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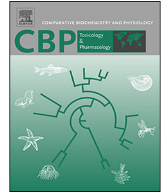
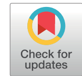


Effects of dietary crude oil exposure on molecular and physiological parameters related to lipid homeostasis in polar cod (*Boreogadus saida*)



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A R T I C L E I N F O

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A B S T R A C T

Polar cod is an abundant Arctic key species, inhabiting an ecosystem that is subjected to rapid climate change and increased petroleum related activities. Few studies have investigated biological effects of crude oil on lipid metabolism in this species, despite lipids being a crucial compound for Arctic species to adapt to the high seasonality in food abundance in their habitat. This study examines the effects of dietary crude oil exposure on transcription levels of genes related to lipid metabolism (*peroxisome proliferator-activated receptors [ppar-α, ppar-γ], retinoic X receptor [rxr-β], palmitoyl-CoA oxidase [aox1], cytochrome P4507A1* [*cyp7a1*]), reproduction (*vitellogenin [vtg-β], gonad aromatase* [*cyp19a1*]) and biotransformation (*cytochrome P4501A1 [cyp1a1], aryl hydrocarbon receptor [ahr2]*). Exposure effects were also examined through plasma chemistry parameters. Additional fish were exposed to a PPAR-α agonist (WY-14,643) to investigate the role of PPAR-α in their lipid metabolism. The dose-dependent up-regulation of *cyp1a1* reflected the activation of genes related to PAH biotransformation upon crude oil exposure. The crude oil exposure did not significantly alter the mRNA expression of genes involved in lipid homeostasis except for *cyp7a1* transcription levels. Plasma levels of cholesterol and alanine transaminase showed significant alterations in fish exposed to crude oil at the end of the experiment. WY exposure induced a down-regulation of *ppar*-α, an effect contrary to studies performed on other fish species. In conclusion, this study showed clear effects of dietary crude oil exposure at environmentally relevant concentrations on xenobiotic biotransformation but revealed only weak alterations in the lipid metabolism of polar cod.

1. Introduction

Polycyclic aromatic hydrocarbons (PAHs) are regarded as the primary toxic constituents in crude oil and are commonly studied with regard to biological effects of petroleum exposure in fish (*e.g.* Kane Driscoll et al., 2010; Vignet et al., 2014). Several effects have been related to PAH exposure in fish such as reduced growth (Meador et al., 2006; Vignet et al., 2014), diminished biological fitness (Kennedy and Farrell, 2006), immune dysfunction (Reynaud and Deschaux, 2006) and impaired reproduction (Nicolas, 1999). PAHs have also been shown to cause peroxisome proliferation in fish, a response characterized by an increased number and volume density of peroxisomes, usually accompanied by the transcriptional up-regulation of peroxisomal β-oxidation genes (Cajaraville et al., 2003). This process is suggested to be mediated through a subfamily of nuclear receptors called peroxisome proliferator activated receptors (PPARs) (Cajaraville et al., 2003), which also have been recognized as important lipid sensors and transcription factors that regulate lipid homeostasis in mammals (Feige et al., 2006). The three PPAR isotypes (α, β/δ, γ) are identified in marine fish (Andersen et al., 2000; Leaver et al., 2005; Raingeard et al., 2009) and a study on sea bass (*Dicentrarchus labrax*) suggested similar functions of marine fish PPARs as in mammals (Boukouvala et al., 2004). Although PAHs are identified as ligands for PPAR-α in human cells (Kim et al., 2005), this interaction is not known for fish. However, several studies have shown that petroleum compounds affect transcription levels of genes related to lipid metabolism (Bilbao et al., 2010; Adeogun et al., 2016; Xu et al., 2016; Cocci et al., 2017). Furthermore, PAH exposure altered lipid plasma parameters in Chinook salmon (*Oncorhynchus tshawytscha*) in a similar pattern of that found in starving fish (Meador et al., 2006). The physiological fasting response has been related to an up-regulation

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of PPAR-α transcription in mammals (Leone et al., 1999) and consequently, the effects of PAHs on lipid metabolism in fish could potentially be governed by the key regulator of lipid homeostasis, PPAR-α.

