Comparative Biochemistry and Physiology, Part C 183–184 (2016) 7–19

Contents lists available at ScienceDirect

Comparative Biochemistry and Physiology, Part C

journal homepage: www.elsevier.com/locate/cbpc

Peroxisome proliferator-activated receptors and biotransformation responses in relation to condition factor and contaminant burden in tilapia species from Ogun River, Nigeria

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ARTICLE INFO

Article history:
Received 30 October 2015
Received in revised form 16 December 2015
Accepted 19 December 2015
Available online 29 December 2015

Keywords:
Contaminants
PPAR isoforms
cyp1 isoforms
Obesogenicity
Tilapias
Condition factor

ABSTRACT

A major development in fishery science has been the Fulton's condition factor (CF) as a reliable physiological index of fish growth and health status (Fulton 1902). As a general rule, CF-value greater than 1 (>1) should be regarded as an indicator for good growth and health. Therefore, exposure of fish to contaminants in the environment will be expected to produce a reduction in scope for growth, since energy for growth will be allocated to overcome stressful conditions. In the present study, we hypothesized that tilapia species from Ogun River (Nigeria) are experiencing severe contaminant-induced obesogen effects leading to high CF (≥2) in fish with pathological alterations. The environmental obesogen hypothesis has related the interaction between environmental pollutants and PPAR isoform activation In this respect, peroxisome proliferator-activated receptors (PPARs) and biotransformation responses in relation to contaminant burden were investigated in a total of 1074 specimens of Tilapias species (*Tilapia guineensis*, *Sarotherodon galileaus* and *Oreochromis niloticus*) collected from three areas with different degrees of anthropogenic contamination and from a putative control site along the Ogun River. Liver mRNA expression of cytochrome cyp1 isoforms (cyp1a, 1b and 1c) and PPAR isoforms (*ppar*-α, β and γ) were analyzed using validated real-time PCR. Fish were also analyzed for CF and muscle contaminant burden (aliphatic and polycyclic aromatic hydrocarbons, organochlorine pesticides, and polychlorinated biphenyls). A significant increase in mRNA expression of *cyp*1- and *ppar* isoforms was observed in fish from polluted areas, and these results paralleled data on PCBs and PAHs tissue concentrations. Further, *cyp*1 isoforms showed clear sex-related differences, with higher mRNA expression in male fish than in females. Principal component analysis revealed a relationship between *cyp*1 isoforms, *ppar*-α, β, PCBs and PAHs and these interactions may suggest a crosstalk between AhR- and PPARs mediated pathways on metabolic and energetic processes. The PCA biplot also highlighted a positive relationship between *ppar*-γ, body weight, total length and PAHs. The CF for fish from all the sites was ≥2 indicating that this parameter may not be a reliable index for evaluating fish growth and health condition, especially in wild fish population exposed to complex cocktails of environmental pollutants.

1. Introduction

Accumulating evidence suggests that environmental contaminants may alter endocrine signaling, and consequently developmental and metabolic processes, including obesity (Newbold et al. 2009, Grun and Blumberg 2007, 2009a, 2009b). Recent data suggest that the commonly assumed causes of obesity such as over eating, inactivity and genetic pre-disposition, may not fully explain the current obesity epidemic (Swedenborg et al. 2009). In this regard, the increasing production and use of several synthetic chemicals suggested that environmental contaminants with endocrine disruptive mode of action (MOA) may play crucial roles in obesity development, by altering physiological and metabolic control mechanisms (Baillie-Hamilton 2002; Elobeid & Allison, 2008). Recent scientific reports have suggested a number of mechanisms linking endocrine disrupting chemicals (EDCs) to obesity which include, but are not limited to—alterations in thyroid and steroid hormone function, elevated triglyceride and cholesterol levels, activation of peroxisome proliferator activated receptor-γ (PPARγ) which play integral roles in adipocyte differentiation and energy storage (Janesick and Blumberg 2011; Pereira-Fernandes et al. 2013a, 2013b).

PPARs (α, β and γ isoforms) are nuclear receptors that regulate key molecular and cellular functions, including glucose and lipid metabolism, cell differentiation and adipogenesis (Latruffe et al. 2001; Tan et al. 2001; Casals-Casas and Desvergne 2011). PPAR isoforms

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http://dx.doi.org/10.1016/j.cbpc.2015.12.006
1532-0456/

