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RESEARCH ARTICLE

Effects of contaminated harbour sediment on the growth and histopathology of Nile tilapia (*Oreochromis niloticus*): A long-term study

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Abstract: Growth and histopathology of juvenile *Oreochromis niloticus* (L.) was studied upon continuous exposure to contaminated sediments from the maritime fisheries harbour of Galle, Sri Lanka. Sixteen week long experiment comprised of four duplicated groups of fish in freshwater (NC: negative control without sediment, C: control with unit-dose of pristine sediment, T1 with unit-dose of contaminated sediment, T2 with three unit-doses of contaminated sediment) with total renewal of water and sediment on each fourth day serially. Unit-dose of sub-lethal wet sediment was arbitrarily decided as 38ml through a series of tolerance tests. Total length and weight of fish were recorded (n=20x4 per group) initially and on completion of weeks 4, 10 and 16. Gill and liver histology was qualitatively examined using four sacrificed fish per group at termination. Results showed that harbour sediment in water significantly ($p<0.05$) decreased cumulative growth rates of T1 and T2 fish in terms of absolute growth rate (0.58-0.59 mm/day and 0.17g/day), relative growth rate (1.52-1.56% length gain day^{-1} and 0.73% weight gain day^{-1}) and specific growth rate (0.89-0.91% day^{-1} in length, 2.60-2.61% day^{-1} in weight) as compared to C and NC groups. T1 and T2 juveniles developed toxicopathic signs in gill (lamellae-fusion, decreased inter-lamellar space) and in liver (extensive necrosis, hydropic vacuolation). Growth parameters remained similar between C and NC groups throughout the study with no histological alterations suggesting that sediment borne salinity had no differential effect across groups. This study shows that exposure to contaminated sediment from Galle fisheries harbour causes growth reduction and pathologic lesions in gills and liver of *O. niloticus* juveniles.

Keywords: Gill histopathology, growth retardation, harbour sediment, liver lesions, *Oreochromis niloticus*

Introduction

World harbours remain prone to high level of chemical contamination. This is because of human activities and the semi-enclosed nature that limits natural exchanges. The former includes bilge-water dumping and fuel leaks from boats, boat-hull repair, on site commercial activities and surface runoff. From the water column, chemical agents may adsorb onto particulates and continue to settle as surface sediment (Tessier & Campbell, 1987). In addition to naturally occurring sand, silt, clay and gravel in varying amounts, harbour sludge are shown to have high concentrations of metals (Bothner et al., 1998; Hartl et al., 2007; Huerta-Diaz et al., 2008) including Cd, Cr, Cu, Mn, Ni, Pb, V, Zn and Al (Kerambrun et al., 2012b) and organic chemical pollutants such as polycyclic aromatic hydrocarbons (PAH) (Hellou et al., 2002; Miller et al., 2003; Kerambrun et al., 2012b), polychlorinated biphenyls (PCB) (Manyin & Rowe 2006; Sprovieri et al., 2007; Mohammed et al., 2011), polychlorinated dibenzo *p*-dioxins (Pruell et al., 1990; Mohammed et al., 2009), organochlorine pesticides (Mohammed et al., 2011) and antifouling organotin compounds (Hartl et al., 2007).

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These compounds may be persistent in the environment and are known to cause a wide range of toxic effects on fish (reviewed by Gunawickrama, 2011). Whole-sediment-exposure experiments provide an option to investigate the collective impact of such contaminant-laden compartments and causal relations of aquatic contaminants. Furthermore, as field monitoring relies on captured feral fish whose heterogeneity may obscure results, sediment-exposure experiments under controlled laboratory conditions offer a distinctive advantage over *in situ* exposure (e.g. caging) and field monitoring approaches by excluding confounding factors in the field so that biological responses can be reliably attributed to sediment constituents. Long-term harbour-sediment exposure studies appear to be limited in literature and it curtails the understanding, predictability and promotion of health in such aquatic systems. Present study in this context aims at growth and histopathology of a euryhaline teleost, *Oreochromis niloticus* (L.) juveniles upon continuous (renewed) exposure to maritime harbour sediment over 16 weeks. Additionally, it explores the feasibility of employing *O. niloticus* for exposure tests of sediments carrying trace salinities. In effect, the study biomonitors the contamination of the harbour environment. It remains important as pollution trends pertaining to Galle harbour are obscure.

