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Oxidative stress parameters in red sea bream, *acanthopagrus berda* exposed to environmental pollutant phenanthrene

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Phenanthrene (Phe) is a major contaminant of the aquatic ecosystem by releasing of wastewater from the oil industry into common water sources. The current study aims to clarify the expected action of phenanthrene on tissue antioxidant enzymes, hepatic and kidney functions of bream fish. Eighty alive adult male sea bream fish were used and allocated into 4 groups;control, the low-phenanthrene dose (10 ng/ml water), the high-phenanthrene dose (50 ng/ml water) and vehicle control group in 4 glass aquaria. The time of exposure was 15 days while maintaining the same experimental conditions. Results revealed that in the high-dose group and at day 15 of exposure , the hepatic and gill levels of Glutathione (GSH) , Glutathione-s-transferase (GST), and catalase (CAT) were significantly increased .In addition , hepatic Glutathione peroxidase (GP_x) and Malondialdehyde (MDA) were also increased significantly .Phenanthrene at low dose significantly elevates activities of transaminases (ALT & AST) at 10 and 15 days of exposure while, they were significantly elevated at 7 ,10 and 15 days at a high dose group , however no changes in the levels of urea and creatinine were recorded all over the experimental time. Results obtained confirmed that Phe caused oxidative stress and raised concerns about hepatotoxicity in bream fish. The lower vulnerability of the gills to oxidative damage (compared to the liver) appears to be related to the higher basal levels of antioxidants .Conclusively, this suggests a high ecological risk of phenanthrene to aquatic organisms.

Keywords: Oxidative parameters , Sea Bream, liver , Gills ,Phenanthrene, Urea

INTRODUCTION

Polycyclic aromatic hydrocarbons (PAHs) are a widely distributed group of pollutants that have drawn attention for their toxicity(Ranjbaret al. 2020; Liet al. 2020). Many PAHs are released into the aquatic environment from accidental oil spills, drilling leaks, and industrial wastewater and can affect aquatic life (Naudinet al.2019)

In recent decades, many researches showed higher rate of malformations, lesions, tumors and other toxic effects as a consequences for PAHs toxicity (Sunet al.2019). Diseasesyndromesincluding heartfailure, edem a and spinal abnormalities have been observed in zebrafish embryos after exposure to a mixture of PAHs (Incardona et al.2004). PAH-contaminated sediments resulted in liver injury in mummichogs (Fundulusheteroclitus) (Lourenço et al.2021).Incidence of hepatic cancer in brown bull (Ameiurus nebulosus) was also used as a signs of PAH exposure (Baumann et al.1996) .

The sea bream, *Sparus aurata,Lis* a bottom dweller, usually living solitary or in small mobile groups. It is one of the predominant sparid fishes and is widespread throughout the Mediterranean Sea and on the east coast of the Atlantic, representing an important fishery resource on both coasts. This species of fish has been known for many years, mainly as a bycatch (Aydin,2018).

The oxidative enzymes are used to detect the harmfull effects of xenobiotics on organisms (González-Fernández et al.2016 ; Lam,2004) .Crude oil significantly decreased SOD content in sea bass (Danion et al.2009) and elevates the lipid peroxidation products in the livers of Lateolabraxjaponicus (Lin et al.2005) .At gene level, many studies have been performed to clarify the PAH-induced oxidative harms in zebrafish and other aquatic species (Wincent et al.2015 ;Dasgupta et al.2014)).However, the impact of PAHs on these oxidative parameters in bream have not been reported. Metabolites are the most important biomarkers, which can explaine the metabolic changes in response to the Surrounding variables (Goodale et al.2014 ; Jayasundara et al.2015) .Many researchers have

studied the effects of PAHs on zebrafish metabolism and demonstrated toxic effects on protein production,heart and mitochondrial functions(Nicholson et al.2012; Johnson and Luís et al.2020) .

Plasma is a pole of metabolites suitable for studying the biological alterations as a reflection to pollutants[18].In this study, the effects of phenanthrene on seabream were designed to determine changes in hepatic and gill's antioxidant parameters,liver and renal function indices.These results may help understand PAHs' impact on aquatic ecosystems.

MATERIALS AND METHODS

Phenanthrene and experimental fish groups

Phenanthrene crystals, sublimed grade, (purity ≥ 99.5%) ,695114 - Sigma - Aldrich was purchased from Agitech Company /Jeddah. A stock alcoholic phe solution of 1 mg/ml was prepared by dissolving in ethanol and stored in a dark bottle at 4°C.To prepare the desired Phe concentration, the stock solution was diluted with filtered water .The added volume of ethanol was equal for all groups with final concentration less than <0.1%.

