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Dietary intake of polycyclic aromatic hydrocarbons (PAHs) from coral reef fish in the Persian Gulf — Human health risk assessment

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ABSTRACT

The present study investigated accumulation of petrogenic polycyclic aromatic hydrocarbons (Σ_{39}PAHs) in the livers and muscles of three coral-reef fish (50 specimens) from the Persian Gulf, Kharg Island (Iran), specifically *Lethrinus microdon* (n = 18), *Lutjanus argentimaculatus* (n = 17), and *Scomberomorus guttatus* (n = 15). For all fish, PAHs originated mostly from petroleum and combustion sources. Concentrations of Σ_{39}PAHs were 1004 ngg^{-1} freeze-dried weight (fdw) and 1390 ngg^{-1} fdw for liver and muscle, respectively. The biota-sediment accumulation factor of 20,181 and equivalent concentrations of ΣPAHs (liver) were highest for *L. argentimaculatus*. Most of the abundant PAHs identified were low molecular weight (LMW-PAH) (liver > muscle) with 2–3 aromatic ring. Results for the human health risk assessment concluded the probability of PAHs intake via fish consumption was considerable in this area (lifetime cancer risk (ILCR) > 1×10^{-5}; hazard quotients (HQ_s) > 1; hazard index (HI) ≈6; the excess cancer risk (ECR) > 1×10^{-6}) and, therefore, comprehensive management and long-term monitoring is needed.

1. Introduction

Aquatic environments worldwide are increasingly contaminated by different pollutants (Ranjbar Jafarabadi et al., 2019; Ranjbar Jafarabadi, Mashjoor, Mohamadjafari Dehkordi, Riyahi Bakhtiari, & Cappello, 2020). Polycyclic aromatic hydrocarbons (PAHs) have been attracting attention for decades, since these compounds are associated with risks for environmental and public health. These hazardous hydrophobic bio-compounds have two or more aromatic rings and are found ubiquitously in aquatic environments (Miao, 2015). PAHs can enter into the environment via diverse anthropogenic sources, including defective combustion of wood, coal, or petroleum benzine, ship loading/unloading, sewage sludge, municipal waste water discharge, runoff, and atmospheric precipitation (Santana et al., 2018; Mahugija & Njale, 2018; Cerrillo, Vieira, Ochoa-Gaona, de Jong, & Serrano, 2019). The adverse human health effects, cytotoxicity, genotoxicity/mutagenicity and carcinogenicity, are related to source and tissue concentrations (WHO, 1998). However, the International Programme on Chemical Safety (IPCS), FAO/WHO Expert Committee on Food Additives (JECFA), and the Scientific Committee on Food (SCF) might have been under evaluating PAHs by using site-specific "eco-monitoring" approaches. Based on the US Agency for Toxic Substances and Disease Registry (ATSDR) hazard priority list, toxic properties of PAHs depend on the numbers of rings.

PAHs are characteristically hydrophobic with high octanol/water partition coefficients (K_{ow}/log K_{ow}), low water solubility, and are a significant environmental concern due to their persistence, toxicity, teratogenicity, and bio-accumulation (Abdel-Shafy & Mansour, 2016). Overall, they can affect the growth, metabolism, and survival rate of organisms (Honda & Suzuki, 2020). Humans may be exposed to PAHs via several pathways. Dietary exposure to chemical contaminants, particularly seafood, is a major source of PAH that impact human health (Mahugija & Njale, 2018). However, in many parts of the world, seafood is an indispensable food source, because it contains high-quality protein, ω-3 fatty acids, vitamins, and minerals (Ferrante et al., 2018). Iran's Fisheries Organization (IFO) reported that consumption of fish per annum per capita is 11 kg (IFO, 2019). But, in the Northern regions of the Persian Gulf (Iranian Kharg coral Island/Bushehr province), due to limitations in local transportation, consumption of fish and shellfish is a part of the daily diet (FAO, 2015). The quality of seafood is, however, dependent on the marine environment, which can also affect consumer health indirectly (Ferrante et al., 2018).

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Biomagnification of PAHs through the food chain (PAH troph) can affect negatively the health and productivity of the marine environment and, eventually, the health of who consume these marine organisms as food. Consumption of PAHs is associated with increased rates of cancer, birth defects, and mutations (Moorthy, Chu, & Carlin, 2015). Rapid increase in human population, industrialization, and coastal development, and increased pollution, particularly with oil-based pollutants, have considerable implications for the sustainability and utilization of marine resources including those around Kharg Island (Iran). However, investigation of PAHs in the environment and seafoods from Kharg Island are scarce (Mirvakili & Hadjizadeh Zaker, 2014; Mirza et al., 2012). Considering increasing environmental pressures on the unique ecosystems around this Island, seafood contamination by PAHs, particularly fish, could be expected to affect adversely the health of communities who consume these resources locally. Therefore, the objectives of the present study were to 1) estimate rates of contamination in three fish species, Smalltooth emperor (*Lethrinus microdon*), mangrove red snapper (*Lutjanus argentimaculatus*), and Indo-Pacific king mackerel (*Scomberomorus guttatus*), 2) identify plausible sources of PAHs around the Island, and 3) assess the implications for human health risks.

2. Materials and methods

2.1. Sampling

Fish were collected from local fishermen in July 2015 at the main fishing dock and fish market of Kharg Island (Fig. 1). Kharg Island (29° 15′ N, 50° 20′ E) is located in the Northwest of the Persian Gulf, 35 km south of Ganaveh Harbor in Bushehr Province (Iran). The Island is the most important Iran's primary oil export terminal in the Persian Gulf (http://www.iotco.ir/en/oilterminals/kharg).

2.2. Fish, sediments and seawater sampling

Three species of fish were collected from local fishermen between July 2015, including *L. argentimaculatus* (n = 17, weight: 7.81–10.82 Kg, length: 71.34–184.21 cm), *L. microdon* (n = 18, weight: 4.57–6.70 Kg, length: 36.84–83.47 cm), *S. guttatus* (n = 15, weight: 4.19–5.47 Kg, length: 55.63–72.81 cm). The fish were washed with fresh water and packed in bags (on ice) before transfer to the laboratory of Tarbiyat Modares University (Tehran, Iran). Once in the lab, muscle and liver tissues were collected, freeze-dried, ground, sieved, and stored at −20 °C until further analysis. Surface sediment (0–5 cm) and top layer seawater (0–40 cm) samples (n = 5) were collected from each sampling site. Surface sediment sampling was performed using a Van Veen grab sampler (Riddle, 1989).

2.3. Reagents

PAH standards were purchased from Sigma-Aldrich Chemie GmbH (Schnelldorf-Germany). *n*-hexane, methanol, dichloromethane, acetone, chloroform, potassium hydroxide pellets, and silica gels (63–230 mesh) were purchased from Merck Inc., Darmstadt and Augsburg, Germany. All reagents were of analytical grade and Millipore-Q water (purified by Millipore Milli-Q® Water System, Bedford, MA, USA) were used throughout.

Lipid content from PAHs-contaminated fish was determined according to the procedure described in Bandowe et al. (2014) using dichloromethane. Quality control (QC) and quality assurance (QA) were performed as described in previous works (Ranjbar Jafarabadi et al., 2017; Ranjbar Jafarabadi et al., 2019). For each individual PAH congener, the limit of quantification (LOQ) of analytical methods was determined by sample amounts and procedural blanks. In addition, the set of samples was subjected to forth GC–MS runs to specify PAHs. Then, the relative percent difference (RPD) of forth samples was measured to produce a specified signal-to-noise ratio (S/N) or/and quantification limits. For all PAH congeners (Table S1), LOQs ranged from 0.02 to 0.1 ng g^{-1} fdw. The relative percent difference (RPD) of forth samples were < 7%. For each PAH, detection limits were > 3 and quantification limits (factor of 10 for LOQ) ranged from 0.02 to 0.33 ng g^{-1} fdw, and 0.03–3.10 ng g^{-1} fdw, respectively.

2.4. Bioaccumulation factor

For PAHs and other hydrophobic organic compounds, bio-accumulation factors (BAFs) and bio-concentration factors (BCFs) are commonly defined as the ratio of tissue (Table S1) to water (Table S2) (BCF) (Eq. (1)) or the ratio of tissue to sediment (Table S2) (BAF) (Eq. (2)) (Ranjbar Jafarabadi et al., 2018; Yakan, Focks, Klasmeier, & Okay, 2017; Loh, Yim, Ha, & Kim, 2017). In addition, other researchers have expressed the biota–sediment accumulation factor (BSAF) (Eq. (3)), which is useful for normalizing the lipid and organic carbon in bioaccumulation. The formulae can be written as follows:

$$BCF = \frac{[tissue]}{[water]} \quad (1)$$

$$BAF = \frac{[tissue]}{[sediment]} \quad (2)$$

$$BSAF = \left(\frac{[tissue]}{f(lip)}\right) / \left(\frac{[sediment]}{f(oc)}\right) \quad (3)$$

where the mass of tissue and sediment are based on the freeze-dried weight (fdw) and dry sample weight (dw) (parts per billion/million [ppb/ppm]), respectively, f_{oc} is the fraction of organic carbon in sediment ($dwg.g^{-1}$), and $f_{(lip)}$ is the fraction of lipid in tissue ($fdwg.g^{-1}$). However, for sediment–water partitioning or tissue steady-state, several other factors can affect each of these bioaccumulation factors such as elimination rates, variable uptake, reduced bioavailability, and insufficient time.