The Arctic is undergoing rapid climatic changes and climate models predict an ice-free Arctic Ocean during summer month by the middle of this century (IPCC, 2013). A reduction in sea ice unveils new opportunities for the petroleum industry, allowing exploration of petroleum resources on the Arctic continental shelves. The exploitation of petroleum resources in Arctic waters would, however, increase the risk of marine oil spills in this pristine ecosystem which could have detrimental effects on fish health and ultimately on fish populations (Peterson et al., 2003). Hence, the present study aimed to investigate the biological effects of crude oil exposure on an Arctic fish species that has been studied comprehensively during the last years. Polar cod (*Boreogadus saida*) is an important key species of the Arctic marine ecosystem that is highly abundant and circumpolar distributed in Arctic waters (Hop and Gjøsæter, 2013). It is an energy-rich and favored food item for Arctic marine predators such as sea birds and marine mammals and thereby represents an essential trophic link in the marine ecosystem of the Arctic (Hop and Gjøsæter, 2013). The high seasonality in light availability in this environment causes strong fluctuations in the availability of food for polar cod throughout the year and requires the rapid accumulation of energy in the form of lipids during summer month. This allows polar cod to survive months with sparse food available in the water column and it also enables the successful reproduction in winter. The present study examined the effects of crude oil exposure on important metabolic processes related to lipid homeostasis, reproduction and xenobiotic biotransformation in polar cod (*Boreogadus saida*). Although biotransformation of PAHs has been previously investigated (Nahrgang et al., 2010a, b), only few studies have examined the effects of petroleum-related compounds on processes relevant for lipid homeostasis and reproductive development in this species (Geraudie et al., 2014; Andersen et al., 2015; Bender et al., 2016). We hypothesized that crude oil exposure would affect mRNA expression of genes relevant for key processes in lipid metabolism (*ppar-α*, *ppar-γ*, *retinoic X receptor [rxr-β]*, *palmitoyl-coenzyme A oxidase [aox1]*, *cytochrome P4507A1 [cyp7α1]*), reproduction (*vitellogenin [vtg-β]*, *gonad aromatase* [*cyp19a1*]) and biotransformation metabolism (*cytochrome P4501A1 [cyp1a1]*, *aryl hydrocarbon receptor 2 [ahr2]*). We also expected crude oil to alter physiological indicators for lipid metabolism, as was found in salmon after dietary PAH exposure (Meador et al., 2006).

To study the biological effects of crude oil exposure we performed an experiment with wild polar cod that were exposed to three different crude oil doses at environmentally relevant concentrations for 4 weeks. Samples for molecular and physiological analyses were taken at five time points during the experiment and analyzed for endpoints related to lipid metabolism, reproduction and biotransformation. In addition, samples were also used for a parallel study that examined the effects of crude oil exposure on the antioxidant defense system and further endpoints related to biotransformation processes in polar cod, published in Vieweg et al. (2017). As PPARs have been suggested to mediate the adverse effects of PAH exposure on lipid homeostasis in marine organisms (Cajaraville et al., 2003; Bilbao et al., 2010), the present study included a potent peroxisome proliferator (WY-14,643 [WY]) as additional treatment in the exposure experiment. WY is a PPAR-α agonist and *aox1* regulator in mammals (Berger and Moller, 2002) and fish (Colliar et al., 2011; Urbatzka et al., 2015) and was used to investigate the potential role of PPARs in regulating lipid metabolism in polar cod. Previous experimental work on polar cod suggested dietary exposure as a relevant exposure route of lipophilic petroleum compounds (George et al., 1995; Nahrgang et al., 2010b; Bender et al., 2016). Polar cod shows slow gastrointestinal evacuation rates (Hop and Tonn, 1998) and high assimilation efficiencies (Hop et al., 1997), which was suggested to cause a high metabolic absorption of petroleum compounds (Nahrgang et al., 2010b). Other experimental fish studies have identified food as an important pathway for crude oil compounds to enter the organism and elicit adverse effects (*e.g.* Saborido-Rey et al., 2007; Martin-Skilton et al., 2008; Olsvik et al., 2011; Bratberg et al., 2013).

