbons: Methods for estimating health risks from carcinogenic PAHs. In: Research Report of MPCA (Minnesota Pollution Control Agency): Health Risk Assessment Unit, Minnesota, USA.

Moradi A M, 2001. The kinetics of uptake and release of polycyclyc aromatic hydrocrbons in the green mussels *Perna viridis* for biomonitoring of marine pollution. Ph.D Thesis. University Putra Malaysia, Serdang, Malaysia.

Neff J M, 1979. Polycyclic Aromatic Hydrocarbons in the Aquatic Environment: Sources, Fate and, Biological Effect. London, England.

Nikolaou A, Kostopoulou M, Lofrano G, Meric S, Petsas A, Vagi M, 2009. Levels and toxicity of polycyclic aromatic hydrocarbons in marine sediments. *Trends in Analytical Chemistry*, 28(6): 676–694.

O'Riordan J, 1993. Water quality: Ambient water quality criteria for polycyclic aromatic hydrocarbons (PAHs), overview Report. In: Research Report of Ministry of Environment, Environmental Protection Division, Canada.

Roberts S, Goff K, 2008. Human health based surface water criteria, 12. Retrieved 7.12.2009, from http://www. floridadep.com/water/wqssp/docs/tr_review/roberts_ver_7.pdf.

Sakari M, Zakaria M P, Junos M B M, Annuar N A, Yun H Y, Heng Y S et al., 2008a. Spatial distribution of petroleum hydrocarbon in sediments of major rivers from East Coast of Peninsular Malaysia. *Coastal Marine Science*, 32(1): 9–

18.

Sakari M, Zakaria M P, Lajis N H, Mohamed C A R, Bahry P S, Anita S et al., 2008b. Characterization, distribution, sources and origins of aliphatic hydrocarbons from surface sediment of Prai Strait, Penang, Malaysia: A widespread anthropogenic input. *Environment Asia*, 2: 1–14.

Schwarzenegger A, Adams L S, Denton J E, 2007. Report on the safety of consuming fish and shellfish from areas impacted by the M/V Cosco Busan Oil Spill in San Francisco, California. In: Research Report of California Environmental Protection Agency State of California, USA.

Scientific Committee on Food, 2002. Polycyclic aromatic hydrocarbons – Occurrence in foods, dietary exposure and health effects. In: Research Report of European Commission Health and Consumer Protection Directorate-General Directorate C – Scientific Opinions C2 – Management of Scientific Committees; Scientific Co-operation and Networks Brussels, Belgium.

SRP (Scientific Review Panel), 2009. TSD for cancer potency factors: Technical support document for cancer potency factors, Appendix C: Use of the toxicity equivalency factor Scheme for Estimating Toxicity of Mixtures of Dioxin-Like Chemicals. In: Research Report No. TEFWHO-97 and TEFWHO-05 of SRP draft.

Shi Z, Tao S, Pan B, Liu W X, Shen W R, 2007. Partitioning and source diagnostics of polycyclic aromatic hydrocarbons in rivers in Tianjin, China. *Environmental Pollution*, 146: 492–500.

Shor L M, Kosson D S, Rockne K J, Rockne L J, Young L Y, Taghon G L, 2004. Combined effect of contaminant desorption and toxicity on risk from PAH contaminated sediments. *Risk Analysis*, 24(5): 1109–1120.

Siddiqi H A, Ansari F A, Munshi A B, 2009. Assessment of hydrocarbons concentration in marine fauna due to Tasman Spirit oil spill along the Clifton beach at Karachi coast. *Environmental Monitoring and Assessment*, 148: 139–148.

Syracuse Research Corporation, 2002. Baseline human health risk assessment for the Intermountain West Oil Refinery Site Bountiful, Utah. In: Research Report of U.S. Environmental Protection Agency Region 8 and Syracuse Research Corporation Denver.

Tong S L, Goh S H, Abdulah A R, Tahir N M, Wang C W, 1999. Asian marine water quality criteria for oil and grease. In: Research Report No. ASEAN-Canada CPMS-II of Pollution Control Department, Kuala Lumpur, Malaysia.

US Department of Health and Human Services, 2009. Health consultation: WYCKOFF/Eagle Harbor Superfund Site Bain Bridge Island, KITSAP County, Washington. In: Research Report No. EPA FACILITY ID: WAD009248295 of Public Health Service: Agency for Toxic Substances and Disease Registry, Division of Regional Operations, Atlanta, USA.

US EPA, 1991. Standard Default Exposure Factors. Retrieved December 2009. http://www.epa.gov/oswer/riskassessment/sghandbook/pdfs/riskcalc-bap.pdf.

US EPA, 2000. Methodology for deriving ambient water quality criteria for the protection of human health. Final Office In: Research Report No. 822-B-00-004 of Office of Science and Technology: Office of Water EPA),Washington DC, USA.

US EPA. 2009. Updated Dermal Exposure Assessment Guidance. http://www.epa.gov/reg3hscd/risk/human/info/dermalag.htm.

Verbrugge L A, 2008. Health consultation: Evaluation of blue mussel samples collected in May 2006 M/V Seledang Ayu Oil Spill, Unalaska, Alaska. In: Research Report of U.S. Department of Health and Human Services, Public Health Service, Agency for Toxic Substances and Disease Registry, USA.

Vinas L, Franco M A, Soriano J A, Gonzalez J J, Ortiz L, Bayona J M et al., 2009. Accumulation trends of petroleum hydrocarbons in commercial shellfish from the Galician coast (NW Spain) affected by the Prestige Oil Spill. *Chemosphere*, 75: 534–541.

Washington State Department of Ecology, 2007. Evaluating the toxicity and assessing the carcinogenic risk of environmental mixtures using toxicity equivalency factors, 8. https://fortress.wa.gov/ecy/clarc/FocusSheets/tef.pdf.

Yender R, Christine M J, 2002. Managing seafood safety after an oil spill. In: Research Report of Hazardous Materials Response Division, Office of Response and Restoration, National Oceanic and Atmospheric Administration, Seattle, USA.

Yim U H, Hong S H, Shim W J Oh J R, Chang M, 2005. Spatio-temporal distribution and characteristics of PAHs in sediments from Masan Bay, Korea. *Marine Pollution Bulletin*, 50: 319–326.

Zakaria M P, Mahat A A, 2006. Distribution of polycyclic aromatic hydrocarbon (PAHs) in sediment in the Langat Estuary. *Coastal Marine Science*, 30(1): 387–395.

Zakaria M P, Okuda T, Takada H, 2001. Polycyclic aromatic hydrocarbons (PAHs) and hopane in stranded tar-balls on the coast of Peninsular Malaysia: Applications of biomarkers for identifying source of oil pollution. *Marin Pollution Bulletin*, 42(12): 1357–1366.

Zakaria M P, Takada H, Tsutsumi S, Ohno K, Yamada J, Kouno E et al., 2002. Distribution of polycyclic aromatic hydrocarbons (PAHs) in rivers and estuaries in Malaysia: A Widespread input of petrogenic PAHs. *Environmental Science and Technology*, 36: 1907–1918.