2.5. Human health risks assessment

In order to have a comprehensive human health risks assessment, the potency equivalent concentration (PEC) (Nisbet & LaGoy, 1992), daily dietary intake (DDI) (Shi et al., 2016), lifetime cancer risk (ILCR) (Xia et al., 2010), chronic daily intake (CDI) (Yang, Lang, & Li, 2014), target hazard quotient (THQ) (USEPA., 2000), hazard index (HI) (USEPA., 2000), screen value (SV) (Fairey et al., 1997), and excess cancer risk (ECR) (Bandowe et al., 2014) were calculated. In order to understand the impacts of dietary intake of PAHs through seafood, to quantify the carcinogenicity of other PAHs relative to BaP, and to estimate BaP-equivalent concentration (BaPeq), the toxic equivalency factors (TEFs) were applied. This estimation expresses the environmental levels of other PAHs compared to BaP. The potency equivalent concentrations (PEC) of total PAHs were described as BaPeq (Vuorinen et al., 2006).

To the estimation of non-carcinogenic risk through ingestion and dermal adsorption, HQ was applied. Value of HQ < 1 indicates low or no significant adverse effects (human safety-health). HQ > 1 indicates the potential negative impacts (human health risk) (Frédéric & Yves, 2014).

2.5.1. PEc

The general equation for estimating PEC ($ng\text{-}TEQ.g^{-1}$) of 30 total PAHs in each fish sample is as follows:

$$PEC = \Sigma C_i \times TEF_i \quad (4)$$

where C_i is estimated concentration ($ng\ g^{-1}$) of PAH congeners (i) in fish tissue and TEF_i is the toxicity equivalence factor of i congeners relative to that of Banzo (a) pyrene (BaP). The TEF data for PAH congeners, obtained from the Nisbet and LaGoy (1992).

Fig. 1. Map of study area, Kharg Island, Persian Gulf, Iran.

2.5.2. Ddi

The estimated absolute daily intake (ADI, μg.day^{-1}) and relative daily intake (RDI, μg/(kg BW day)) for local adults who consume fish were calculated applying equations (5) and (6):

$$ADI = C \times IR \tag{5}$$

$$RDI = C \times IR/BW \tag{6}$$

where C (μg.g^{-1}) is measured for the concentration of PAHs in the muscle of fish, IR (g.day^{-1}) is the intake rate of fish, and BW (kg) is local adults body weight (Shi et al., 2016).

2.5.3. Life time cancer risk (ILCR)

The ILCR for dietary exposure to PAHs was estimated applying equation (7) and (8) (Xia et al., 2010). For ILCR assessment, a public screening criterion of 1.0E-06 was applied as a carcinogenic risk level to humans.

$$ILCR = ED \times EF \times EDI \times SF \times CF/AT \tag{7}$$

$$EDI = CR \times C/BW \tag{8}$$

where CF is the conversion factor (1.0E-06 mg.ng^{-1}); SF is the oral cancer slope factor of BaP (geometric mean of 7.3 mg.kg^{-1}.day^{-1}); EDI is the estimated daily intake of PAH exposure for human (ng.kg^{-1} body weight (BW) day^{-1}); EF is the exposure frequency (365 days.yr^{-1}); ED is estimated the exposure duration for 43 adults per year; AT is the average lifespan for carcinogens (25500 days); CR is the consumption rate of fish (38.9 g.day^{-1}) (NBSC, 2008), and BW is the average body weight of consumers (70 kg for adults).

2.5.4. CDi

To estimate the rate of contaminant intake, the mean concentration of environmental contaminants exposure, and variables of the exposed population were applied. The general equation for chemical intake is:

$$CDI = \frac{CW \times IR \times EF \times ED}{BW \times AT} \tag{9}$$

where the CDI is chronic daily intake by ingestion (mg.(kg.day)$^{-1}$), ED is exposure duration (years), EF is exposure frequency (days. Year^{-1}), IR is ingestion rate (L.day^{-1}), CW is a chemical dose in water (mg. L^{-1}), AT is averaging time (days), and BW is body weight (kg) (Yang et al., 2014).

2.5.5. THq

THQ is defined as the ratio of exposure to the toxic element and the reference dose (RfD) which is the highest level at which no adverse health effects are expected. THQ describes the non-carcinogenic health risk posed by exposure to a contaminant. If the ratio is < 1, then non-carcinogenic effects are not expected. If the ratio is equal or > 1, then adverse health risks could be experienced (Chien et al., 2002). The THQ

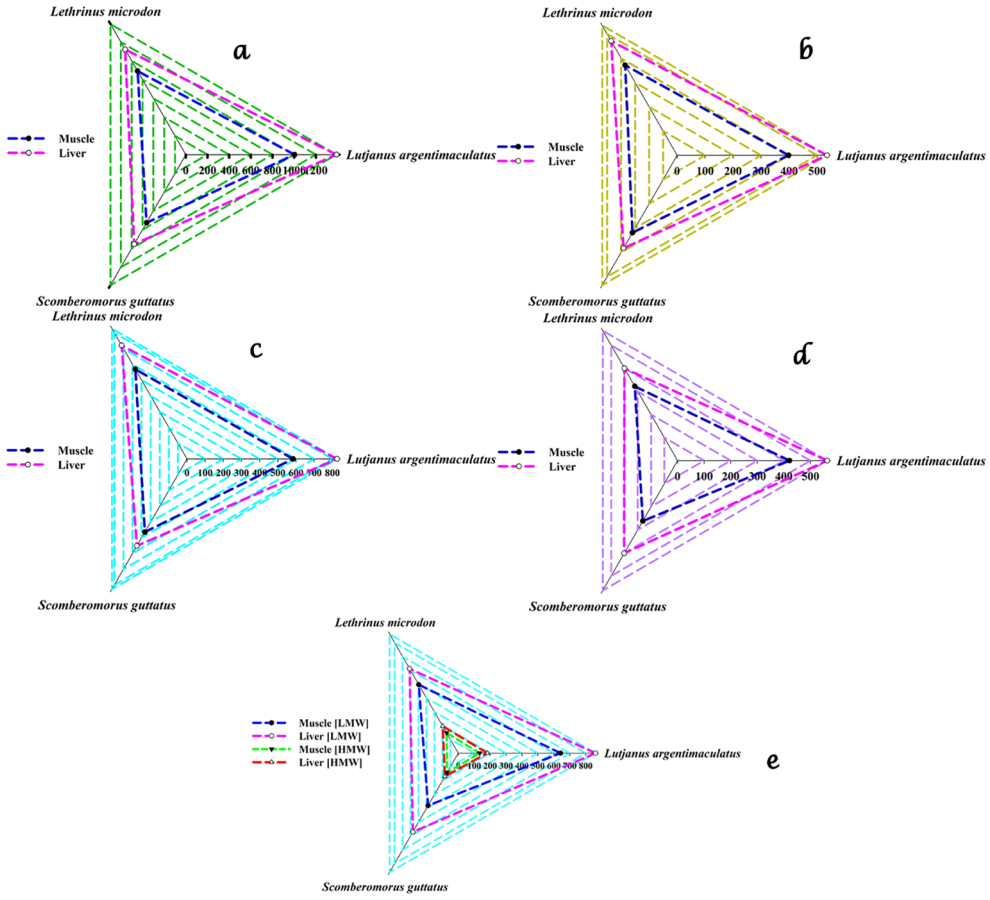

Fig. 2. Mean concentration (ng g^{-1}fdw) of (a) the sum of 39 PAHs (Σ_{39}PAHs), (b) Σ16PAHs, (c) parental PAHs (ΣPPAHs), (d) methylated PAHs ((ΣMPAHs), and (e) lower molecular and higher molecular PAHs (LMW, HMW-PAHs) in muscle and liver tissues of three fish (*L. argentimaculatus*, *L. microdon*, and *S. guttatus*) in Kharg Island.

was determined according to the method provided in the United States Environmental Protection Agency (US EPA) methodology based on the Region III risk-based concentration table (US EPA, 2000). To calculate the dose, standard exposure assumptions were acquired from US EPA integrated risk analysis (US EPA, 2000).

THQ is calculated by the following Eq. (10):

$$THQ = \frac{EFr \times EDtot \times IFR \times C}{RFDo \times BWa \times ATn} \times 10 - 3 \quad (10)$$

where EFr is exposure frequency (365 days. $Year^{-1}$), EDtot is the exposure duration (70 years, average lifetime), IFR is the food ingestion rate (kg/day), C (μg.g^{-1}) is PAH dose, RFDo (mg.kg^{-1}.day^{-1}) is the oral reference dose, BWa is the adult body weight (kg), and ATn is the average time for non-carcinogens (it is equal to EFr × EDtot) (days).

2.5.6. Hi

To estimate the total potential effects of more than one PAH, the sum of THQs for each individual PAH was computed and indicated as a hazard index (HI) (USEPA, 2000). The HI assumes that the consumption of a particular food type would result in simultaneous exposure to several potentially toxic elements. HI is calculated by the following Eq. (11):

$$HI = THQ1 + THQ2 + THQ3 + \cdots + THQn \quad (11)$$

Value of HQ < 0.2 for any given pathway is generally regarded that the risk is acceptable; conversely, HI < 1.0 is represented as acceptable (Canada, 2004). If the purpose of the research is the preliminary assessment of the quantity of risk, exposures associated with an HQ = 0.2 will be deemed negligible (CCME, 1996; OMEE, 1996; Canada, 2004). Value of HQ > 0.2, or the HI > 1 indicates the risk assessment should either be refined and/or risk management should be undertaken.

