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Title: Fingerprinting the effects of plastic additives in the lipids of PLHC-1 topminnow liver cells

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Resumo

Plasticizers are widespread environmental contaminants that have been described as obesogens or chemicals that disrupt lipid metabolism in terrestrial vertebrates. However, its effects on aquatic vertebrates are almost unknown. This work explores the use of PLHC-1 cells as a tool to detect disruption of hepatic lipids after exposure to plastic additives and to gather information on the mode of action of the chemicals. PLHC-1 lipid extracts were analyzed by flow injection coupled to high resolution mass spectrometry (FIA-ESI(+/-)-Orbitrap-Exactive) after 24 h exposure of the cells to the selected plasticizers: dibutyl phthalate (DBP), diethyl-hexylphthalate (DEHP), bisphenols (BPA, BPF) and bisphenol A diglycidyl ether (BADGE·2HCl). The analysis of the intracellular concentration of chemicals revealed the highest intake of BADGE·2HCl and DEHP, which was in agreement with the strongest alteration of the cell lipidome. Thus, BADGE·2HCl induced a significant depletion of triacylglycerides (TGs) after 24 h exposure, while DEHP mainly affected membrane lipids, which led to a significant reduction of phosphatidylcholines (PC), phosphatidyl-ethanolamines (PE), phosphatidylinositols (PI) and diacylglycerols. Exposure to BPF induced the generation of reactive oxygen species in PLHC-1 cells and led to a significant depletion of lipids involved in cell protection against oxidative stress (PC- and PE-plasmalogens) and TGs (cell depots of PUFAs). DBP was the only compound that induced the intracellular accumulation of TGs. Overall, this study evidences the different modes of action of plastic additives in topminnow liver cells, describes differential lipidomic fingerprints, and highlights the higher toxicity of BADGE·2HCl and BPF compared to BPA.