2. Materials and methods

2.1. Fish sampling and rearing

Polar cod were caught by trawling in Billefjorden and Rijpfjorden (Svalbard, Norway, latitude 79° N) during late January 2013, using the same trawling set-up as described in Nahrgang et al. (2010b). The research vessel R/V Helmer Hanssen, owned by the UiT-The Arctic University of Norway, is authorized by the Norwegian Fishery Directorate to perform bottom trawling to catch fish for scientific purposes. Fish were kept on board the research vessel in 500 L tanks supplied by constant running seawater until transferred to the research facilities of UiT-The Arctic University of Norway in Kårvika (Norway, latitude 69° N). Here, polar cod were kept in 60 μm filtered seawater supplied from the nearest fjord (Kvalsundet) with water flow at 7–10 L/min and temperature of 3 to 4 °C. Fish were acclimated for 3 months to the laboratory conditions. During acclimation, polar cod were given frozen *Calanus* sp. (purchased from CALANUS AS) *ad libitum* three times per week. One month prior to the start of the experiment, 250 fish were distributed into six experimental tanks (300 L) with 40 fish allocated to each of the 5 treatment tanks (3 crude oil treatments, 1 treatments for the PPAR-α model agonist WY and 1 treatment tank for the solvent control) and 50 fish allocated to the control tank. During this final acclimation step and the subsequent experiment, seawater supplied to the tanks was maintained at a mean (± SD) water temperature of 3.6 °C (± 0.3) and a mean dissolved oxygen level of 91.7% (± 5.2). The light regime in the tanks reflected *in situ* conditions in Svalbard (latitude 69° N) between April and May that is civil twilight, with 24 h daylight and lower light intensities during night. The experimental work was done in accordance with the laws of the Animal Welfare Act and regulations of the Norwegian Animals Research Authority (ID 5271). The experimental work was performed by the lead author, who has the necessary training and certificate (FELASA Category C) to perform experimental work with animals.

2.2. Experimental design

The set-up of the study consisted of two parallel feeding experiments, where polar cod specimens were exposed for 32 days to either Goliat Kobbe crude oil at four different doses (control, low, medium and high) or to the PPAR-α model agonist WY-14.643 (WY) and the appurtenant solvent control (acetone). Kobbe crude oil is a light crude oil that is produced and transported in the Barents Sea (Sørheim and Moldestad, 2008), hence a crude oil type that polar cod could be exposed to in a potential oil spill in Arctic waters. The feed preparation for the crude oil treatments and a detailed PAH composition of the fish feed are described in detail in Vieweg et al. (2017). Briefly, *Calanus* sp., a relevant and important natural food of polar cod (Hop and Gjøsæter, 2013), was mixed with four different doses of crude oil (0, 0.5, 2, 4 mg crude oil/g feed) without any solvent vehicle with a magnetic stirrer for 5 min. For the WY feeding experiment, the WY chemical was at first dissolved in acetone (16.8 μg/μL acetone) and subsequently mixed to *Calanus* sp. at a final concentration of 1.7 mg WY per g feed (Table 1). The appurtenant solvent control (So-Co) was prepared by mixing 101 μL acetone per g *Calanus* sp. Following, the acetone was volatilized both from the WY and So-Co treatments by constant stirring on a magnetic stirrer for 2.5 h at 30 °C. For all six feed preparations, starch (20% of the total feed weight) was added in order to increase the consistency of the feed and to allow force-feeding with minimal regurgitation from the fish. Food was supplied to the fish through force-feeding in order to control the exact dose received by each individual fish.

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