A total of 80 freshly alive adult male gilthead seabream fish of local seabream fish that were randomly collected from the central fish market, Al-Kakiyahdistrict, Makkah Al-Mukarramah Governorate (Latitude 21.422487, Longitude 39.826206) .Their lengths were 25 – 35 cm and weight were 200 - 250 grams. The sex and maturity were determined via observation of motile sperms in drop of milt released after multiple stripping.

At the laboratory, fish were maintained into glass stock tank (100 cm x 55 cm x 50 cm) filled with 120 liters of aerated filtered sea water and kept at 27 ± 1 ^{0}C Under the influence of light and dark for 12 hours alternately and fed twice daily with 3% body weight of commercial dry pellets .Water parameters including temperature, pH and salinity were adjusted, at 27±1^{o}C, 7.1±0.5 and, 41±0.5%, respectively (Elieet al.2015). The fish were kept in laboratory conditions for two weeks before use for the experiments (Sreekumaret al.2009). Water in the tank was replaced each 3 days while maintaining the same experimental conditions. Experiments were performed according to the guidelines of the National Institutes of Health (NiH) for the use and care of animals, and the study protocol was performed according to the guidelines of Umm - Al Qura University for the use of laboratory animals. Efforts were made to reduce the number and suffering of fish used. Fish were equally grouped into 4 equal experimental groups, 20 males / group , maintained in 60 L tank (75x35x30) contained aerated filtered sea water with sand substratum and designated as control , low-phenanthrene dose , high-phenanthrene dose and vehicle groups. Water temperature, PH and salinity were adjusted at 27± 1 ^{0}C , 7.1 ± 0.5 and 41±0.5 ,respectively. Fish of all groups were left for one week in their corresponding tank without treatment for acclimatization. No mortality was observed during the experiments.

Treatment

Fish of control group were left without treatment, the low-phenanthrene dose group was treated with phenanthrene alcoholic solution in adose of 10 ng/ml, the high-phenanthrene dose group was treated with phenanthrene alcoholic solution in adose of 50 ng/ml and the fishes of the 4th group were treated with less than 1% ethylealcohole solution and served as vehicle control group. After the period of acclimatization, treatment for each fish group was started. The time of exposure was 15 days and the water in each tank was replaced each 3 days while maintaining the same experimental conditions.

Serum biochemical markers

Five fish were randomly selected from each tank on days 0, 7, 10 and 15 during the exposure period. Using a 3 cc disposable syringe and a 21-gauge needle, blood samples from the tail vein were collected and transferred to the anticoagulant-free Eppendorf for collection of serum for estimation of blood urea, creatinine and transaminases (ALT&AST) spectrophotometerically (Lab-Med Co.,American Inc., USA) according to the manufacturer's guides. After blood sampling was complete, tissue samples (liver and gills) were taken from 4 fish/per group, followed by rinsing with buffered saline to remove excess blood, then weighed and stored at -80° C in liquid nitrogen.

Biomarkers for tissue antioxidants and lipid peroxidation

The frozen liver and gills homogenates were prepared in 10 volumes of 0.1 M Tris - EDTA buffer (pH 7.4) and 30 minutes centrifugation at 1000xg at4 °C. An aliquot of supernatant has been used For further colorimeteric assessments. GSH was determined based on the reductive breakdown of 2,5'-Hithiobis acid groups (2-nitrobenzoic acid) (DTNB) and sulfhydryl (-SH) to produce a yellow color. Reduced chromophore is directly proportional to the concentration of GSH. Absorbance measured at 412 nm. GST activity was measured according to the method of Habiget al.1974). Depending on measuring the conjugation of 1-chloro-2,4-dinitrobenzene (CDNB) and reduced glutathione at 340 nm . GPx activity was determined colorimetrically through indirect measure of GPx activity. An aliquot of tissue homogenate was added to the solution containing GSH, GR, and NADPH. The enzymatic reaction was initiated by tert-butyl hydroperoxide and measured at 340 nm.The activity of CAT was measured according to Aebi(1984). It reacts with H_2O_2 and In the presence of peroxidase, residual H2O2 reacts with 3,5-dichloro-2-

hydroxybenzenesulfonic acid and 4-aminophenazone to form a chromophore whose color density is proportionate inversily to the content of CAT in the sample . Absorbance was measured at 510 nm.Lipid peroxidation was measured as described by Ohkawaet al.(1979). Thiobarbituric acid reacts with MDA in acidic medium / 95°C / 30 min forming TBA-reactive product. The product absorbance was measured at 534 nm.