Materials and methods

***Sediment and fish collection*:** Contaminated bottom sediment (approx. topmost 30cm) from about 3-4 meters off pier at the Fisheries Harbour, Galle, Sri Lanka (N 06º 02.127, E 80º 13.793) was collected (February, 2010). Sediment to be used for the control group was obtained from a pristine area (N 06º 01.456, E 80º 13.184) of the outer harbour region beyond the mouth of the same bay. Following a preliminary trial of tolerance, 38ml of wet-native sediment (after decanting water) was considered as the unit-dose. Unit packs were prepared, and frozen at -20°C to cease microbial activity until use (Thomson et al., 1980). Advance fingerlings of *Oreochromis niloticus* (total length range 37-40mm and descendants of a single stock) were obtained from Udawalawa Fish Breeding Station, Sri Lanka and transported to Department of Zoology, University of Ruhuna. Fish were then maintained for 2 weeks in an outdoor pond by feeding twice a day up to satiation with a formulated high-protein (39%) commercial diet (PRIMA®, Sri Lanka).

***Experimental setup*:** Fingerlings were subsequently transferred into the laboratory experimental setup which consisted of eight identical fiberglass tanks with 165 liter of aged municipal water and moderate aeration. Four groups were set up in duplicate; namely two treatment groups (T1 and T2 with contaminated harbour sediment), a negative control (no sediment), and a control (pristine sediment). Control and T1 tanks were added with one unit-dose each of the designated sediment whereas T2 tank was added with three unit-doses. Sediment was gently dispersed with a glass rod and allowed to settle in the bottom overnight with no aeration before 50 juveniles/ tank were introduced (size range 30-40 mm). Continuous exposure was achieved by transferring the fish into an identical system as described above on completion of every fourth day. Water quality in terms of temperature, conductivity, dissolved Oxygen, alkalinity, pH, nitrate concentration and phosphate concentration were monitored on day 3 after each renewal. Fish were fed twice a day with an adjustment of the amount (g feed/ tank) done bi-weekly to match 2% of mean body weight per fish using concomitant fish weight in a random sub-sample. Experiment was carried out over 16 weeks and under continuous exposure.

***Sampling and measurements*:** A random sub-sample of 20 fish per tank was taken from each tank at the start of the exposure experiment and on completion of weeks 4, 10 and 16 for measurements. Total length (mm) and wet weight (g) were obtained individually by using dial caliper and top-loading balance respectively. A duplicate sub-sample (n=20) was taken from the same tank for measurement after replacement. This was done on all tanks separately so that four replicates of n=20 fish per group was measured for growth rate determination.

Growth rates and indices: Absolute growth rate (GR) in terms of length (L) and weight (W) (Hopkins 1992) was determined by GR (mm day^{-1}) = $L_1 - L_0 / t$ where L_1-L_0 is the length gain during time t (day) and by GR (g day^{-1}) = $(W_1 - W_0)/t$ where W_1-W_0 is the mean weight gain during time t (day). Relative growth rate (RGR) as percentage length gain (%LG) and weight gain (%WG) per day (Hopkins 1992) was calculated by RGR (% L gain day^{-1}) = 100*(total gain/ initial L)/ t and by RGR (% W gain day^{-1}) = 100*(total gain/ initial W)/t. Specific growth rate (SGR) (Hopkins 1992) was calculated by SGR (% day^{-1}) = 100*(*ln* L_1- *ln* L_0/t) and by SGR (% day^{-1}) = 100*(*ln* W_1- *ln* W_0/t).

Statistical Analyses**:** Statistical significance ($p<0.05$) of growth indices and water quality were determined between groups of the same sampling time using parametric one-way ANOVA and Tukey HSD test. All statistical analyses were done using STATISTICA v.7 (StatSoft, USA) software package.

Gill and liver histology: Slices (1-3 mm) of liver and filaments of gill were immediately obtained from sacrificed fish (4 per group) on week 16 sampling for a qualitative study of toxicopathic lesions attributable to contaminated harbour sediment exposure. Slices were fixed in Bouin's solution, and embedded in wax to make 3-4 µm thick sections, that were subsequently stained with hematoxylin and eosine. Images were acquired at 10x40 by light microscopy using Motic Images Plus 2.0 software (Motic, PR of China) and digitally enlarged as necessary.