2.5.7. Sv

To have a comprehensive estimation, the obtained results from the PEC values were compared with an SV for carcinogenic PAHs. The general equation for estimating SV is as follows (Fairey et al., 1997):

$$SV = [RL/SF - \times BW]/FDC \quad (12)$$

where SV is estimated the screening value (μg.g^{-1}), RL is the highest acceptable risk level (dimensionless), BW is body weight (kg), SF is the USEPA oral slope factor (mg.g.day)$^{-1}$, and FDC is fish daily consumption (g.day^{-1}).

2.5.8. Ecr

Excess cancer risk (ECR) can be estimated in terms of incremental probability of developing cancer over a lifetime of total exposure to potential carcinogen to humans. It was estimated according to Eqs. (13) (Bandowe et al., 2014).

$$ECR = \frac{\Sigma Q \times TEQBaP \times IR \times ED}{BW \times AT} \quad (13)$$

where ED is the exposure duration average time (70 years), TEQ BaP is the toxic equivalence quotient that weights the toxicity relative to that of Benzo(a)pyrene (7.3 mg.kg^{-1}.day^{-1}), IR is ingestion rate (mg.kg^{-1}.day^{-1}), AT is the average life span for carcinogens (25500 days), and BW is the average adult body weight (70 kg).

2.6. Risk assessment by Monte Carlo simulation approach

The Monte Carlo Simulation (MCS) (EPA, 1997) was applied to probabilistic health risk assessment (PRA) based on the quantity of the uncertainties and variabilities in the distribution of the input data. Microsoft Excel was used to create MCS model without uncertainty, then, by using the Easy-Fit Professional software (Version 5.5) and the

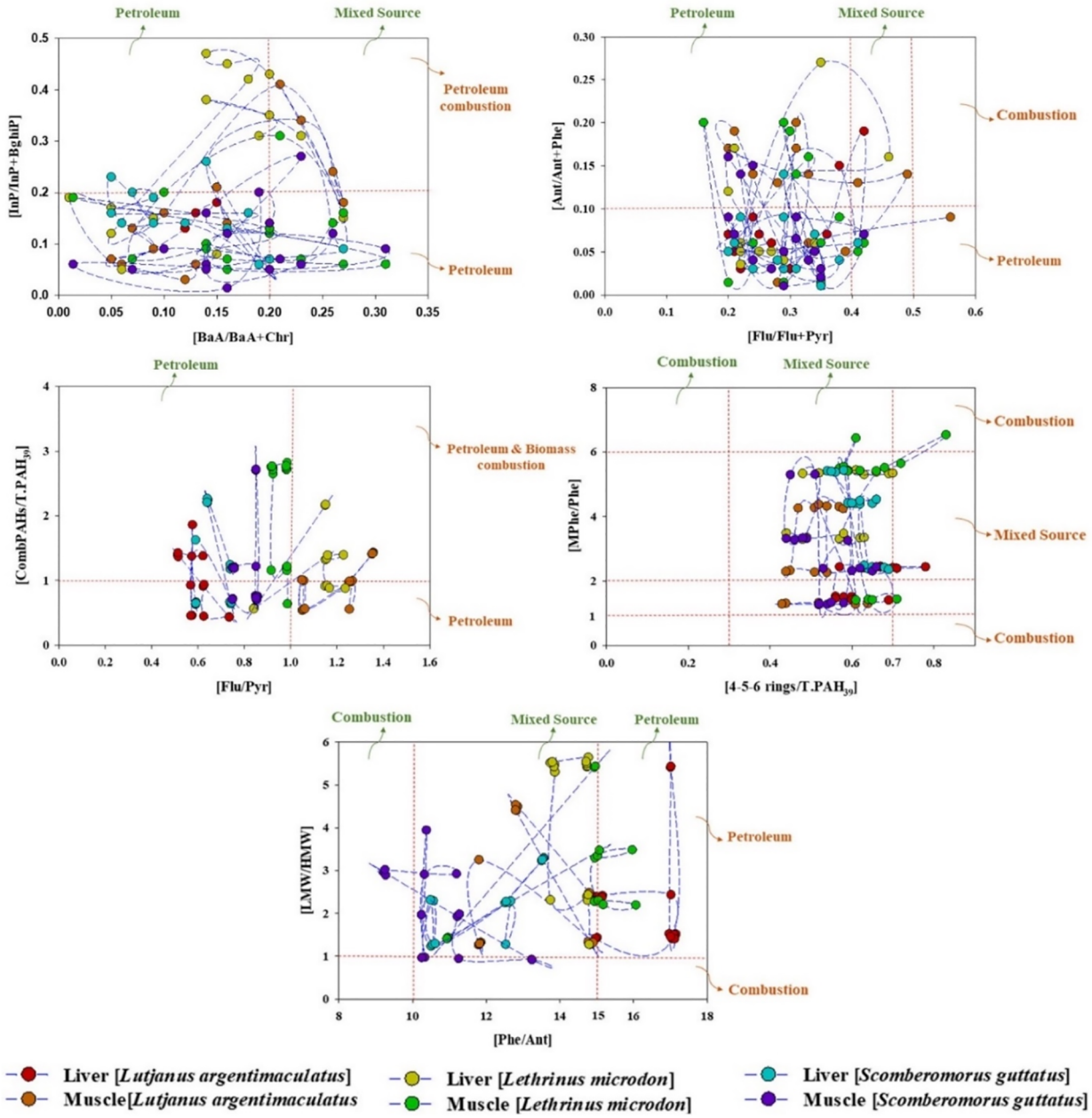

Fig. 3. The PAH parents and their isomers diagnostic ratios for identifying source appointment in three edible fish tissues (muscle and liver) of Kharg coral Island, Iran, Persian Gulf.

spreadsheet-based application @RISK software (Version 7.6), the complete risk assessment model was performed. The analyzed Monte Carlo parameters are summarized in Table 2.

2.7. Statistical analysis

In the present investigation, all data are reported as mean ± SE. All reported concentration for tissues is indicated by the abbreviation fdw (freeze-dried weight) and for abiotic samples by the abbreviation dw (dry weight). Σ_{39}PAHs is the sum of all PAH congeners concentrations. ΣLMWs is the sum of all Low Molecular Weight (LMW) PAHs with 2–3 ring, whereas, ΣHMWs is defined as the sum of all High Molecular Weight (HMW) PAHs with 4–6 ring. Σcarcinogenic–PAHs is defined as the sum of B(a)A, B(k)P, B(a)P, Chr, D(ah)A, BP, In (1,2,3-c, d)P. For normality and equality of variance, Shapiro–Wilk test on residues with 1% risk and Levene test with 5% risk, were carried out, respectively. One-way non-parametric ANOVA (Kruskal-Wallis test) and its post-hoc test (analogous to Bonferroni–Dunn's test) were also applied ($p < 0.05$) for non-normal distribution. By using one-way ANOVA followed by Tukey's HSD test, significant differences in average concentrations with respect to species and fish parts were tested. Pearson's correlation analysis was performed to examine the relationship between the PAH congeners. By categorizing the PAHs into three groups: LMW-PAH (2–3 ring PAHs), medium molecular weight PAHs (MMW-PAH) (4 ring PAHs) and HMW-PAH (5–6 ring PAHs), comparisons among fingerprints were conducted. All statistical analyses were computed by utilizing the R statistical programming environment.

3. Result

3.1. Lipid content and concentration of Σ_{39}PAHs in muscle and liver

Total fat content in the muscles of fish varied between 7.13 and 9.52% fdw (*L. microdon*), 4.31 to 6.92% fdw (*S. guttatus*) and 9.51 to

