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Accumulation of PCDDs/DFs and Coplanar PCBs and Induction of Cytochrome P450 in Black-tailed Gull and Black-footed Albatross

Hisato Iwata[1)], Mafumi Watanabe[1)], Tatsuya Kunisue[1)], Nobuhiro Fujii[1)] Shinsuke Tanabe[1)], Hiroyuki Tanaka[2)], Haruo Ogi[3)] and Yasuyuki Shibata[4)]

1) Center for Marine Environmental Studies, Ehime University, Japan

2) National Research Institute of Fisheries and Environment of Inland Sea, Japan

3) Faculty of Fisheries, Hokkaido University, Japan

4) National Institute of Environmental Studies, Japan

Planar halogenated aromatic hydrocarbons (PHAHs) including polychlorinated dibenzo-*p*- dioxins (PCDDs), furans (PCDFs) and coplanar PCBs are widely distributed and biomagnified in higher trophic birds through the food web. The PHAH exposure has resulted in a number of adverse effects on their reproductive potential, such as deformities and lethality of embryos. PHAHs bind to AhR and participates in regulating the transcription of cytochrome P450 1A (CYP1A) and other genes. The changes in these translated products are speculated to associate with disruption of cell cycle control, apoptosis, oxidative stress and endocrine signalling. These integrated knowledges indicate that accurate measurements of the AhR-mediated responses including CYPlA may lead to the detection of PHAN exposure and their subtle effects. Therefore, this study presents the current residue levels of PCDDs/DFs and coplanar PCBs in black-tailed gull (BTG; *Larus crassirostris*) and black-footed albatross (BFA; *Diomedea nigripes*), and further evidences of the induction of GYP by such PHAHs. Liver samples of BTG and BFA were collected from Rishiri island, Hokkaido in 1999-2001 and from the North Pacific in 1998, respectively. All the liver samples contained detectable amounts of PCDDs/DFs and coplanar PCBs. Coplanar PCBs, particularly non-*ortho* coplanar PCB 126 made a greater contribution to total TEQs. In BTG, TEQs from PCB 126 and PCB 169 exhibited significant positive correlations with EROD activity. In BFA, PCB 169 and some highly chlorinated PCDDs/DFs were correlated with EROD activity. Lower chlorinated PCDDs/DFs and coplanar PCBs, which are relatively biodegradable congeners, tended to show poor correlations with EROD activity in both species. These results indicate that AhR is activated by the PHAHs, and a CYP isozyme that is responsible for EROD activity, probably CYPIA, is induced in both species. Threshold values for hepatic EROD induction, which is defined as total TEQ at EROD level = 0 from simple regression analyses of total TEQ and EROD in the liver, were estimated to be approximately 5 pgTEQ/g wet wt in BTG and 30 pgTEQ/g wet wt in BFA. Interestingly, BTG microsomal samples showed a decrease in EROD activity at higher TEQ values. There are likely multiple causes for declining EROD activity at high TEQs, including suppression of the catalytic function and CYP1A protein expression.