Results and discussion

Sediment from contaminated site showed surficial liquid-oil aggregations, emanated hydrocarbon odor, and generated surface oil slicks in collection containers and on experimental tank water. This observation verified that the harbour sediment of the present study was polluted with petroleum hydrocarbons understandably from fuel and bilge water discharges from vessels, and surface drainage.

Mean length of fish (37.6-39.2 mm) was homogeneous among groups initially (ANOVA, F= 2.26, P=0.084), and increased with time thereafter (Table 1). Initial mean weight (1.09-1.22 g) also did not differ significantly (ANOVA, F= 1.96, p=0.123). Fish agility prevailed with zero deaths in the groups during 16-weeks of the exposure experiment. Within the 16-week period, mean length of negative control, control, T1 and T2 increased by 184.9%, 198.2%, 173.4% and 169.2% respectively. Concomitant increases of mean weights of the groups in the same order were 20.3, 23.5, 18 and 18 times the initial value. Length and weight trajectories (not shown) of T1 and T2 groups were lying quantitatively below compared to negative control and control groups. One-way ANOVA revealed that exposure to contaminated harbour sediment had a differential effect on mean length and weight in both T1 and T2 on weeks 4, 10 and 16 suggesting a cumulative growth retardation in them (Table 1).

All three growth indices appeared to have affected ($p<0.05$) by sediment exposure with a notable growth rate (GR) reduction in T1 or T2 groups (Table 2). GR both by weight and length was significantly lower compared to negative control and control at weeks 10 and 16, and this was concomitant to reduced length and weight in the same groups. Growth parameters did not differ between the negative control and the control group throughout the study (Table 2).

Gill sections in the negative control and the control groups contained regularly arranged gill lamellae with apparently monotonous blood supplies onto them. Normal Gill tissues possessed clear filaments and their dorsal and ventral lamellae with a continuous epithelium. Toxicopathic lesions were not observed in gill lamellae, filaments or in inter lamellae area of negative control or control groups (Figure 1). Both in T1 and T2, gills as compared to negative control and control groups were markedly more reddish with greater amounts of surface mucus. Gill histopathology of T1 and T2 fish showed extensive lamellae-fusion and decreased inter-lamellar space.

Table 1. Length and weight (mean ± SD, n=80) of juvenile *Oreochromis niloticus* during continuous exposure to contaminated harbour sediment over 16 weeks (NC= negative control without sediment; C= Control with pristine sediment; T1= contaminated harbour sediment; T2= three times of T1 harbour sediment) (significant difference between groups at $p<0.05$ is indicated by the absence of a shared superscript letter within a row separately for length and weight; ANOVA and Tukey HSD test)

	Mean length (mm)				Mean weight (g)			
	NC	C	T1	T2	NC	C	T1	T2
Initial	39.22± 2.44^{a}	38.66± 3.64^{a}	37.62± 2.35^{a}	38.23± 2.70^{a}	1.22± 0.22^{a}	1.15± 0.33^{a}	1.09± 0.23^{a}	1.11± 0.23^{a}
Week 4	61.06± 5.99^{a}	59.67± 6.14^{ab}	58.56± 6.23^{b}	57.68± 7.29^{b}	3.87± 1.10^{a}	3.54± 1.22^{ab}	3.48± 1.10^{b}	3.31± 1.37^{b}
Week 10	92.12± 2.30^{a}	92.71± 3.10^{a}	84.49± 1.16^{b}	83.09± 0.69^{b}	14.26± 1.26^{a}	14.37± 1.42^{a}	11.22± 0.51^{b}	10.73± 0.22^{b}
Week 16	111.72± 16.45^{a}	115.30± 14.48^{a}	102.85± 15.72^{b}	102.90± 21.46^{b}	24.84± 10.40^{a}	26.99± 9.79^{a}	19.74± 8.37^{b}	20.31± 10.65^{b}