Statistical Analysis

Data were expressed as overall mean ± SE for all parameters and analyzed using analysis of variance (ANOVA) tests. All statistical analyzes were performed using SPSS software (v.15.0). Results were significant at P≤ 0.05 and highly significant at P ≤ 0.01.

RESULTS

The current results clarify the toxic impact of phenanthrene exposure (10 ng/mL and 50 ng/ml) on liver and kidney functions, hepatic and renal tissue's contents of antioxidative enzymes and (MDA) formation in adult male sea bream fish.

Hepatic and Renal Parameters

Data revealed that the 10ng/ml phenanthrene administration significantly increased serum AST and ALT levels on days 10 and 15 of the exposure period ($p<0.05$) comparing to control values .They were increased significantly on days 7, 10 and 15 of Phenanthrene exposure at a dose of 50 ng/ml but had no effect on serum urea and creatinine levels throughout the trial period (Table 1).

Tissues antioxidants and lipid peroxidation parametersHepatic Antioxidants and lipid peroxidation levels

Table 2 illustrates the hepatic content of antioxidants and lipid peroxidation level in adult male seabream fish exposed for different doses of phenanthrene.Results revealed no significant alterations in hepatic GSH contents after 7 days of exposure among all experimental groups ($p>0.05$) ; meanwhile, they were significantly ($p<0.05$) increased after phenanthrene exposure at 10 and 15 days for both phenanthrenedoses.No significant changes was recorded in the activity of hepatic GST enzyme on exposure to low phenanthrene dose while,it was significantly ($p<0.05$) increased on exposure to higher phenanthrene dose (50 ng/ml) at 15 days of exposure.

Gills Antioxidants and lipid peroxidation levels

Table 3 shows the gill's content of antioxidants and lipid peroxidation level in adult male seabream fish exposed for different doses of phenanthrene. Data revealed that no significant changes in GSH, GST and CAT levels were recorded in fish subjected to low concentration of phenanthrene (10ng/ml) while, they were significantly increased in fish subjected to higher concentration of phenanthrene (50ng/ml) at day 15 of exposure as compared to controls ($p>0.05$). However, no significant ($p>0.05$) changes in GPx activity and MDA level were recorded in all experimental fish groups with different doses of phenanthrene and at different times of exposure.

Table 1: Liver and kidney parameters (AST, ALT, urea and creatinine) in adult male seabream fish exposed to phenanthrene for 15 days.

Parameters	Group	Exposure Time /Day			
		0	7	10	15
AST (U/L)	Control	6.60 ± 1.05	7.00 ± 1.05^{a}	7.90± 1.11^{a}	8.00± 0.90^{a}
	Phenanthrene (10 ng/ml)	7.50± 1.00	7.50± 1.00^{a}	11.20± 1.13^{b}	15.30± 1.09^{b}
	Phenanthrene (50 ng/ml)	6.88± 1.10	13.40 ± 1.05^{b}	16.30± 1.15^{c}	19.90± 1.08^{c}
	Vehicle (Alcohole) Control	7.10± 1.30	6.00 ± 1.10^{a}	7.80± 1.18^{a}	6.99± 0.95^{a}
ALT (U/L)	Control	13.00 ± 2.20	14.00 ± 3.00^{a}	13.90± 3.20^{a}	14.70± 4.02^{a}
	Phenanthrene (10 ng/ml)	13.80 ± 2.80	13.80 ± 2.80^{a}	17.90± 2.80^{b}	20.20± 4.01^{b}
	Phenanthrene (50 ng/ml)	12.40 ± 3.10	19.50± 3.20^{b}	22.50± 4.02^{c}	25.20± 2.20^{c}
	Vehicle (Alcohole) Control	14.00 ± 2.70	13.00 ± 2.90^{a}	14.10± 3.09^{a}	14.66± 2.80^{a}
UREA (mg/dl)	Control	6.90 ±1.02	6.90 ±1.02	7.90 ±1.02	8.10 ±1.02
	Phenanthrene (10 ng/ml)	6.88±1.05	6.88±1.05	7.88±1.05	7.88±1.05
	Phenanthrene (50 ng/ml)	7.70±1.12	7.70±1.12	8.70±1.12	8.20±1.12
	Vehicle (Alcohole) Control	7.50±1.10	7.50±1.10	8.50±1.10	8.70±1.10
Creatinine (mg/dl)	Control	0.32 ± 0.11	0.34 ± 0.11	0.36 ± 0.13	0.35 ± 0.12
	Phenanthrene (10 ng/ml)	0.31±0.12	0.36±0.12	0.39±0.11	0.37±0.11
	Phenanthrene (50 ng/ml)	0.32±0.14	0.38±0.14	0.39±0.11	040±0.13
	Vehicle (Alcohole) Control	0.35± 0.12	0.40± 0.12	0.33± 0.11	042± 0.11

Data are Mean ± SE, values within the same column of the same parameter carrying different letters are significantly

different from each other (p<0.05). (ALT): Alanine transaminase enzyme ,(AST) : Aspartate transaminase enzyme

Table 2: Liver oxidative enzymes and Malondialdehyde level in adult male Seabream fish groups exposed to phenanthrene for 15 days.