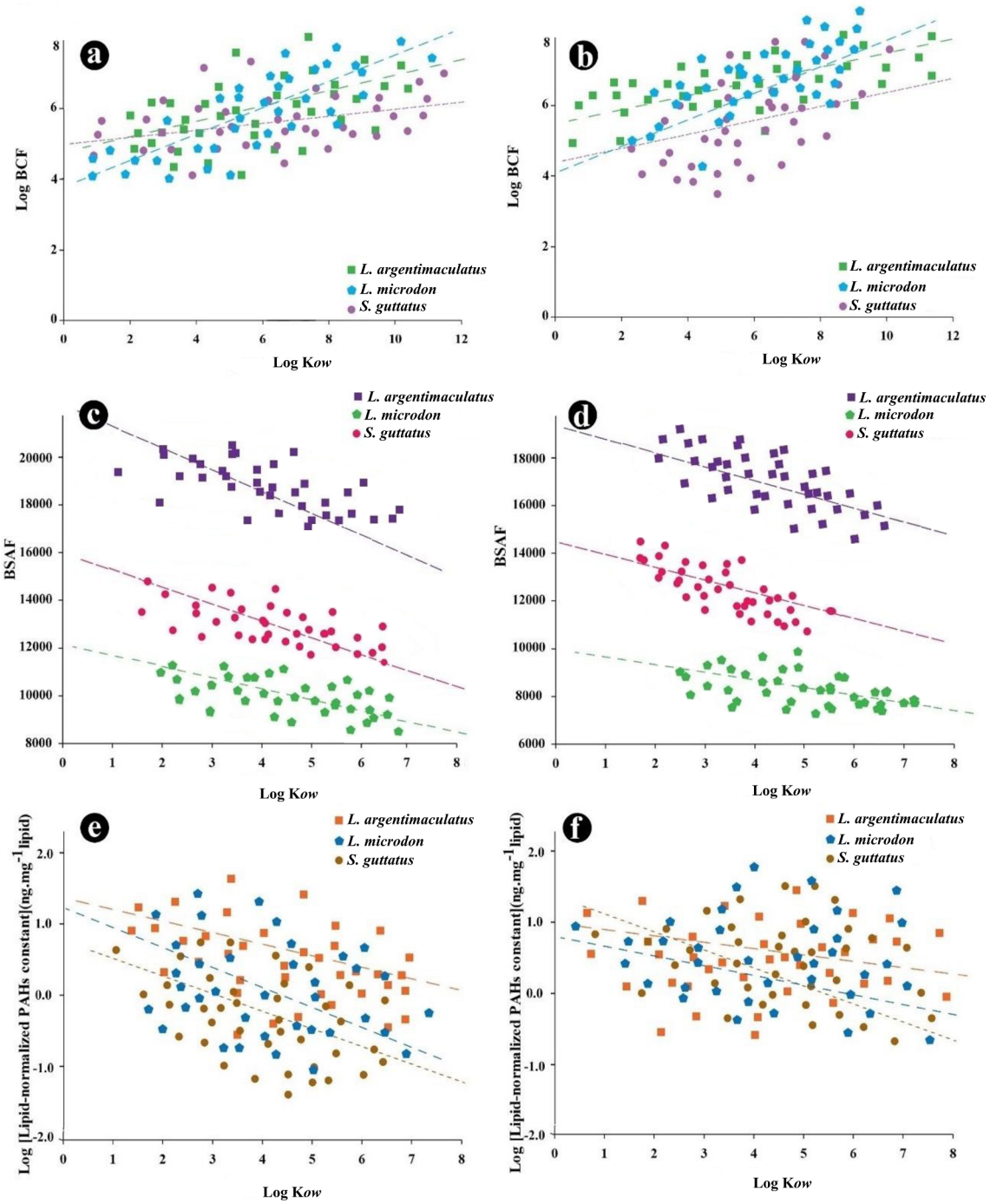

Fig. 4. Relationship between log BCF and log Kow (a = muscle, b = liver), log BSAF and log Kow (c = muscle, d = liver), and log lipid-normalized PAHs constant (e = muscle, f = liver) in three edible fish (*L. argentimaculatus*, *L. microdon*, and *S. guttatus*) in Kharg coral Island, Iran, Persian Gulf.

13.62% fdw (*L. argentimaculatus*). In the livers, total lipids content varied from 7.32 to 8.96% fdw in *S. guttatus*, 10.67 to 14.65% fdw in *L. microdon*, and 12.72 to 17.34% fdw in *L. argentimaculatus*. The mean doses of total PAHs (Σ_{39}PAHs) in liver and muscle were 1004 and 1390 ng g^{-1} fdw in *L. argentimaculatus*, 891 and 1121 ng g^{-1} fdw in *L. microdon* and 726 and 954 ng g^{-1} fdw in *S. guttatus*, respectively (Fig. 2; Table S1).

Significant differences between species were observed ($p < 0.05$). The minimum doses were calculated in the *S. guttatus*, whereas the highest were observed in *L. argentimaculatus* and *L. microdon*. Similar to Σ_{39}PAHs, LMW-PAHs accumulation was different between the species studied ($p < 0.05$). Average doses of LMW-PAHs ranged from 642 ng g^{-1} fdw in muscle to 861 ng g^{-1} fdw in the liver in *L. argentimaculatus*, from 496 (muscle) to 611 ng g^{-1} fdw (liver) in *L. microdon*, and from 379 (muscle) to 570 ng g^{-1} fdw (liver) in *S. guttatus*. A similar trend with lower concentrations was recorded for HMW-PAHs.

3.2. Identification of PAH contamination sources

In our study, InP/(InP + Bghi) and BaA/(BaA + Chr) varied from 0.05 to 0.47 and 0.02 to 0.27, respectively. Ant/(Ant + Phe) ranged from 0.02 to 0.27 and Flu/(Flu + Pyr) varied between 0.18 and 0.57. This indicated that PAHs originated mostly from petroleum and combustion sources. To complete the source identification, the ratio of the

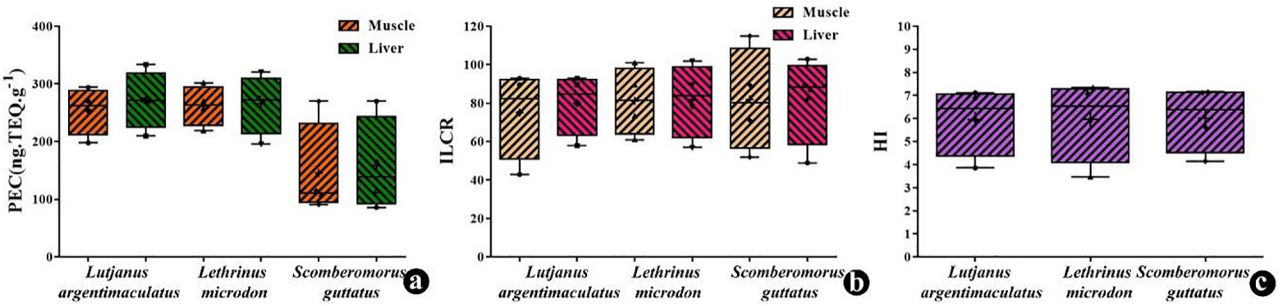

Fig. 5. Calculated PEC (a), ILCR (b), and HI (c) in muscle and liver tissue of fish studied (*L. argentimaculatus*, *L. microdon*, and *S. guttatus*) in Kharg coral Island, Persian Gulf, Iran.

sum of the all methylated phenanthrenes (3 M−Phe + 2 M−Phe + 9Mphe + 1 M−Phe) to phenanthrene was also calculated. It varied from 1.35 to 6.2 in our study area, confirming the strong petrogenic signature of PAHs. However, it was observed that the mean ratios of Flu/Pyr in the species studied were < 1.0 with values varied from 0.56 to 1.3 for both tissues. Phe/Ant was > 10, varied between 10.3 and 17, signing to the dominance of petroleum and biomass combustion originated PAHs (CO-PAHs). If LMW/HMW < 1 for all fish samples (Fig. 3), again suggesting petrogenic sources for PAHs in the samples. Furthermore, CO-PAHs/Σ_{39}PAHs ratios for tissues ranged between 0.4 and 0.82, reflecting the combustion of petroleum.

The results of Pearson correlation analysis between the PAH congeners in muscles and livers in studied fishes revealed that the positive correlations between LMW-PAHs ($r > 0.87$), in particular Naph and Phe ($r = 0.92$), except DBT, and HMW-PAHs ($r > 0.74$) especially DahA and BaP ($r = 0.80$), except BeP, and BbF. These stronger correlations between LMW-PAHs and HMW-PAHs implied that their analogous sources, whereas for HMW-PAHs seem that there are both pyrogenic and petrogenic origins. The pyrolytic originated PAHs (Pr-PAHs) are dominated by HMW- PAHs; while those of the petrogenic originated PAHs (Pt-PAHs) are characterized by LMW-PAHs.

3.3. Distribution and mechanism of bioaccumulation of deleted PAHs in tissues

Calculated BSAFs for individual PAHs were overall high (9613 for *S guttatus*, 13,192 for *L microdon* and 20,181 for *L argentimaculatus*) (Fig. 4c,d). Mean BSAF values showed significantly variation (One-way ANOVA, $p < 0.05$). In all surface sediments samples, with increasing log K_{ow} ($p < 0.05$), especially for PAHs with log $K_{ow} > 5$, BSAF decreased. Similar to BSAF, the maximum and minimum calculated BCF was observed in *L. argentimaculatus* (liver: 8.06; muscle: 7.57) and *S. guttatus* (liver: 7.65; muscle: 7.16) (Fig. 4 a, b). Values of Log BCF demonstrated a positive linear correlation to log K_{ow} ($p < 0.05$). This linear relationship demonstrated an essentially passive process based on partitioning between PAHs freely dissolved in pore water and the lipophilic phase of the organism.

3.4. Health notion regarding fish consumption

The result of PECs analysis of Σ_{39}PAHs in three species of edible fish of Kharg Island were above the safety human consumption guidelines of 2.6 ng g^{-1}ww (Fig. 5a). The maximum values of PEC were found in the liver of *L. argentimaculatus* and *L. microdon*. If ILCR is $\leq 1 \times 10^{-5}$, carcinogenic risks will be essentially negligible, but, if ILCR $> 1 \times 10^{-5}$, then the risk assessment should either be refined and/or a risk management should be undertaken. In this study, ILCR was $> 1 \times 10^{-5}$ documenting high potential risk for cancer for local adults who consume fish. Maximum levels of ILCR are detected in the liver and muscle of *S. guttatus* (Fig. 5b). Additionally, values of HQ > 1 were found for all measured PAHs indicating potential negative effects on residents' health.

The highest HI values were similar in all three fish species, while the lowest values were found in *L. microdon* (Fig. 5c). According to EPAs recommendations for SV, BW and CR values used were 70 kg and 142.4 g.day^{-1}, respectively (USEPA., 2000). However, for Kharg Island, CR has been estimated at 55.1 g.day^{-1} (Nozar et al., 2013). Therefore, SV for Kharg Island is 1.89 ngg^{-1}. Comparison of the obtained B(a)P_{eq} and calculated SV for Kharg local consumers revealed that the studied reef seafood contained B[*a*]P equivalents higher than SV, therefore vigorous effort to strengthen risk management is needed. The results of ECR values from PAHs from fish consumption are represented in Table 1. Compared to the screening criteria for carcinogen risk proposed by the EPA, all the samples exceeded the acceptable risk level of 1×10^{-6} above which, consequences are expected to occur.