Table 2: Indices of cumulative growth rates (mean ± SEM, n=4) of juvenile *Oreochromis niloticus* during continuous exposure to contaminated harbour sediment over 16 weeks (NC= negative control without sediment; C= Control with pristine sediment; T1= contaminated harbour sediment; T2= three times of T1 harbour sediment) (significant difference between groups at $p<0.05$ is indicated by the absence of a shared superscript letter within a row separately for length and weight based indices; ANOVA and Tukey HSD test)

		length based (mm)				weight based (g)			
Index*	week	NC	C	T1	T2	NC	C	T1	T2
GR (day^{-1})	4	0.73± 0.03^{a}	0.70± 0.03^{a}	0.70± 0.01^{ab}	0.65± 0.05^{b}	0.09± 0.01^{a}	0.08 ± 0.01^{a}	0.08± 0.005^{a}	0.07± 0.007^{a}
	10	0.74± 0.04 ^{a}	0.76± 0.03 ^{a}	0.66± 0.02 ^{b}	0.63± 0.01^{b}	0.18± 0.02 ^{a}	0.18 ± 0.02 ^{a}	0.14 ± 0.01 ^{b}	0.13 ± 0.01^{b}
	16	0.65± 0.02 ^{a}	0.69± 0.03 ^{a}	0.59± 0.01 ^{b}	0.58± 0.05 ^{b}	0.21± 0.01^{a}	0.23 ± 0.01^{a}	0.17± 0.01^{b}	0.17± 0.03^{b}
RGR (% day^{-1})	4	1.855± 0.028^{a}	1.813± 0.028^{a}	1.856± 0.028^{a}	1.696± 0.028^{b}	7.223± 0.305^{a}	6.904± 0.305^{a}	7.298± 0.305^{a}	6.597± 0.305^{a}
	10	1.899± 0.034^{bc}	1.969± 0.034^{c}	1.755± 0.034^{ab}	1.653± 0.034^{a}	15.032± 0.534^{bc}	16.126± 0.534^{c}	13.066± 0.534^{ab}	12.186± 0.534^{a}
	16	1.665± 0.042^{ab}	1.786± 0.042^{b}	1.562± 0.042^{a}	1.525± 0.042^{a}	17.413± 0.727^{ab}	20.187± 0.727^{b}	15.383± 0.727^{a}	15.547± 0.727^{a}
SGR (% day^{-1})	4	1.475± 0.018^{a}	1.448± 0.018^{a}	1.475± 0.018^{a}	1.371± 0.018^{b}	3.839± 0.099^{a}	3.734± 0.099^{a}	3.864± 0.099^{a}	3.633± 0.099^{a}
	10	1.202± 0.015^{a}	1.231± 0.015^{a}	1.139± 0.015^{b}	1.093± 0.015^{b}	3.457± 0.045^{bc}	3.549± 0.045^{c}	3.281± 0.045^{ab}	3.193± 0.045^{a}
	16	0.943± 0.015^{ab}	0.984± 0.015^{b}	0.906± 0.015^{a}	0.891± 0.028^{a}	2.713± 0.038^{ab}	2.839± 0.038^{b}	2.607± 0.038^{a}	2.610± 0.038^{a}

* GR (absolute growth rate) = absolute gain/time; RGR (relative growth rate) = 100* (total gain/ initial)/time; SGR (specific growth rate) = 100*(*ln* L_2- *ln* L_0/t) and 100*(*ln* W_2- *ln* W_0/t).

Liver parenchymal cells were visible with spherical nucleus each on the sections from negative control and control tanks (Figure 2). The sinusoidal arrangement of parenchymal cells had clear margins and intra-hepatic pancreatic tissues were visible with acinar arrangement. In comparison, toxicopathic hepatic lesions developed in both sediment exposed groups on week 16 notably including extensive necrotic areas and reduced nuclear density. In addition, hydropic vacuolation and disrupted sinusoids occurred. T1 fish showed fragmented nuclei which is likely to be apoptosis. This evidence collectively suggest that the biological responses of the T1 and T2 fish exposed to contaminated sediment in the study can at least partly be attributed to the occurrence of sediment borne hydrocarbon pollution. Sediment borne contaminants are bioavailable and taken up by fish. Bioavailability of hydrocarbons and consequent pathological effects were confirmed by using oil-spiked sediment on English sole (McCain et al., 1978). Harbour sediment induced cytochrome P4501A (CYP1A) in European flounder, plaice (Eggens et al., 1996), greenback flounder (Mondon et al., 2001) and turbot (Kilemade et al., 2009) indicating the presence of specific inducer hydrocarbons and their uptake by fish from harbour sediments. Kubin (1997) observed a growth reduction in juvenile English sole exposed to sediment contaminated with PAH in the laboratory.