Organ	Group	Exposure Time /Day			
		0	7	10	15
Reduced Glutathione level (GSH) (mmol/g wet tissue)					
Liver	Control	0.78 ± 0.09	0.85 ± 0.07	0.76 ± 0.08^{a}	0.74 ± 0.06^{a}
	Phenanthrene (10 ng/ml)	0.73 ± 0.06	0.82 ± 0.11	0.90 ± 0.09^{b}	0.98 ± 0.07^{b}
	Phenanthrene (50 ng/ml)	0.79 ± 0.11	0.86 ± 0.09	0.96 ± 0.09^{b}	1.12 ± 0.09^{b}
	Vehicle (Alcohole) Control	0.76 ± 0.14	0.87 ± 0.11	0.75 ± 0.05^{a}	0.69 ± 0.08^{a}
Glutathione-s-tranferase activity (GST) (U/g wet tissue)					
Liver	Control	0.29 ±0.08	0.25 ±0.06	0.28 ±0.09	0.26 ±0.08^{a}
	Phenanthrene (10 ng/ml)	0.27 ±0.03	0.27 ±0.06	0.26 ±0.09	0.25 ±0.08^{a}
	Phenanthrene (50 ng/ml)	0.31 ±0.05	0.29 ±0.06	0.29±0.09	0.40 ±0.09^{b}
	Vehicle (Alcohole) Control	0.25 ±0.07	0.26 ±0.09	0.27 ±0.08	0.22 ±0.09^{a}
Glutathione Peroxidase activity (GP_x) (U/g wet tissue)					
Liver	Control	60.30±8.30	59.27±7.20	61.40 ±5.29	58.30 ±4.29^{a}
	Phenanthrene (10 ng/ml)	58.30±9.10	62.30±8.30	60.30±7.50	60.23 ±5.44^{a}
	Phenanthrene (50 ng/ml)	62.30±6.90	60.30±8.80	59.30±9.00	40.90 ±5.11^{b}
	Vehicle (Alcohole) Control	58.30±8.49	57.27±9.82	56.38±6.29	61.38 ±6.20^{a}
Catalase activity (CAT) (U/g wet tissue)					
Liver	Control	4.30 ± 0.20	5.30 ± 0.30	4.40 ± 0.13	5.20 ± 0.10^{a}
	Phenanthrene (10 ng/ml)	3.90 ± 0.30	4.30 ± 0.28	5.30 ± 0.60	6.10 ± 0.13^{a}
	Phenanthrene (50 ng/ml)	5.10 ± 0.55	5.10 ± 0.18	5.12 ± 0.20	11.25 ± 0.18^{b}
	Vehicle (Alcohole) Control	4.40 ± 0.22	5.25 ± 0.35	4.90 ± 0.13	5.10 ± 0.13^{a}
Malondialdehyde (MDA) levels					
Liver	Control	19.82 ± 3.00	21.66 ± 4.04	20.03 ±1.09	19.03 ±1.30^{a}
	Phenanthrene (10 ng/ml)	20.82 ± 3.05	19.82 ± 3.05	21.82 ±4.10	17.10 ±1.40^{a}
	Phenanthrene (50 ng/ml)	17.82 ± 4.05	20.82 ± 2.90	20.82 ± 3.05	27.06 ±1.45^{b}
	Vehicle (Alcohole) Control	18.82 ± 4.20	19.66 ± 4.80	18.03 ± 3.98	17.03 ± 1.11^{a}

Data are Mean ± SE, values within the same column of the same parameter carrying different letters are significantly different from each other (p< 0.05).

Table 3:Gills oxidative enzymes and Malondialdehyde level in adult male Seabream fish groups exposed to phenanthrene for 15 days.

