

Perfluoroheptanoic Acid Affects *Xenopus* Embryo Embryogenesis by Inducing the Phosphorylation of ERK and JNK

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Abstract : Perfluoroalkyl compounds (PFCs) are globally distributed synthetic compounds that are known to adversely affect human health. Developmental toxicity assessment of PFCs is important to facilitate the evaluation of their environmental impact. In the present study, we assessed the developmental toxicity and teratogenicity of PFCs with different numbers of carbon atoms on *Xenopus* embryogenesis. An initial frog embryo teratogenicity assay-*Xenopus* (FETAX) assay was performed that identified perfluorohexanoic (PFHxA) and perfluoroheptanoic (PFHpA) acids as potential teratogens and developmental toxicants. The mechanism underlying this teratogenicity was also investigated by measuring the expression of tissue-specific biomarkers such as phosphotyrosine-binding protein, xPTB (liver); NKX2.5 (heart); and Cyl18 (intestine). Whole-mount in situ hybridization, reverse transcriptase-polymerase chain reaction (RT-PCR), and histologic analyses detected severe defects in the liver and heart following exposure to PFHxA or PFHpA. In addition, immunoblotting revealed that PFHpA significantly increased the phosphorylation of extracellular signal-regulated kinase (ERK) and c-Jun N-terminal kinase (JNK), while PFHxA slightly increased these, as compared with the control. These results suggest that PFHxA and PFHpA are developmental toxicants and teratogens, with PFHpA producing more severe effects on liver and heart development through the induction of ERK and JNK phosphorylation.

Keywords : PFCs, ERK, JNK, *xenopus*

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