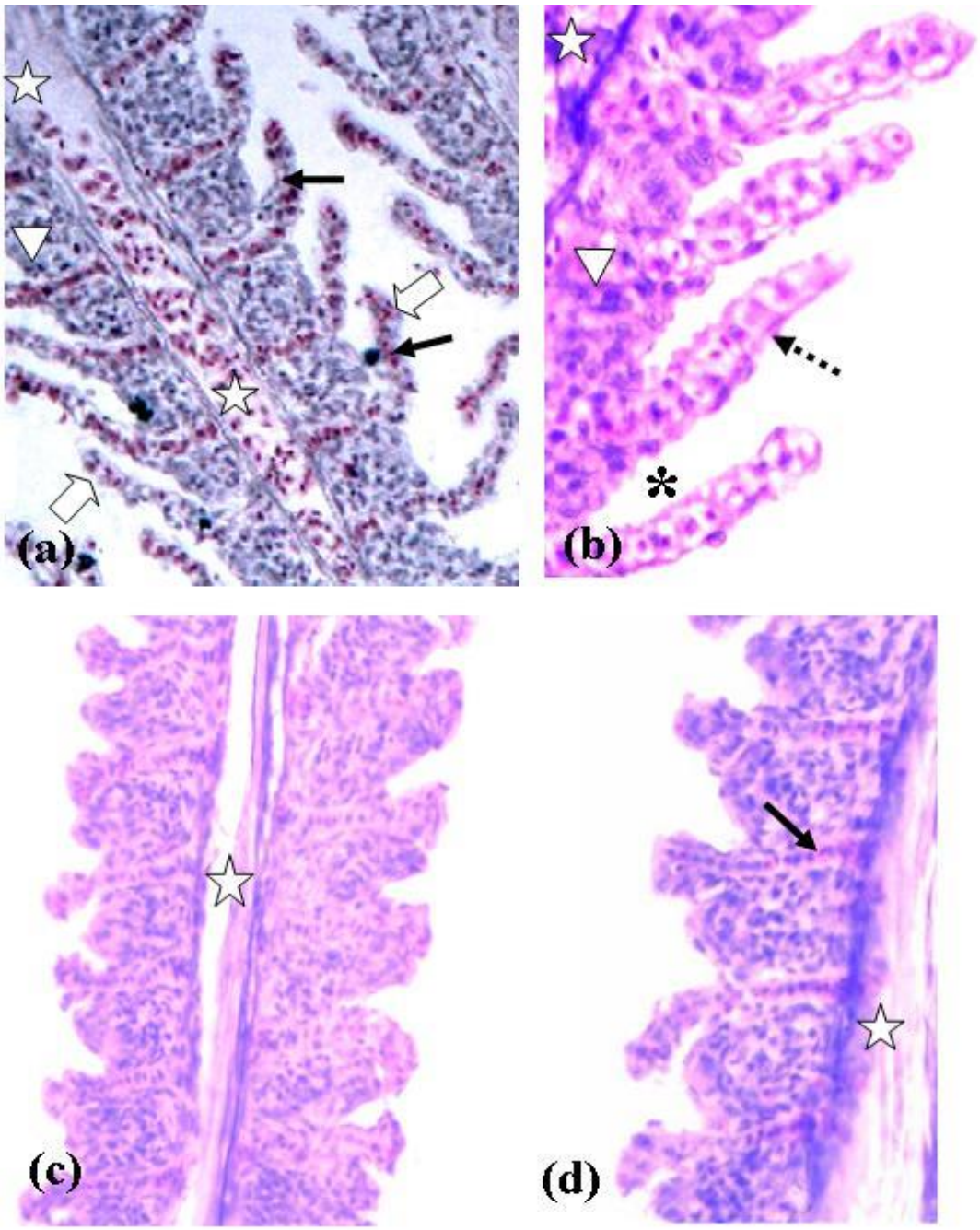

Figure 1. Photomicrographs of juvenile *Oreochromis niloticus* gill following 16 weeks of continuous exposure to harbour sediment; (a) negative control without sediment, (b) control with unit dose of pristine sediment, (c) T1 with unit dose of contaminated sediment, (d) T2 with 3 times T1 sediment by volume (locator symbols: white triangle= gill filament; white arrow= dorsal or ventral lamellae; asterisk= inter-lamellar space; star= filament artery with blood flow; line arrow= lamellar blood spaces; dashed line arrow= lamellar epithelium) (note: lamellae fusion and declined inter-lamellae space in inserts C and D)