Organ	Group	Exposure Time / Day			
		0	7	10	15
Reduced Glutathione level (GSH) (mmol/g wet tissue)					
Gills	Control	0.30 ± 0.02	0.25 ± 0.07	0.26 ± 0.03	0.30 ± 0.02
	Phenanthrene (10 ng/ml)	0.33 ± 0.02	0.22 ± 0.05	0.28 ± 0.01	0.33 ± 0.02
	Phenanthrene (50 ng/ml)	0.38 ± 0.00	0.26 ± 0.09	0.31 ± 0.02	0.38 ± 0.00
	Vehicle (Alcohole) Control	0.35 ± 0.05	0.30 ± 0.06	0.28 ± 0.03	0.35 ± 0.05
Glutathione-s-tranferase activity (GST) (U/g wet tissue)					
Gills	Control	0.30 ±0.05	0.27 ±0.05	0.31± 0.05	0.28 ±0.06^{a}
	Phenanthrene (10 ng/ml)	0.32 ±0.07	0.29 ±0.08	0.30 ±0.05	0.33 ±0.05^{a}
	Phenanthrene (50 ng/ml)	0.33 ±0.09	0.30 ±0.05	0.30 ±0.05	0.46 ±0.06^{b}
	Vehicle (Alcohole) Control	0.29 ±0.06	0.31 ±0.07	0.30± 0.02	0.32 ±0.03^{a}
Glutathione Peroxidase activity (GPx) (U/g wet tissue)					
Gills	Control	43.63±6.75	44.97±7.07	42.30±6.02	46.97 ±7.08
	Phenanthrene (10 ng/ml)	41.63±8.65	43.63±5.95	42.63±8.75	42.35 ±8.01

	Phenanthrene (50 ng/ml)	44.63±6.45	43.63±8.55	45.63±8.65	45.67 ±8.011
	Vehicle (Alcohole) Control	41.03±5.60	43.37±5.23	44.50±5.11	42.57±4.12
Catalase activity (CAT) (U/g wet tissue)					
Gills	Control	5.10 ±0.16	5.06 ±0.41	4.83 ±0.55	5.03 ± 0.39[a]
	Phenanthrene (10 ng/ml)	6.10 ±0.15	6.10 ±0.15	6.10 ±0.33	7.14 ± 0.42[a]
	Phenanthrene (50 ng/ml)	7.10 ±0.13	7.18 ±0.16	6.30 ±0.16	11.12 ± 0.55[b]
	Vehicle (Alcohole) Control	5.90 ±0.10	5.30 ±0.21	5.23 ±0.58	5.33 ± 0.40[a]
Malondialdehyde (MDA) levels					
Gills	Control	121.01±14.22	123.45±15.40	127.03±17.82	133.45 ±18.20
	Phenanthrene (10 ng/ml)	125.41±17.22	126.01±15.02	125.01±13.12	137.15 ±16.44
	Phenanthrene (50 ng/ml)	119.24±14.00	129.01±17.00	129.01±16.29	139.30 ±17.50
	Vehicle (Alcohole) Control	125.11±15.29	122.65±16.70	128.13±16.80	130.45 ±17.25

Data are Mean ± SE, values within the same column of the same parameter carrying different letters are significantly different from each other ($p < 0.05$).

DISCUSSION

PAHs are considered a persistent organic pollutants (POPs) causing harms to the environment and humans, causing stress and affecting the health of marine life(Al-Zahabyet al.2018).

Blood indices including transaminases (ALT & AST) , urea, and creatinine are considered as parameters for detecting hepatic and renal damage and their measurement has been suggested to be useful to clarify organs functions(Mar Huertaset al.2014 ; Hodson,2017) .

In the present study, exposure of adult seabream to phenanthrene (Phe) at high dose (50 ng/ml) significantly increased serum AST and ALT activity at 7, 10, and 15 days of exposure and at days 10 and 15 of exposure to low Phe dose (10 ng/ml). These results indicate liver damage. Previously, exposure of yellow sea bream to Phe significantly increased activities of transaminases , ALT and AST , 7 days after the exposure ($P<0.05$)(Ololadeet al.2021)) .Elevated blood transaminase activities after Phe exposure may be due to cell destruction possibly in the liver, heart, or muscle(McDonald and Grosell,2006) There was no significant change in renal function after Phe exposure. The difference in the effect of Phe for both liver and kidney can be attributed to that the liver is the major organ for the metabolic and degradation pathways of the compound. It appears that the lowered vulnerability of the kidney to oxidative damage (compared to the liver), might be due to its higher basal level of antioxidants (Oliveiraet al.2008)

Several studies have shown that exposure to pollutants in aquatic ecosystems, including the compound phenanthrene, can increase intracellular ROS generation, causing oxidative damage to biological systems (Shirmohammadi et al.2017; Bordier et al.2020) . Antioxidant enzymes are protective factors that act as early indices for cellular damage caused by free radicals(Yadetie et al.2021).In the present study, antioxidants and lipid peroxidation levels were significantly activated in hepatic tissue (GSH, GST, GPx, CAT, and MDA) and gills (GSH, GST, and CAT) after exposure to 50 ng/mL phenanthrene at 15 days of exposure. As a result, significant oxidative stress was induced, indicating a major toxic effect of phenanthrene on the physiological metabolism of sea bream.

