A genome-wide expression analysis revealed the effects of hydroxylated polychlorinated biphenyls on the thyroid hormone function in metamorphosing African clawed toad *Xenopus laevis*.

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Production of polychlorinated biphenyls (PCBs) was banned in the 1970s. However, large amounts of these compounds remain ubiquitous in the environment. Hydroxylated polychlorinated biphenyls are the metabolites produced from parent compounds by the drug-metabolizing enzyme cytochrome P450. These compounds are suspected to disrupt post-embryonic neural development in the brains of mammals including humans. Because thyroid hormones play a role in the perinatal development of the central nervous system, the thyroid system is thought to be one of the important endpoints affected by several endocrine-disrupting chemicals including PCBs. We studied the effects of these compounds on thyroid hormone function in the brain by using metamorphosing tadpoles of the African clawed toad as a model for mammalian post-embryonic development. The metamorphosis assay revealed that these compounds inhibit thyroid hormone-induced metamorphosis. Genome-wide gene expression analysis in the brain following short-term exposure demonstrated that delayed metamorphosis could partially be caused by disruption of thyroid hormone-induced gene expression. Furthermore, we associated the terms of functional ontology with the genes whose expression was disrupted by these compounds. We suggest that use of a genome-wide analysis coupled with bioinformatics might provide an overview of the molecular mechanism underlying thyroid-disrupting activities *in vivo*.