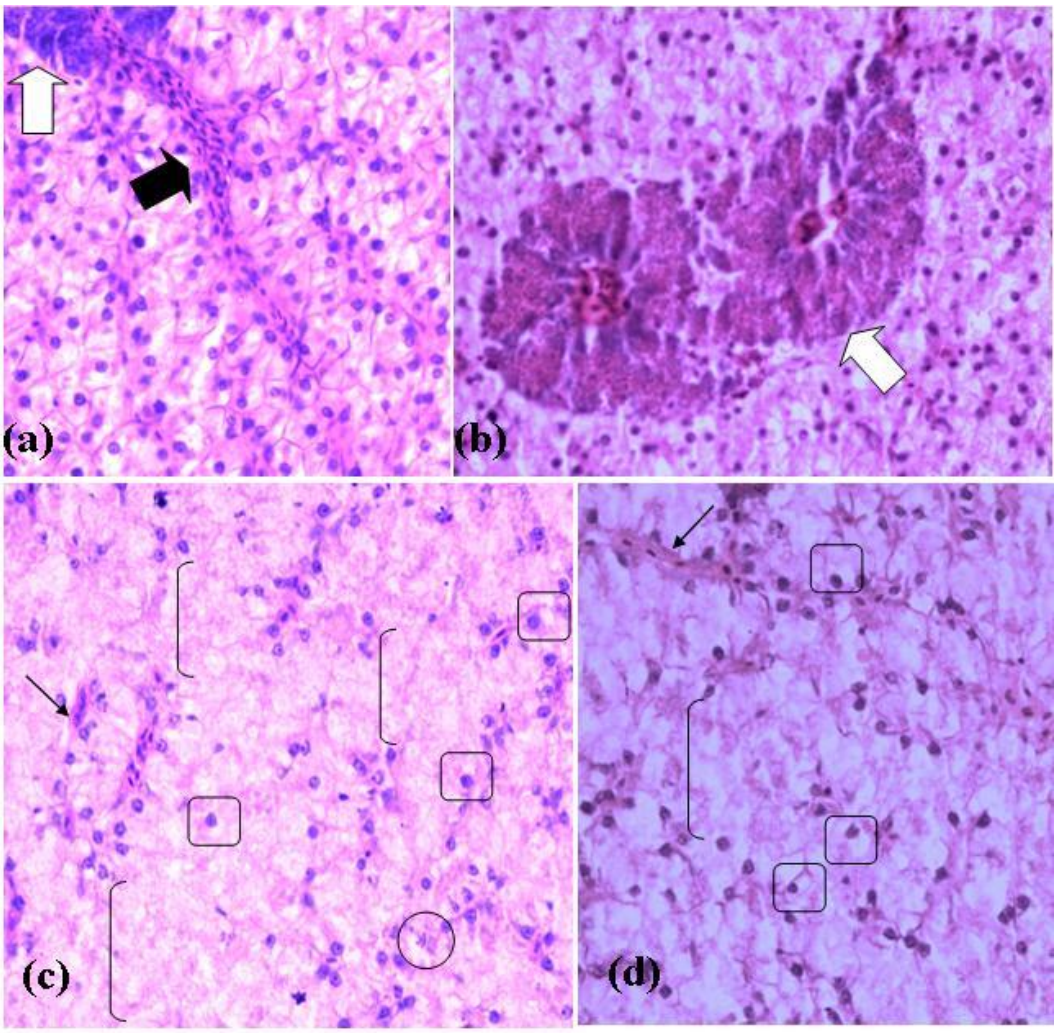

Figure 2. Photomicrographs of juvenile *Oreochromis niloticus* liver following 16 weeks of continuous exposure to harbour sediment; (a) negative control without sediment, (b) control with unit-dose of pristine sediment, (c) T1 with unit-dose of contaminated sediment, (d) T2 with 3 times T1 sediment by volume; (locator symbols: black arrow= sinusoidal arrangement; white arrow= intra-hepatic pancreatic tissue with central blood vessels; rectangle= hydropic vacuolation with characteristically eccentric nuclei; black line-arrow= disrupted sinusoids; circle= fragmented nucleus which is likely to be apoptosis; left bracket= necrotic areas with declined nuclear density in c and d).

The experiment was controlled so that the observed impact on growth could be attributed to pollutants present in harbour sediment. Results from previous laboratory studies point to harbour sludge elicited growth reductions in finfish. For instance, Rice et al., (2000) reported a decreased growth in juvenile English sole (*Pleuronectes vetulus*) that preyed on deposit-feeding polychaetes (*Armandia brevis*) in sediments from Puget Sound, Washington, USA. Moreover, sediments contaminated with organic pollutants and metals from French Boulogne-sur-Mer harbour also decreased growth of juvenile turbot, *Scophthalmus maximus* (Kerambrun et al., 2012b). Further, juvenile sea bass (*Dicentrarchus labrax*) and turbot (*Scophthalmus maximus*) cadged on sediments for 38 days in the same harbour underwent growth reductions (Kerambrun et al., 2012a). Although such diverse effects of individual contaminants of harbour sludge on fish are known, their collective impact as a distinct environmental compartment remains unclear to a large extent.