Antioxidant enzymes and MDA are indicators often used to evaluate oxidative stress and lipid peroxidation affecting xenobiotic (Fìratet al.2009).In this study, hepatic level of MDA was significantly increased after exposure to Phe, indicating cell membrane peroxidation. Similarly,Lin et al.(2011) found that pyrene exposure increased MDA content in Lateolaprax japonicus. In consistent, exposure to phe significantly elevated the level of MDA in the muscles of the estuarine guppy Buciliavivipara(Vijayavelet al.2006) .

Furthermore, benzo (a) pyrene significantly increased the MDA level of Ruditapes philippinarum (Todorova et al.2005). However, there was no significant change in MDA content in the gill tissues after 15 days of Phe exposure. The difference in hepatic and gill responses may indicate an increase in the level of lipid peroxidation, suggesting more ROS was generated in hepatic tissue.

It is known that anti-oxidation enzymes are activated to counteract damage of oxidative stress (Machadoet al.2014) . The CAT, the more active antioxidant enzyme can reduce H_2O_2(Giuliani and Regoli,2014).In this study, TPS, GPX and CAT activities were significantly elevated after 15 days of Phe exposure which could be a reaction to oxidative stress. These results corresponded to previous studies in which the same SOD, GST and CAT activities were significantly increased in Carassius Autus in response to Phe exposure(Wang et al.2018) The study also came to the conclusion that GSH induction can be attributed to the primary immune system, in which GSH is included in the protection of fish against free radicals. Most of the radicals produced can be neutralized by GSH, that acts as the first line of cellular defense against oxidative stress by

scavenging oxygen radicals and sharing in detoxification pathways through glutathione peroxidase (Sunet al.2006).Glutathione-S-transferases are a group of biotransferases in the cytoplasm of manycells and participate in neutralizing of reactive compounds through the combination of glutathione to various compounds and perform other indirect antioxidant functions(Espinosa-Diez et al.2015)The present resultsare consistent withprevious studies(Dasar i et al.2017) that showed different responses of GPx to Phe in hepatic and gill's tissues. They showed tissue-specific responses to exposure to GPxPhe and O. niloticus exposed to diazinon(Julia et al.2022).

CONCLUSIONS

In conclusion, this study highlights the Phe potential to induce oxidative stress which should affect the welfare of the seabream fish. Also, the obtained results showed an organ-specific antioxidant defense mechanism dependent on Phe concentration. The liver showed a high adaptive capacity manifested by the activation of antioxidant defenses, especially GSH and GPX. The lowered vulnerability of gills for oxidative damage as compared to liver seems to be related to the basic level of the antioxidant

Supplementary materials

The supplementary material / supporting for this article can be found online and downloaded at: https://www.isisn.org/article/10.3390/antiox12081524/s1,

Author contributions

The author contributed to the study conception, design, material preparation, data collection and analysis. The author also wrote the first draft of the manuscript and approved the final manuscript.

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Institutional Review Board Statement

The study was approved by the Bioethical Committee of the Al-Jammoum University college, Umm-Alqura University, Makah, Saudi Arabia.

Informed Consent Statement

Not applicable.

Data Availability Statement

All of the data is included in the article/Supplementary Material.

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Conflict of interest

The author declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

REFERENCES

Aebi H. Catalase in vitro. Methods Enzymol 1984;105:121-6.

Aydin M .Maximum length and age report of Sparusaurata (Linnaeus,1758) in the Black Sea. J Appl Ichthyol 2018 ; 34: 964–966.

Al-Zahaby AS, El-drawany MA, Mahmoud HH, et al. Some biological aspects and population dynamics of the gilthead sea bream from Bardawil lagoon, Sinai, Egypt. Egyptian Journal of Aquatic Biology & Fisheries 2018;22(5): 295- 308.

Baumann PC, Smith IR, Metcalfe CD.Linkages between chemical contaminants and tumors in Benthic Great Lakes fish.J Great Lakes Res 1996; 22: 131–152.

Bordier M., Uea-Anuwong T., Binot A., Hendrikx P., Goutard F.L. Characteristics of one health surveillance systems: A systematic literature review. Prev. Vet. Med. 2020;181:104560. doi: 10.1016/j.prevetmed.2018.10.005.