3.5. Uncertainty analysis

Cumulative probability distribution curve and health risk estimation histograms from the MCS for the consumption of *L. argentimaculatus*, *L. microdon*, and *S. guttatus* from the Kharg coral Island are showed in Fig. 6, which reflects the highest toxicological dietary risk for these three reefs edible fish. Based on the probabilistic risk analysis of the Monte-Carlo simulation, all health indices presented a positive effect, but ADD of *L. argentimaculatus* is the greatest one followed by the carcinogenic risk of *L. microdon*, and *S. guttatus* (Table 2). Although, for the uncertainties of the PAH-exposure risk parameters, the results of the MCS presented no positive effect for ADD of *L. microdon*, and *S. guttatus,* but, *L. microdon* had the highest hazard quotient in this area (Table 2).

4. Discussion

Aquatic environments are increasingly polluted worldwide which can impact not only marine life but human health. In Kharg Island, the doses of Σ_{39}PAHs in *L. argentimaculatus* were higher than another species and among 39 PAH congeners analyzed, the 2-Methylphenanthrene (2MeP) and Dibenz [a,h]anthracene (BahA) were found to be the predominant PAHs (liver > muscle) (Table S1). These results could be due to different reasons such as metabolism kinetics, lipid content, bioaccumulation rate, size, and age (Pointet & Milliet, 2000; Agency for Toxic Substances and Disease Registry (ATSDR), 1995). The values obtained for MAC of BaP, and PECs of total PAHs in all species studied were higher than the European Union standard value for BaP (0.0049 mg/kg) as an indicator of PAHs-carcinogenesis in fish muscle meat (European Union & Regulation, 2006). Regarding EC recommendation 1881/2006 for maximum admissible concentration of BaP in fish muscle (2 ng/g ww), in all species studied muscle tissues was not a target tissue for PAHs accumulation, which is consistent with previous BaP-exposure tests showing that doses of PAHs in liver can be 100 times more than in muscle (Varanasi & Stein, 1991). In the case of lipophilicity of PAHs to accumulate in lipids from fish/tissue, there were previous related studies that reported the liver and muscle tissue

Table 1
Calculated Cancer Slope Factor (CSF) mg/kg-day, Reference Dose (RfD) mg/kg-day, Average Daily Dose (ADD), Hazard Quotient (HQ), Hazard Index (HI), and cancer risk by consumption of *L. argentimaculatus*, *L. microdon*, and *S. guttatus* caught from the Kharg coral Island, Persian Gulf, Iran.

Fish species	PAHs	HI	Nap	Ace	Fl	Phe	Ant	Flu
L. argentimaculatus								
(n = 17)	CSF	0.73 ± 0.02	–	0.73 ± 0.04	–	–	–	–
	RfD	0.041 ± 0.02	0.06 ± 0.03	0.04 ± 0.01	0.04 ± 0.02	0.3 ± 0.02	0.04 ± 0.01	0.03 ± 0.01
	ADD	0.031 ± 0.03	0.032 ± 0.02	0.041 ± 0.02	0.040 ± 0.02	0.031 ± 0.02	0.031 ± 0.02	0.033 ± 0.02
	HQ_S	0.81 ± 0.01	0.56 ± 0.04	0.87 ± 0.03	0.89 ± 0.03	0.10 ± 0.03	0.78 ± 0.03	0.10 ± 0.03
	Cancer risk	0.02 ± 0.00	–	–	–	–	–	–
L. microdon								
(n = 18)	CSF	0.73 ± 0.03	–	0.73 ± 0.04	–	–	–	–
	RfD	0.041 ± 0.04	0.06 ± 0.03	0.04 ± 0.03	0.04 ± 0.03	0.3 ± 0.04	0.04 ± 0.01	0.03 ± 0.00
	ADD	0.034 ± 0.01	0.032 ± 0.02	0.034 ± 0.02	0.034 ± 0.02	0.030 ± 0.00	0.031 ± 0.02	0.032 ± 0.01
	HQ_S	0.84 ± 0.02	0.54 ± 0.05	0.85 ± 0.00	0.87 ± 0.05	0.10 ± 0.03	0.79 ± 0.04	1.07 ± 0.05
	Cancer risk	0.01 ± 0.00	–	0.01 ± 0.00	–	–	–	–
S. guttatus								
(n = 15)	CSF	0.73 ± 0.05	–	0.73 ± 0.02	–	–	–	–
	RfD	0.041 ± 0.03	0.06 ± 0.01	0.04 ± 0.03	0.04 ± 0.02	0.3 ± 0.03	0.04 ± 0.01	0.03 ± 0.00
	ADD	0.034 ± 0.02	0.029 ± 0.01	0.033 ± 0.03	0.034 ± 0.03	0.029 ± 0.01	0.029 ± 0.00	0.029 ± 0.01
	HQ_S	0.83 ± 0.03	0.49 ± 0.03	0.82 ± 0.04	0.85 ± 0.02	0.09 ± 0.01	0.72 ± 0.04	0.96 ± 0.06
	Cancer risk	0.01 ± 0.00	–	0.01 ± 0.00	–	–	–	–

Fish species	Pyr	BaA	Chr	BbF	BkF	BaP	DahA	BghiP	I(1,2,3cd)P
L. argentimaculatus									
(n = 17)	0.73 ± 0.03	0.0073 ± 0.00	0.73 ± 0.09	0.073 ± 0.01	7.3 ± 0.05	7.3 ± 0.02	–	0.73 ± 0.02	
	–	–	–	–	–	–	0.04 ± 0.01	–	
	0.030 ± 0.02	0.032 ± 0.01	0.030 ± 0.00	0.030 ± 0.02	0.033 ± 0.02	0.032 ± 0.03	0.033 ± 0.03	0.031 ± 0.03	
	–		–	–	–	–	0.84 ± 0.07	–	5.95
	0.02 ± 0.00	0.0002 ± 0.00	0.02 ± 0.01	0.002 ± 0.00	0.24 ± 0.03	0.23 ± 0.05	–	0.02 ± 0.01	
L. microdon									
(n = 18)	0.73 ± 0.05	0.0073 ± 0.00	0.73 ± 0.02	0.073 ± 0.03	7.3 ± 0.06	7.3 ± 0.06	–	0.73 ± 0.04	
	–	–	–	–	–	–	0.04 ± 0.01	–	
	0.030 ± 0.01	0.031 ± 0.02	0.030 ± 0.01	0.030 ± 0.02	0.030 ± 0.04	0.030 ± 0.02	0.034 ± 0.03	0.030 ± 0.03	
	–		–	–	–	–	0.85 ± 0.09	–	5.95
	0.01 ± 0.00	0.0001 ± 0.00	0.01 ± 0.00	0.001 ± 0.00	0.13 ± 0.08	0.13 ± 0.07	–	0.01 ± 0.00	
S. guttatus									
(n = 15)	0.73 ± 0.03	0.0073 ± 0.00	0.73 ± 0.03	0.073 ± 0.03	7.3 ± 0.06	7.3 ± 0.06	–	0.73 ± 0.06	
	–	–	–	–	–	–	0.04 ± 0.02	–	
	0.029 ± 0.04	0.032 ± 0.01	0.029 ± 0.04	0.029 ± 0.04	0.031 ± 0.03	0.030 ± 0.03	0.033 ± 0.03	0.029 ± 0.02	
	–		–	–	–	–	0.82 ± 0.05	–	5.63

(*continued on next page*)

Table 1 (*continued*)

Fish species	Pyr	BaA	Chr	BbF	BkF	BaP	DahA	BghiP	I(1,2,3cd)P
	0.01 ± 0.00	0.0001 ± 0.00	0.01 ± 0.00	0.001 ± 0.00	0.13 ± 0.03	0.13 ± 0.05	–	0.01 ± 0.00	

Naphthalene (Nap), Acenaphthene (Ace), Fluoranthene (Fl), Phenanthrene (Phe), Anthracene (Ant), Fluorene (Flu), Pyrene (Pyr), Benzo(a)anthracene (BaA), Chrysene (Chr), Benzo(b)fluoranthene (BbF), Benzo(k)fluoranthene (BkF), Benzo[*a*]pyrene (BaP), dibenzo[*a,h*]anthracene (DahA), Benzo[*g,h,i*]perylene(BghiP), Indeno[1,2,3-*cd*]pyrene (I(1,2,3cd) P), hazard index (HI). NA not available/ND not determined (–).

of mangrove red snapper (*L. argentimaculatus*) contained relatively high level of docosahexaenoic acid (DHA), eicosapentaenoic acid (EPA), and arachidonic acid (ARA) (Ogata et al., 2004) that may be caused to lipid peroxidation (LPO). Most of the PAHs identified in coral fish of Kharg Island were of LMW. The presence of LMW-PAHs could be due to their higher solubility in the aqueous medium than the HMW-PAHs, which can increase their bioavailability to fish or other aquatic organisms through absorption, adsorption, and ingestion (Agency for Toxic Substances and Disease Registry (ATSDR), 1995).

In Iran and the Arab Gulf States, the consumption of *L. argentimaculatus*, *L. microdon*, and *S. guttatus* is very popular due to the high-quality of the meat and is sold fresh daily on the market. Fish stock of these three species are thought to be overfished in the Persian Gulf (Al Zeyoudi & Al Dhaheri, 2019; Anulekshmi et al., 2018; Al-Abdulrazzak, Zeller, Belhabib, Tesfamichael, & Pauly, 2015). This event indicates that balancing between nutritional value and eco-health risks of these three species of fish for the consumption of the local community is a key issue and challenging field in fisheries legislation.