Table 3. Physico-chemical parameters (mean ± SD) of tank water over 16 weeks (NC= negative control without sediment; C= control with pristine sediment; T1= contaminated harbour sediment; T2= three times of T1 harbour sediment).

	NC	C	T1	T2
Temperature (°C)	27.8 ± 0.2	27.7 ± 0.2	27.6 ± 0.2	27.6 ± 0.2
Conductivity (mS)	0.043 ± 0.011	0.044 ± 0.011	0.042 ± 0.011	0.042 ± 0.012
Salinity (ppt)	0.16 ± 0.02	0.16 ± 0.03	0.15 ± 0.02	0.16 ± 0.03
Alkalinity (m mol L^{-1})	1.35 ± 0.32	1.49 ± 0.25	1.51 ± 0.22	1.49 ± 0.31
pH	6.5 ± 0.6	6.5 ± 0.3	6.6 ± 0.7	6.4 ± 0.3
Dissolved O_2 (mg L^{-1})	3.32 ± 1.13	3.56 ± 1.44	3.02 ±1.67	2.89 ± 1.75
Nitrates (μg L^{-1})	228.9 ± 10.4	232.1 ± 11.7	231.2 ± 8.9	231.0 ± 5.0
Phosphates (μg L^{-1})	290.7 ± 160.6	272.9 ± 167.3	265.1 ± 153.1	259.1 ± 149.4

Growth may be envisaged as a manifestation of the genetic potential under the influence of environmental factors and nutrition. Although, effects of many environmental chemicals at molecular to organ levels are known mostly on short-term exposure context, the impact of individual toxicants or their mixtures on growth remains hitherto unclear perhaps because it needs long-term laboratory studies which are rare and arduous. It is generally accepted that chronic stress may not be conducive for growth of fish (reviewed by van Weerd & Komen, 1998). The reduced growth of juvenile *O. niloticus* over 16 weeks of continuous exposure to harbour sediment in the present study provides wanted proof on this regard. Novelty of this reporting is further marked by the fact that growth reduction was shown by tropical freshwater species whose response emerged comparable to the reported results from the marine counterparts.