Danion M, Floch SL, Lamour FO, et al. EROD activity and antioxidant defenses of sea bass (Dicentrarchuslabrax) after an in vivo chronic hydrocarbon pollution followed by a post-exposure

period. Environ SciPollut R 2014; 21: 13 769–13 778 .

Dasari S, Ganjayi M, Oruganti L, etal.Glutathione s-transferases detoxify endogenous and exogenous toxic agents-mini review. J Dairy Vet Anim Res 2017;5(5):157-159

Dasgupta S, Cao A, Mauer B, et al. Genotoxicity of oxy-PAHs to Japanese medaka (Oryziaslatipes) embryos assessed using the comet assay. Environ SciPollut Res 2014; 21: 13 867–13 876 .

Elie MR, Choi J, Nkrumah-Elie YM, et al. 2015 Metabolomic analysis to define and compare the effects of PAHs and oxygenated PAHs in developing zebrafish. Environ Res 2015; 140: 502–510 .

Espinosa-Diez C, Miguel V, Mennerich D, et al. Antioxidant responses and cellular adjustments to oxidative stress. Redox Biol 2015; 6: 183–197.

Fìrat O, Cogun HY, Aslanyavrusub S, et al. Antioxidant responses and metal accumulation in tissues of Nile tilapia Oreochromisniloticus under Zn, Cd and Zn + Cd exposures. J ApplToxicol 2009;29: 295-301.

Goodale BC, Tilton SC, Corvi MM, etal.Structurally distinct polycyclic aromatic hydrocarbons induce differential transcriptional responses in developing zebrafish. ToxicolAppl Pharm 2013; 272: 656–670 .

Giuliani ME, RegoliF.Identification of the Nrf2–Keap1 pathway in the European eel Anguilla anguilla: role for a transcriptional regulation of antioxidant genes in aquatic organisms. AquatToxicol 2014; 150: 117–123 .

Habig W H, Pabst M J, Jakoby W B. Glutathione S-transferases. The first enzymatic step in mercapturic acid formation. J Biol Chem 1974; 249(22):7130-9.

Hodson P.V. The Toxicity to Fish Embryos of PAH in Crude and Refined Oils. Arch. Environ. Contam.Toxicol. 2017;73:12–18. doi: 10.1007/s00244-016-0357-6 .

Incardona JP, Collier TK, ScholzNL.Defects in cardiac function precede morphological abnormalities in fish embryos exposed to polycyclic aromatic hydrocarbons. ToxicolAppl Pharm 2004; 196: 191–205 .

Jayasundara N, Van TGL, Meyer JN, et al. 2015 AHR2-mediated transcriptomic responses underlying the synergistic cardiac developmental toxicity of PAHs. ToxicolSci 2015; 143 : 469–481 .

Johnson CH, Gonzalez FJ. 2012 Challenges and opportunities of metabolomics. J Cell Physiol 2012; 227:2975–2981 .

Lam PKS.2009 Use of biomarkers in environmental monitoring. Ocean Coast Manage 2009; 52: 348–354 .

Li R., Hua P., Zhang J., Krebs P. Effect of anthropogenic activities on the occurrence of polycyclic aromatic hydrocarbons in aquatic suspended particulate matter: Evidence from Rhine and Elbe Rivers. Water Res. 2020;179:115901. doi: 10.1016/j.watres.2020.115901 .

Lin JQ, Hong HS, Wang XH, et al. Response of PAHs exposure in seawater to the lipid peroxidation in Lateolabraxjaponicus. J Oceanogr Taiwan Strait 2005; 24: 310–315 .

Lin Y, Tang ZS, Cao XW, Acute Toxicity of Cadmium on the Antioxidant Defense Systems and Lipid Peroxidation in the Juveniles of Genetically Improved Farmed (GIFT) Tilapia OreochromisNiloticus. J Environ Sci Health A 2011; 5 :1043-1052

Luís de Sá Salomão A., Hauser-Davis R.A., Marques M. Critical Knowledge Gaps and Relevant Variables Requiring Consideration When Performing Aquatic Ecotoxicity Assays.Ecotoxicol. Environ. Saf. 2020;203:110941. doi: 10.1016/j.ecoenv.2020.110941 .

Lourenço R.A., Taniguchi S., da Silva J., Gallotta F.D.C., Bícego M.C. Polycyclic aromatic hydrocarbons in marine mammals: A review and synthesis. Mar. Pollut. Bull. 2021;171:112699. doi: 10.1016/j.marpolbul.2021.112699.

Machado AAdeS, Hoff MLM, Klein RD, etal.Oxidative stress and DNA damage responses to phenanthrene exposure in the estuarine guppy Poecilia vivipara. Mar Environ Res.2014; 98: 96–105..