The presented results are in agreement with previous studies showing that the most abundant PAHs in seafood are LMW-PAHs (Σ2-3 aromatic rings PAHs) (Zelinkova and Wenzl, 2015). HMW-PAHs (Σ4-7 aromatic rings PAHs) have more carcinogenic risks than PAHs with lower ring systems (International Agency for Research on Cancer (IARC), 2010). The six PAH isomer pair ratios indicated that PAHs originated mostly from petroleum and combustion sources such as industrial wastewater discharges, shipping oil spills, land runoff, riverine inputs, port activities, fishing boats, waste incineration, Island sewage drainage discharges and diffuse atmospheric fall out. Such a strong petrogenic signature of PAHs confirmed by the abundance of LMW-PAHs is reported in most marine environment, industrial countries, and zones historically containing coal/oil tar production, processing and operations (Ranjbar Jafarabadi et al., 2017; Uno et al., 2017).

In the liver, PAH residues were higher than muscle. This difference results from various factors including the nutrition and metabolism rate of these fish, time of exposure and fat tissues (Rose et al., 2012). All fish analyzed contained detectable amounts of BaP that are used as marker for screening carcinogenic risks of PAHs in human food. Additionally, the significant levels of some of PAHs (Table S1) commonly found were lower than in the other tissues analyzed (Shappell et al., 2003), which may be attributed to high liver metabolic activity in these fish. In fish, liver tissue is a good proxy for fish contamination levels, because major contaminants are first absorbed by the digestive system and then released into the liver for biotransformation and detoxification (Lazartigues et al., 2010). In the liver detoxification pathways (phase I), PAHs can be oxidized to epoxides and hydroxylated by biotransformation enzyme systems that typically involve a cytochrome P450 monooxygenase enzymes (P450s/CYP). During phase II of hepatic PAHs metabolism, to facilitate excretion of toxic and unstable products, these secondary metabolites are quickly converted to ionic compounds such as glucuronides or sulfates that dissolved in water and excretion occurs via the bile or urine. But when the level of the PAHs contamination and its metabolites is very high, the healthy metabolic function of liver is disrupted. This could lead to a short-term accumulation of LMW-PAHs in lipid-rich liver before being metabolized (Nwaichi & Ntorgbo, 2016) or induce hepatic cell damages/liver failure, which caused to release a high percentage of unmetabolized PAHs into the other tissues, including the muscle. By increasing the levels of waterborne PAHs and PAH-metabolites in the liver, GSH content and the ratio of GSH/GSSG is increased to protect against ROS and to maintain the normal function of cells (Zhu et al., 2013).

The result of BSAFs analysis of PAH congener showed that this index was high, but significant differences between species were observed. BSAF decreased with increasing log K_{ow}, especially for PAHs with log $K_{ow} > 5$, in all surface sediments sample. Observing this variation with log K_{ow} reflects stronger affinity of PAHs compound to organic carbon associated with sediments and increasing their hydrophobic properties

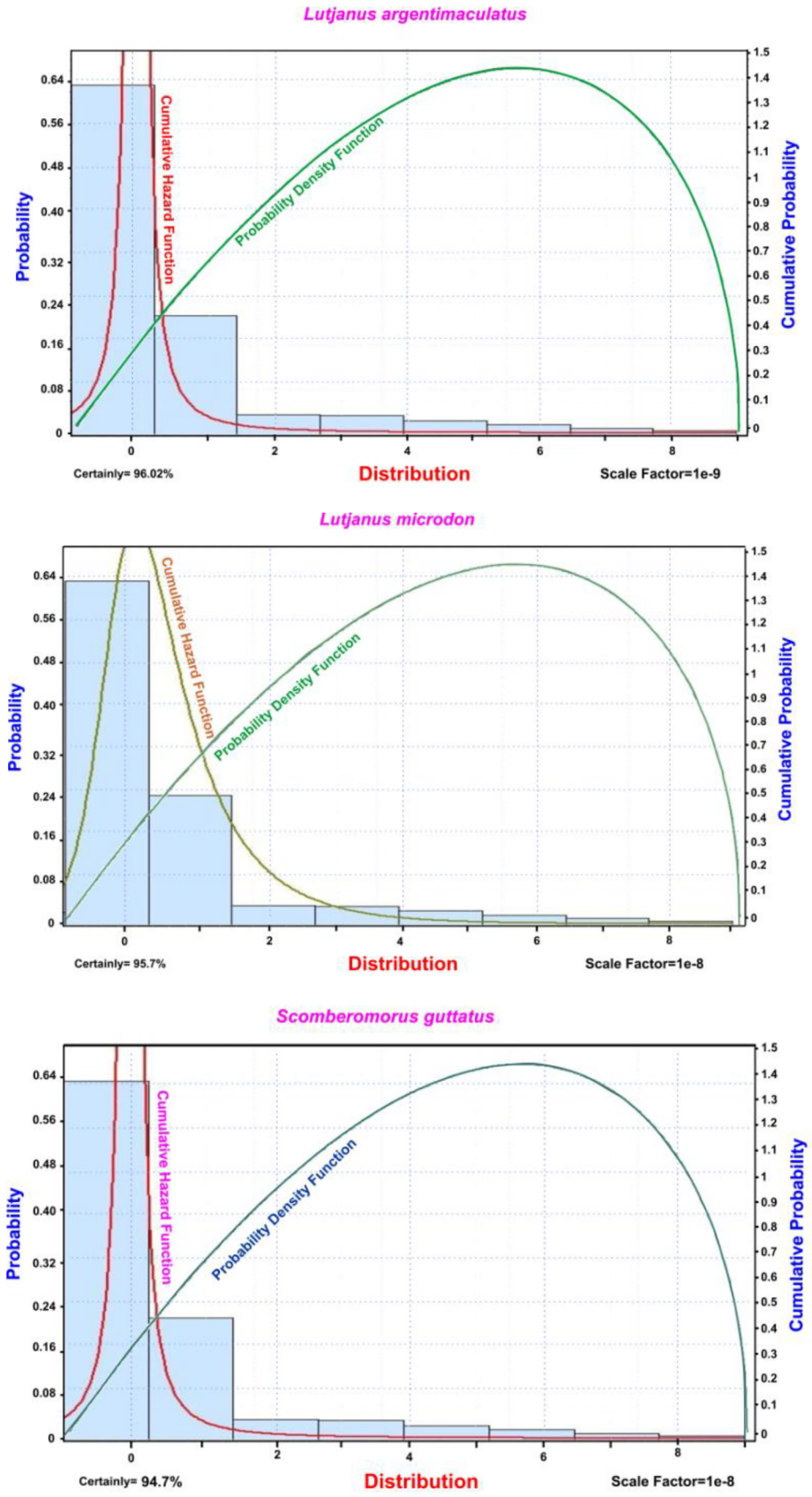

Fig. 6. Cumulative probability analysis for risk assessment via consumption of *L. argentimaculatus*, *L. microdon*, and *S. guttatus* caught from the Kharg coral Island, Persian Gulf, Iran.

Table 2
Monte Carlo parameters for deterministic and probabilistic health risk approaches via consumption of *L. argentimaculatus*, *L. microdon*, and *S. guttatus* caught from the Kharg coral Island, Persian Gulf, Iran.

Fish species	Parameters	Distribution	Mean	SD	Min	Max	Uncertainty range
L. argentimaculatus	CSF	Log-normal	2.641	3.299	−10.984	11.309	21.8% − 28.2%
	RfD	Log-normal	0.069	0.093	−0.219	0.348	18.2% − 24.2%
	ADD	Log-normal	0.032	0.003	0.022	0.042	100%
	HQ_S	Log-normal	0.635	0.338	−0.267	1.708	2.2% − 5.6%
	Cancer risk	Log-normal	0.063	0.102	−0.251	0.331	23.6% − 28.2%
L. microdon	CSF	Log-normal	2.355	2.885	−5.026	10.279	20.2% − 27.4%
	RfD	Log-normal	0.081	0.096	−0.177	0.415	17.2% − 26.8%
	ADD	Log-normal	0.031	0.001	0.026	0.036	0%
	HQ_S	Log-normal	0.738	0.001	0.732	0.743	0%
	Cancer risk	Log-normal	0.075	0.111	−0.271	0.409	23.2% − 30.8%
S. guttatus	CSF	Log-normal	2.431	3.068	−7.258	12.115	19.8% − 27.6%
	RfD	Log-normal	0.072	0.091	−0.250	0.411	18.6% − 25.8%
	ADD	Log-normal	0.030	0.002	0.024	0.036	0%
	HQ_S	Log-normal	0.680	0.265	−0.130	1.599	0.4% − 1.4%
	Cancer risk	Log-normal	0.034	0.053	−0.119	0.204	23.2% − 28.6%

Note: In this study, the indices of RfD and CSF are not-constant according to the computing of Monte Carlo.