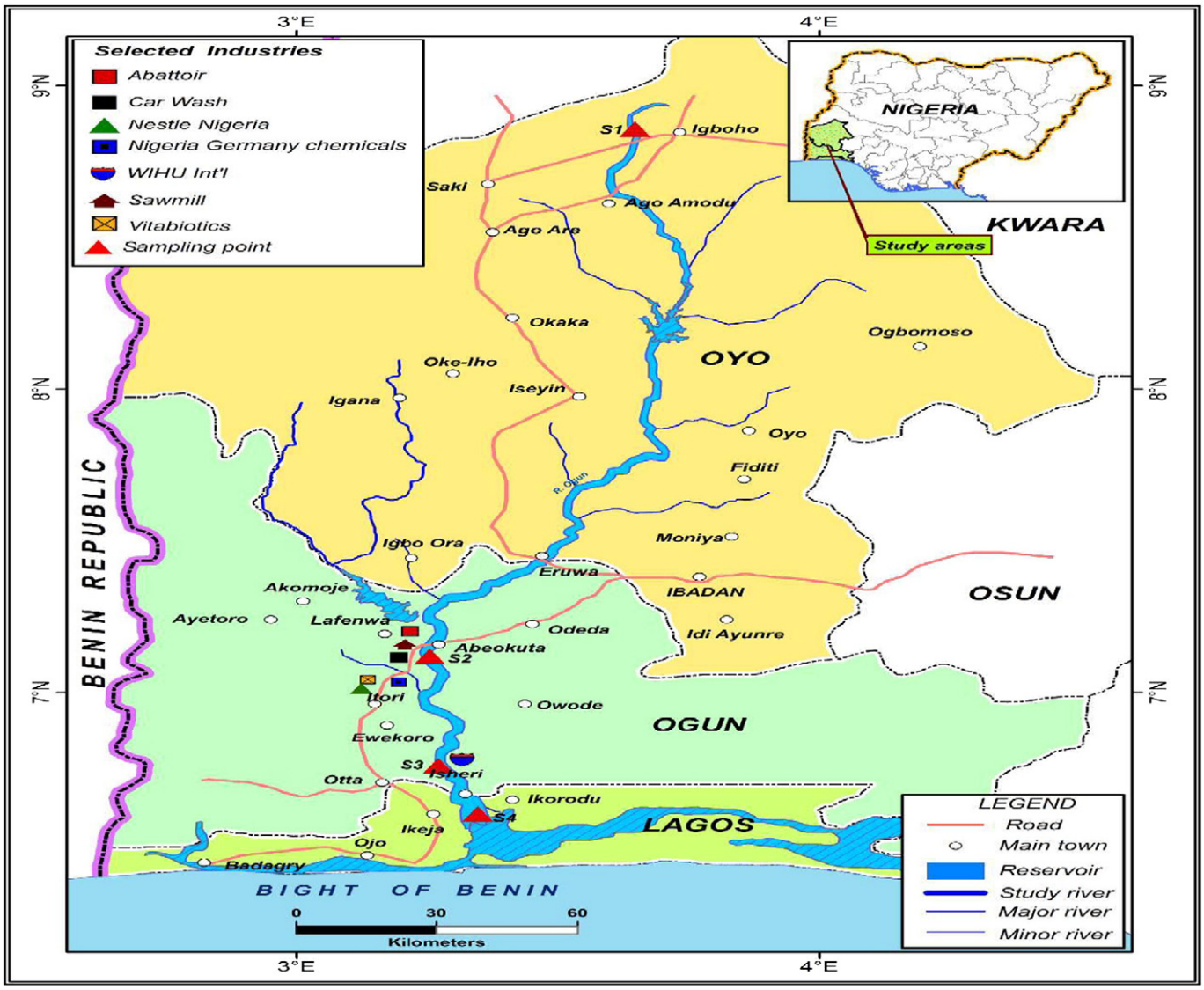

Fig. 1. Map of Ogun River showing the sampling sites.

show tissue-specific distribution patterns, where PPARα is primarily expressed in tissues with high fatty acid oxidation, including liver, heart, skeletal muscle, brain and intestine, PPARβ is ubiquitously distributed, and PPARγ is largely expressed in adipose tissues (Berger and Moller 2002). PPARα and PPARβ have been shown to regulate important biological processes including lipid and glucose metabolism (Mandard et al. 2004; Wang et al. 2008). In general, fish PPAR isoforms are homologous to their mammalian counterparts, although there have been reports on differences in their genomic structure (Leaver et al. 2005). The ligand-binding domains of PPARs are large, resulting in relatively high ligand promiscuity to a number of structurally diverse natural and synthetic compounds, including chemical contaminants (O'sullivan, 2007). In this respect, environmental obesogen hypothesis postulated that increasing exposure to anthropogenic pollutants may influence the development of obesity by activating the PPARγ regulated adipogenesis (Hectors et al. 2011; Janesick and Blumberg 2011; Pereira-Fernandes et al. 2013b). It has been established that persistent environmental contaminants, such as polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs), polychlorinated dibenzo-p-dioxins (PCDDs), polychlorinated dibenzofurans (PCDFs), organochlorine pecticides (OCPs) produces an array of toxicological effects on multiple pathways in teleosts (Isidori et al. 2010; Palermo et al. 2012), including the activation of PPARs and aryl hydrocarbon receptor (AhR) mediated responses (Regoli et al. 2011; Fang et al. 2012; Cocci et al. 2013).

The AhR is a transcription factor belonging to the helix–loop–helix-PAS (bHLH-PER-ARNT-SIM) family of gene regulatory proteins (Gu et al. 2000). After ligand binding, the AhR dimerizes with AhR nuclear translocator (Arnt), translocating to the nucleus where the complex activates mRNA transcription of xenobiotic response elements (XRE) containing genes, including cytochrome P450s (Gu et al. 2000). Cytochromes P450 (CYPs) play important roles in catalyzing the oxidative metabolism of contaminants. In particular, the CYP1 (a, b and c isoforms), 2 and 3 superfamilies catalyze the metabolism and detoxification PAHs, PCBs, TCDDs, PBDEs, OCPs and drugs (Nelson et al. 1996). In addition, they also play significant roles in the activation of pro-toxicants, pro-mutagens, oxidative biosynthesis and degradation of endogenous molecules, including steroids and fatty acids (Nebert et al. 2004; Shumada & Fujii-Kuriyama, 2004; Lewis, 2004). Due to the important roles of CYP1 isoforms in xenobiotic metabolism, the modulation in their expression patterns will ultimately affect the potential toxic mechanisms of EDCs (Williams et al. 1998; Regoli et al. 2014).

The obesogen activity of several contaminants with established endocrine disruptive effects has been reported under laboratory conditions (Grun et al. 2006; Grun and Blumberg 2009b; Penza et al. 2006;