Mar Huertas O, Almeida G, Adelino VM, et al.Tilapia male urinary pheromone stimulates female reproductive axis.General and Comparative Endocrinology 2014;196 : 106–111.

McDonald MD, Grosell M. Maintaining osmotic balance with an aglomerular kidney. Comp BiochemPhysiol C 2006; 143:447– 458.

Naudin G., Bastien P., Mezzache S., Trehu E., Bourokba N., Appenzeller B.M.R., Soeur J., Bornschlögl T. Human pollution exposure correlates with accelerated ultrastructural degradation of hair fibers. Proc. Natl. Acad. Sci.USA. 2019;116:18410–18415. doi: 10.1073/pnas.1904082116 .

Julia, V; Paloma, R; Ivelise, D; Francielli, C; Rafaela, G et al.2022.The Role of the Ecotoxicology Applied to Seafood as a Tool for Human Health Risk Assessments Concerning Polycyclic Aromatic Hydrocarbons. Int J Environ Res Public Health , 19(3): 1211-1218.

Ohkawa H, Ohishi N, Yagi K. Assay for lipid peroxidesbin animal tissues by Thiobarbituric acid reaction. Anal Biochem 1979;95(2):351-8.

Oliveira M, Pacheco M, Santos M. Organ specific

antioxidant responses in golden grey mullet (Liza aurata) following a short-term exposure to phenanthrene. Science of The Total Environment 2008; 396(1):70-78

Ololade I.A., Arogunrerin I.A., Oladoja N.A., Ololade O.O., Alabi A.B. Concentrations and Toxic Equivalency of Polycyclic Aromatic Hydrocarbons (PAHs) and Polychlorinated Biphenyl (PCB) Congeners in Groundwater around Waste Dumpsites in South-West Nigeria. Arch. Environ. Contam.Toxicol. 2021;80:134–143. doi: 10.1007/s00244-020-00790-3.

RanjbarJafarabadi A., Mashjoor S., RiyahiBakhtiari A., Jadot C. Dietary intake of polycyclic aromatic hydrocarbons (PAHs) from coral reef fish in the Persian Gulf-Human health risk assessment. Food Chem. 2020;329:127035. doi: 10.1016/j.foodchem.2020.127035.

Shirmohammadi M, Salamat N, TaghiRonagh M, et al.Effect of Phenanthrene on the Tissue Structure of Liver and Aminotransferase Enzymes in YellowfinSeabream (Acanthopagruslatus). Iranian Jornal of Toxicology 2017; 11(4):33-41

Sun S.J., Zhao Z.B., Li B., Ma L.X., Fu D.L., Sun X.Z., Thapa S., Shen J.M., Qi H., Wu Y.N. Occurrence, composition profiles and risk assessment of polycyclic aromatic hydrocarbons in municipal sewage sludge in China. Environ. Pollut. 2019;245:764–770. doi: 10.1016/j.envpol.2018.11.067.

Sreekumar A, Poisson LM, Rajendiran TM, etal.Metabolomic profiles delineate potential role for sarcosine in prostate cancer progression. Nature 2009; 457: 910–914 .

Sun Y, Yu H, Zhang J, et al. Bioaccumulation, depuration and oxidative stress in fish Carassius auratus under phenanthrene exposure. Chemosphere 2006; 63: 1327.

Todorova I, Simeonova G, Kyuchukova D, et al. Reference values of oxidative stress parameters (MDA, SOD, CAT) in dogs and cats. Comp ClinPathol 2005; 13: 190–194 .

Vijayavel K, Gomathi D, Durgabhavani K, et al.Sublethal effect of naphthalene on lipid peroxidation and antioxidant status in the edible marine crab Scylla serrata. Mar Pollut Bull 2006; 48:429– 433.

Wang H, Luqing P, Lingjun S, et al. The role of Nrf2-Keap1 signaling pathway in the antioxidant defense response induced by PAHs in the calm Ruditapesphilippinarum. Fish Shellfish Immun 2018; 80:325–334 .

Wincent E, Jönsson ME, Bottai M, etal.Aryl hydrocarbon receptor activation and developmental toxicity in zebrafish in response to soil extracts containing unsubstituted and oxygenated PAHs. Environ Sci Technol 2015; 49: 3869.

Yadetie F., Brun N.R., Vieweg I., Nahrgang J., Karlsen O.A., Goksøyr A. Transcriptome Responses in Polar Cod (Boreogadussaida) Liver Slice Culture Exposed to Benzo[a]Pyrene and Ethynylestradiol: Insights into Anti-Estrogenic Effects. Toxicol.Vitr. 2021;75:105193. doi: 10.1016/j.tiv.2021.105193 .