The results from gill and liver histopathology substantiate the observed impact on growth of juvenile tilapia. Tissue damage or alteration of gill filament and lamellar structure may compromise the gas exchange. Gill sections from negative control and control groups showed normal histology. T1 and T2 groups following 16 weeks of exposure to harbour sediment however showed increased mucus secretion, extensive lamellar fusion and decreased inter-lamellar space in gills. The symptoms collectively indicate gill damage and suggest impeded gas exchange. Gill lamellae are positioned serially on filaments and with gaps in between for unhindered water flow which ensures adequate oxygenation. When adjacent lamellae are fused, inter-lamellar space for water flow and surface area for gas exchange are lost. McNatt & Rice (2004) reported a hypoxia induced growth rate reduction in Atlantic menhaden (*Brevoortia tyrannus*) and spot (*Leiostomus xanthurus*). In such context, gill pathology could be linked to the observed growth reduction of juvenile tilapia exposed to contaminated harbour sediment.

Toxicopathic liver lesions have been reported in bottom dwelling marine flat fish species in harbour areas. The prevalence statistically associated the presence of aromatic hydrocarbon compounds in bottom sediments suggesting the causal link (Landahl et al., 1990; Schiewe et al., 1991; Myers et al., 1994, 1998; Stehr et al., 2003). Helder (1981) reported vacuolization, degeneration and necrosis of the liver parenchymal cells in rainbow trout juveniles exposed to 2,3,7,8-tetrachlorodibenzo-p-dioxin which is a contaminant of harbour sediment (Mohammed et al. 2009). Large necrotic areas were also seen in tropical freshwater fish *Hoplias malabaricus* in association with bioaccumulated PCB and chlorinated pesticides in liver (Miranda et al., 2008). Similar histopathological damages to liver were also shown by *Oreochromis niloticus* and *Oreochromis aureus* juveniles in relation to heavy metals (El-Naggar et al., 2009) and phenol (Abdel-Hameid, 2007) exposure respectively. The exposure-induced liver lesions in juvenile *O. niloticus* in this study included the signs of cellular necrosis, declined nuclear density, hydropic vacuolation with eccentric nuclei, disrupted sinusoids, and fragmented nuclei which appeared to be apoptotic. Notwithstanding the limiting statistical power (n=4), liver pathology investigation qualitatively revealed toxicopathic damage in general agreement with previous reports. Teleost liver is multifunctional and a vital organ, thus death or disruption of large areas of the liver parenchyma may result in impairment of health, growth and reproduction, and also eventual death.

Despite using marine sediments in the control and treatment groups, water quality remained similar ($p>0.05$) over the experimental period with no significant variations among the groups at time points monitored or among different time points within each group (Table 3). Variation in water quality parameters across the groups was minimal and non-significant possibly due to very low amount of seawater permeation resulted from the use of decanted sediment and low sediment volumes. Those trends collectively suggest that the biological responses of the T1 and T2 fish exposed to contaminated sediment in the study cannot be attributed to traces of sediment borne salts or any other water quality parameter. The results support exploratory notion of the study that juvenile *Oreochromis niloticus* could be employed in marine sediment exposure tests in fresh water. In addition, Wong et al., (2001) successfully exposed tilapia (*Oreochromis mossambicus*) to coastal sediments over seven days in sea water tanks and followed CYP1A responses. *O. niloticus* is a freshwater fish with euryhaline tolerance (Payne & Collinson, 1983) and that explains its amenability across wider salinity levels compared to other fresh water fish. Further, being a bentho-pelagic feeder (Oso et al., 2006), it frequently contacts bottom so that the species stands as a realistic candidate for sediment exposure tests. The evidence put forward by the present study is important for the workers who lack sea water systems and genetically monomorphic stocks of marine test species for sediment-exposure studies in laboratory.

The scarcity of long-term laboratory studies of fish responses to harbour sediment exposure remains an impediment in assessing the present results. To the best of our awareness, investigations involving freshwater fish and marine sediment are unprecedented or perhaps very limited. However, growth and histopathology responses of juvenile *O. niloticus* appear to be comparable with available reports of relevance. It promotes the utility of juvenile *O. niloticus* as a test organism for marine sediment exposure in freshwater systems. Such prospect will be supported by its known biology, testing-history in many areas of science, and availability as inbred-groups from breeding stations.

Conclusion

In conclusion, surficial marine sediment from Galle fisheries harbour reduced growth, and caused pathological lesions in gill and liver of *O. niloticus* juveniles over 16 weeks of continuous exposure. The results suggest the presence of environmental toxicants detrimental to growth and tissue health in the harbour sludge and emphasize the importance of cleaner practices pertaining to harbour environments.

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