which may considerably mitigate the PAH bioavailability to fish (Rust et al., 2004). It could be inferred that such high values of BSAF may not depend on the PAHs metabolism in fish or to the major origin of PAHs (Thorsen et al., 2004) but with pyrolytic PAHs being suggested as less available than petrogenic PAHs. Regarding the risk of PAHs-bio-uptake, marine-derived feeds such as fish are chemical-contaminants' carriers from the aquatic environment to edible fish tissues for human consumption, that easily undergo food-chain enrichment. It is apparent from Fig. 4 that the BCF values for the partitioning of PAHs showed a strong correlation with their log K_{ow} (octanol–water). Considering that LMW-PAHs with $5 < \log K_{ow} < 8$ at low metabolic transformation rates(mtr) ($\log kmtr \leq 1.25\ day^{-1}$ normalized to 10 g tissue weight) (Khairy et al., 2019) could be net accumulated in aquatic food, there is a concern about the ability of PAHs-type pollutants and other lipophilic oiled products to magnify in fish tissues. The present results confirmed that doses of PAH congener in muscles of fish were positively correlated with values of log K_{ow} in all fish sampled. It is suggested that the main physiological mechanism for PAHs-bioaccumulation in reef fish is in equilibrium between the alkyl/parent-PAHs doses in the fish tissues and water/sediment. However, exposure to environmental contaminants is usually not an exposure to a single compound but to a mix of PAH-components that can be metabolized by a single or several pathways (Abdel-Shafy & Mansour, 2016). PAHs and their metabolites can cause public concern due to their immune-toxicity and carcinogenicity. This was the case in this study. Indeed, the human health risk assessment conducted using BaP toxicity (a biomarker of ILCR, HQ, HI, and ECR) in Kharg Island, Iran, indicated a high carcinogenic potential risk of PAH contamination with coral-reef fish consumption. Similar findings were reported by Tongo et al. (2018) for high carcinogenic potency of BaP in humans from PAH-exposed consumption of mullet fish *Mugil cephalus* ($0.0049\ mg\ kg^{-1}$), prawn *Penaeus monodon* ($0.0047\ mg\ kg^{-1}$), and crab *Uca tangeri* (0.0038 mg/kg). Furthermore, it has been reported that PAH, when metabolized to the diol epoxides of BaPDE (benzo[*a*] pyrene-7,8-diol-9,10-epoxide), may react with DNA through the BaPDE binding with nucleophilic centers of exocyclic amino groups of purines, thus inducing DNA adducts or other genotoxic damage (Abdel-Shafy & Mansour, 2016). However, there is limited ecotoxicological information about health risk assessment of PAHs through dietary components (Xia et al., 2010) and mechanisms of PAH induced immune-suppression or genetic alterations are still unclear. Some regulatory agencies set a standard for quantitative risk assessment of PAHs (especially for BaP) in food, because BaP is the only PAH for which toxicology data network (TOXNET) are sufficient for derivation of a cancer potency factor (CPF) among all known other PAHs that may cause cancer. The European Commission's Scientific Committee on Food (SCF) in their assessment of the 33 PAHs have identified 15 PAHs with carcinogenic and genotoxic/mutagenic properties (Wenzl et al., 2006). In the present study, the MAC of BaP is $5\ ng\ g^{-1}$ ww, which is > 8 times higher than that of BaP doses in most of the samples we collected. In addition, the PECs of Σ_{39}PAHs in all fish species studied were higher than human health SV ($2.6\ ng\ g^{-1}$ ww). Therefore, accurate assessment of the possible dangers of PAHs in Kharg reef food-groups is necessary and this will guide to choose a well PAHs remediation strategy.

From the view of the biomathematical modeling of Monte Carlo, the order of multiple health risks in studied edible fish was basically consistent with human carcinogenic health hazard values retrieved from the International Toxicity Estimates for Risk Assessment (ITER) database (http://www.iter.tera.org). Coherently, a comparison between the risk parameters related to the contaminants in the sea diet, and probability model revealed that some toxicity indices, such as RfD and CSF, are not constant in the computing of MCS, and it might have an effective on the distribution functions of toxicity values for PAHs compounds. From this perspective, the possibility of PAHs-contamination occurrence in Kharg Island should be reduced by performing specific environmental monitoring and fisheries management authorities.

5. Conclusion

In the current study, the doses and origin of PAHs and risks attributable to PAH dietary intake were investigated in fifty edible coral fish from Kharg Island, Persian Gulf, Iran. Most of the fish samples were contaminated with petrogenic originated PAHs (Pt-PAHs). The concentrations of Σ_{39}PAHs in liver tissue of *L. argentimaculatus* were higher than other fish. These results could be due to different reasons such as metabolism kinetics, lipid content, bioaccumulation rate, size, and age. Pearson correlation revealed positive correlations between the LMW-PAHs ($r > 0.87$), in particular Naph and Phe ($r = 0.92$) and among the HMW-PAHs ($r > 0.74$) especially DahA and BaP ($r = 0.80$). These positive correlations among LMW- PAHs, and HMW-PAHs indicates their similar origin. Compared to the screening criteria recommended by the EPA, the carcinogen risk of PAHs in all samples exceeded acceptable levels above which consequences are expected to occur. Comprehensive management plans to mitigate the situation are needed urgently.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

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References

Abdel-Shafy, H., & Mansour, M. S. M. (2016). A review on polycyclic aromatic hydrocarbons: Source, environmental impact, effect on human health and remediation. *Egyptian Journal of Petroleum, 25*(1), 107–123.

Agency for Toxic Substances and Disease Registry (ATSDR)(1995). Toxicological profile for polycyclic aromatic hydrocarbons. Atlanta, GA: Centers for Disease Control and Prevention.

Al-Abdulrazzak, D., Zeller, D., Belhabib, D., Tesfamichael, D., & Pauly, D. (2015). Total marine fisheries catches in the Persian/Arabian Gulf from 1950 to 2010. *Regional Studies in Marine Science, 2*, 28–34.

Al Zeyoudi, T., & Al Dhaheri, S. (2019). *The UAE National Framework Statement for Sustainable Fisheries (2019–2030).* United Arab Emirates: Minister of Climate Change and Environment https://www.ead.gov.ae/storage/Post/files/8207d3c3daec608-b12636ef7ab2793ff.pdf.

Anulekshmi, C., Sarang, J. D., Kamble, S. D., Akhilesh, K. V., Deshmukh, V. D., & Singh, V. V. (2018). Biological aspects of spotted seerfish Scomberomorus guttatus (Bloch & Schneider, 1801) (Scombridae) from north-eastern Arabian Sea. *Indian Journal of Fisheries. 65*(2), 42–49.

Bandowe, B. A. M., Bigalke, M., Boamah, L., Nyarko, E., Saalia, F. K., & Wilcke, W. (2014). Polycyclic aromatic compounds (PAHs and oxygenated PAHs) and trace metals in fish species from Ghana (West Africa): Bioaccumulation and health risk assessment. *Environment International, 65*, 135–146.

Canada, H. (2004). Development of a resource model for infection prevention and control programs in acute, long term, and home care settings: Conference proceedings of the Infection Prevention and Control Alliance. *American journal of infection control, 32*, 2–6.

CCME, A. (1996). Protocol for the Derivation of Environmental and Human Health Soil Quality Guidelines. Canadian Council of Ministers of the Environment, Winnipeg.

Cerrillo, R. M. N., Vieira, D. J. E., Ochoa-Gaona, S., de Jong, B. H. J., & Serrano, M. M. D. (2019). Land cover changes and fragmentation in mountain neotropical ecosystems of Oaxaca, Mexico under community forest management. *Journal of Forestry Research, 30*(1), 143–155.

Chien, L.-C., Hung, T.-C., Choang, K.-Y., Yeh, C. Y., Meng, P. J., Shieh, M. J., & Ha, B. C. (2002). Daily intake of TBT, Cu, Zn, Cd and As for fishermen in Taiwan. *Science of the Total Environment, 285*, 177–185.

EPA (1997). *Guiding Principles for Monte Carlo Analysis.* Washington, DC 20460: Risk Assessment Forum, U.S. Environmental Protection Agency EPA/630/R-97/001.

EU, 2006. European Union. Commission Regulation (EC) No 1881/2006 of 19December 2006 setting maximum levels for certain contaminants infoodstuffs [Official Journal L 364 of 20.12.2006] http://europa.eu/legislationsummaries/consumers/productlabellingandpackaging/l21101aen.htm.(retrieved 16.1013.).

Fairey, R., Taberski, K., Lamerdin, S., Ross, E., James, P. C., Downing, J. W., ... Petreas, M. (1997). Organochlorines and other environmental contaminants in muscle tissues of sportfish collected from San Francisco Bay. *Marine Pollution Bulletin, 34*, 1058–1071.

FAO (2015). Fishery and aquaculture country profiles: The Islamic Republic of Iran. Accessed 15 Nov 2016 http://www.fao.org/fishery/facp/IRN/en.

Ferrante, M., Zanghì, G., Cristaldi, A., Copat, C., Grasso, A., Fiore, M., Zuccarello, P., ... Oliveri Conti, G. (2018). PAHs in seafood from the Mediterranean Sea: An exposure risk assessment. *Food and Chemical Toxicology. 115*, 385–390.

Frédéric, O., & Yves, P. (2014). Pharmaceuticals in hospital wastewater: Their ecotoxicity and contribution to the environmental hazard of the effluent. *Chemosphere, 115*, 31–39.

Honda, M., & Suzuki, N. (2020). Toxicities of polycyclic aromatic hydrocarbons for aquatic animals. *International Journal of Environmental Research and Public Health, 17*, 1363.

IARC. (2010). IARC monographs on the evaluation of carcinogenic risks to humans. Ingested nitrate and nitrite, and cyanobacterial peptide toxins. https://monographs.iarc.fr/wp-content/uploads/2018/06/mono94.pdf.

Iran Fisheries Organization (IFO) (2019). *Annual Iranian Fisheries Statistics 2013 to 2018.* Tehran, Iran: Fisheries Design and Program Office33.

Khairy, M. A., Noonan, G. O., & Lohmann, R. (2019). Uptake of hydrophobic organic compounds, including organochlorine pesticides, polybrominated diphenyl ethers, and perfluoroalkyl acids in fish and blue crabs of the lower Passaic River, New Jersey, USA. *Environmental Toxicology and Chemistry, 38*(4), 872–882.

Lazartigues, A., Thomas, M., Brun-Bellut, J., & Cyril, F. (2010). Bioaccumulation of PAHs and their hydroxylated metabolites in common carp (Cyprinus carpio Linnaeus 1758) in controlled environment. *Polycyclic Aromatic Compounds, 30*, 141–152.

