

**MP108 EROD activity as a biomarker of exposure in field and laboratory birds exposed to environmentally relevant PCB mixtures**

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Ethoxyresorufin-*O*-deethylase (EROD) activity has been used as a biomarker of dioxin exposure and for assessing exposure of wildlife to dioxin-like compounds such as polychlorinated biphenyls (PCBs). We studied EROD activity in experiments conducted in field birds (*Tachycineta bicolor* and *Sialia sialis*) and in laboratory Japanese quail (*Coturnix japonica*). Birds were treated during embryonic development with selected doses of one of the following: two PCB mixtures found in the upper Hudson River environs, PCB 126, or PCB 77. Tree swallows had a relatively flat response to PCB 77, while the two PCB mixtures and PCB 126 stimulated increasing levels in EROD. Quail showed similar responses, with a strong response to PCB126 and the two mixtures (GENMOD procedure; personal communication, Franca Barton) . The maximal fold-difference over control in the 126-treated birds was 5.5, however there was increased mortality at the higher doses. Maximal induction was observed (9.3 fold) with one of the mixtures, however again the increased mortality at the higher doses of PCB 126 may have masked the EROD response. The PCB mixture also induced an initial strong response followed by a plateau and a further increase with increasing dose for a final fold-difference of 14, and a significant cubic function for this mixture's dose-response curve ( $p < 0.0001$ ). There were mild gender differences. PCB 77 elicited a slow flat EROD response. These data provide evidence that PCB 77 may not have strong dioxin-like biological activity in some avian species, which is in agreement with a previous WHO report that has proposed a recalculation of the PCB 77 TEF from 0.0005 to 0.0001 (Van den Berg, et al., 1998. Environmental Health Perspectives 106(12): 775-792.). As such, indices of toxicity such as TEQs and TEFs should be utilized to estimate potential effects, with the recognition that these calculations may not fully inform mechanisms associated with endocrine disruption and other non-aryl hydrocarbon receptor mediated actions. Supported in part by the Hudson River Trustees (FWS; MAO). The conclusions and opinions presented here are those of the authors, they do not represent the official position of any of the funding agencies, the Hudson River Trustees, or the United States.