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An evaluation of the endocrine disruptive potential of crude oil water accommodated fractions and crude oil contaminated surface water to freshwater organisms using in vitro and in vivo approaches

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ABSTRACT:

Knowledge regarding the potential impacts of crude oil on endocrine signaling in freshwater aquatic vertebrates is limited. The expression of selected genes as biomarkers for altered endocrine signaling was studied in African clawed frog, Xenopus laevis, tadpoles and juvenile Mozambique tilapia, Oreochromis mossambicus, exposed to weathered bunker and unweathered refinery crude oil water accommodated fractions (WAFs). In addition, the expression of the aforementioned genes was quantified in X. laevis tadpoles exposed to surface water collected from the proximity of an underground oil bunker. The (anti)estrogenicity and (anti)androgenicity of crude oil, crude oil WAFs, and surface water were furthermore evaluated using recombinant yeast. Thyroid hormone receptor beta expression was significantly down-regulated in X. laevis in response to both oil WAF types, whereas a further thyroid linked gene, type 2 deiodinase, was up-regulated in O. mossambicus exposed to a high concentration of bunker oil WAF. In addition, both WAFs altered the expression of the adipogenesis-linked peroxisome proliferator–activated receptor gamma in X. laevis. The crude oil and WAFs exhibited antiestrogenic and antiandrogenic activity in vitro. However, O. mossambicus androgen receptor 2 was the only gene, representing the reproductive system, significantly affected by WAF exposure. Estrogenicity, antiestrogenicity, and antiandrogenicity were detected in surface water samples; however, no significant changes were observed in the expression of any of the genes evaluated in X. laevis exposed to surface water. The responses varied among the 2 model organisms used, as well as among the 2 types of crude oil. Nonetheless, the data provide evidence that crude oil pollution may lead to

adverse health effects in freshwater fish and amphibians as a result of altered endocrine signaling.