Loh, A., Yim, U. H., Ha, S. Y., & Kim, M. (2017). Contamination and human health risk assessment of polycyclic aromatic hydrocarbons (PAHs) in oysters after the Wu Yi San oil spill in Korea. *Archives of Environmental Contamination and Toxicology, 73*, 103–117.

Mahugija, J. A., & Njale, E. (2018). Effects of washing on the polycyclic aromatic hydrocarbons (PAHs) contents in smoked fish. *Food Control. 93*, 139–143.

Miao, Q. (2015). *Polycyclic arenes and heteroarenes: Synthesis, properties, and applications.* Weinheim: Wiley-VCH.

Mirvakili, H. S., & Hadjizadeh Zaker, N. (2014). Evaluation of PAHs in hydrocarbons pollution, biodegradation and weathering extent in surface sediments of the Kharg Island in the Persian Gulf. *International Journal of Environmental Research, 8*(2), 387–394.

Moorthy, B., Chu, C., & Carlin, D. J. (2015). Polycyclic aromatic hydrocarbons: From metabolism to lung cancer. *Toxicological Sciences, 145*(1), 5–15.

Mirza, R., Mohammadi, M., Dadolahi Sohrab, A., Safahieh, A., Savari, A., & Hajeb, P. (2012). Polycyclic aromatic hydrocarbons in seawater, sediment, and rock oyster *Saccostrea cucullata* from the Northern part of the Persian Gulf (Bushehr Province). *Water, Air, and Soil Pollution, 223*, 189–198.

NBSC, N. (2008) China energy statistical Yearbook 2008. Beijing: China Statistics Press.

Nisbet, I. C., & LaGoy, P. K. (1992). Toxic equivalency factors (TEFs) for polycyclic aromatic hydrocarbons (PAHs). *Regulatory Toxicology and Pharmacology, 16*, 290–300.

Nozar, S. L. M., Ismail, W. R., & Zakaria, M. P. (2013). Residual concentration of PAHs in Seafood from Hormozgan Province, Iran: Human health risk assessment for urban population. *International Journal of Environmental Science and Development, 4*, 393.

Nwaichi, E. O., & Ntorgbo, S. A. (2016). Assessment of PAHs levels in some fish and seafood from differentcoastal waters in the Niger Delta. *Toxicology Reports, 3*, 167–172.

Ogata, H. Y., Emata, A. C., Garibay, E. S., & Furuita, H. (2004). Fatty acid composition of five candidate aquaculture species in Central Philippines. *Aquaculture, 236*(1–4), 361–375.

Ontario Ministry of Environment and Energy (OMEE) (1996). Guidance on Site Specific Risk Assessment for Use at Contaminated Sites in Ontario. Version 1.0. Ontario Ministry of Environment and Energy Standards Development Branch. ISBN: 07778405803.

Pointet, K., & Milliet, A. (2000). PAHs analysis of fish whole gall bladdersand liver from the Natural Reserve of Camargue by GC/MS. *Chemosphere, 40*, 293–299.

Ranjbar Jafarabadi, A., Riyahi Bakhtiari, A., Aliabadian, M., & Shadmehri Toosi, A. (2017). Spatial distribution and composition of aliphatic hydrocarbons, polycyclic aromatic hydrocarbons and hopanes in superficial sediments of the coral reefs of the Persian Gulf, Iran. *Environmental Pollution, 224*, 195–223.

Ranjbar Jafarabadi, A., Riyahi Bakhtiari, A., Yaghoobi, Z., Yap, C. K., Maisano, M., & Cappello, T. (2019). Distributions and compositional patterns of Polycyclic Aromatic Hydrocarbons (PAHs) and their derivatives in three edible fishes from Kharg coral Island, Persian Gulf, Iran. *Chemosphere, 215*, 835–845.

Ranjbar Jafarabadi, A., Mashjoor, S., Mohamadjafari Dehkordi, S., Riyahi Bakhtiari, A., & Cappello, T. (2020). Steroid Fingerprint Analysis of Endangered Caspian Seal (Pusa caspica) through the Gorgan Bay (Caspian Sea). *Environmental Science & Technology*. https://doi.org/10.1021/acs.est.0c01479 https://pubs.acs.org/doi/10.1021/acs.est.0c01479.

Ranjbar Jafarabadi, A., Riyahi Bakhtiari, A., Aliabadian, M., Hedouin, L., Shadmehri Toosi, A., & Yap, C. K. (2018). First report of bioaccumulation and bioconcentration of aliphatic hydrocarbons (AHs) and persistent organic pollutants (PAHs, PCBs, and PCNs) and their effects on Alcyonacea and scleractinian corals and their endosymbiotic algae from the Persian Gulf, Iran: Inter and intra-species differences. *Science of the Total Environment, 627*, 141–157.

Riddle, M. R. (1989). Bite profiles of some benthic grab samplers. *Estuarine, Coastal and Shelf Science, 29*(3), 285–292.

Rose, A., Ken, D., Kehinde, O., Alo, B., & Drouillard Ken, D. (2012). Bioaccumulation of polycyclic aromatic hydrocarbons in fish and invertebrates of Lagos lagoon, Nigeria. *Journal of Emerging Trends in Engineering and Applied Sciences, 3*, 287–296.

Rust, A. J., Burgess, R. M., McElroy, A. E., Cantwell, M. G., & Brownawell, B. J. (2004). Influence of soot carbon on the bioaccumulation of sediment-bound polycyclic aromatic hydrocarbons by marine benthic invertebrates: An interspecies comparison. *Environmental Toxicology and Chemistry, 23*, 2594–2603.

Santana, M. S., Sandrini-Neto, L., Neto, F. F., Neto, F. F., Oliveira Ribeiro, C. A., Di Domenico, M., & Prodocimo, M. M. (2018). Biomarker responses in fish exposed to polycyclic aromatic hydrocarbons (PAHs): Systematic review and meta-analysis. *Environmental Pollution, 242*, 449–461.

Shi, J., Zheng, G. J.-S., Wong, M.-H., Liang, H., Li, Y., Wu, Y., ... Liu, W. (2016). Health risks of polycyclic aromatic hydrocarbons via fish consumption in Haimen bay (China), downstream of an e-waste recycling site (Guiyu). *Environmental Research, 147*, 233–240.

Shappell, N. W., Carlino-Macdonald, U., Amin, S., Kumar, S., & Sikka, H. C. (2003). Comparative metabolism of chrysene and 5-methylchrysene by rat and rainbow trout liver microsomes. *Toxicological Sciences, 72*, 260–266.

Thorsen, W. A., Cope, W. G., & Shea, D. (2004). Bioavailability of PAHs: Effects of soot carbon and PAH source. *Environmental Science & Technology, 38*, 2029–2037.

Tongo, I., Etor, E. E., & Ezemonye, L. I. N. (2018). Human health risk assessment of PAHs in fish and shellfish from Amariaria community, Bonny River, Nigeria. *Journal of Applied Sciences and Environmental Management, 22*(5), 731–736.

Uno, S., Kokushi, E., Añasco, N. C., Iwai, T., Ito, K., & Koyama, J. (2017). Oil spill off the coast of Guimaras Island, Philippines: Distributions and changes of polycyclic aromatic hydrocarbons in shellfish. *Marine Pollution Bulletin, 124*(2), 962–973.

US EPA (2000). Risk-based concentration table. Philadelphia PA: United States Environmental Protection Agency, Washington DC.

USEPA. (2000). Supplementary Guidance for Conducting Health Risk Assessment ofChemical Mixtures. EPA/630/R-00/002.

Varanasi, U., & Stein, J. E. (1991). Disposition of xenobiotic chemicals and metabolites in marine organisms. *Environmental Health Perspectives, 90*, 93.

Vuorinen, P. J., Keinänen, M., Vuontisjärvi, H., Barsiene, J., Broeg, K., Förlin, L., ... Schiedek, D. (2006). Use of biliary PAH metabolites as a biomarker of pollution in fish from the Baltic Sea. *Marine Pollution Bulletin, 53*, 479–487.

Wenzl, T., Simon, R., Anklam, E., & Kleiner, J. (2006). Analytical methods for polycyclic aromatic hydrocarbons (PAHs) in food and the environment needed for new food legislation in the European Union. *TrAC Trends in Analytical Chemistry, 25*, 716–725.

WHO (1998). *Environmental health criteria 202: Selected non-heterocyclic polycyclic aromatic hydrocarbons.* World Health Organisation, Geneva: International Programme on Chemical Safety.

Xia, Z., Duan, X., Qiu, W., Liu, D., Wang, B., Tao, S., ... Hu, X. (2010). Health risk assessment on dietary exposure to polycyclic aromatic hydrocarbons (PAHs) in Taiyuan, China. *Science of the Total Environment, 408*, 5331–5337.

Yakan, S., Focks, A., Klasmeier, J., & Okay, O. S. (2017). Numerical evaluation of bioaccumulation and depuration kinetics of PAHs in Mytilus galloprovincialis. *Environmental Pollution, 220*, 1244–1250.

Yang, W., Lang, Y., & Li, G. (2014). Cancer risk of polycyclic aromatic hydrocarbons (PAHs) in the soils from Jiaozhou Bay wetland. *Chemosphere, 112*, 289–295.

Zelinkova, Z., & Wenzl, T. (2015). EU marker polycyclic aromatichydrocarbons in food supplements: Analytical approach and occurrence. *Food Additives & Contaminants: Part A, 32*(11), 1914–1926.

Zhu, H., Wang, J., Santo, A., & Li, Y. (2013). Downregulation of antioxidants and phase proteins. In F. A. Villamena (Ed.). *Molecular basis of oxidative stress: chemistry, mechanisms, and disease pathogenesis* (pp. 113–121). New Jersey: John Wiley & Sons.