

**DIETARY EXPOSURE TO PCB 126 AND BENZO(A)PYRENE [B(A)P]
INTERFERES WITH THE STRESS RESPONSE OF TELEOST FISH
AT THE PITUITARY LEVEL OF THE HPI AXIS**

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EXTENDED ABSTRACT ONLY - DO NOT CITE

Polychlorinated biphenyls (PCBs) and polyaromatic hydrocarbons (PAHs) are highly toxic pollutants structurally resembling steroid hormones, interfering with synthesis and action of gonadal and adrenocortical steroids (Safe, 1984), and impairing the stress response in fish (Hontela, 1998). The stress response of fish, co-ordinated via the hypothalamus-pituitary-interrenal (HPI) axis is affected by environmental toxicants (Wendelaar Bonga, 1997), but the level(s) at which this occurs are unclear. To further elucidate the effects that organic environmental pollutants have on the stress response in teleost fish, we examined the effects of dietary benzo(a)pyrene [B(a)P] and PCB126 exposure on the stress response of fresh-water and marine fish and subjected the fish to additional procedures known to activate the HPI axis, in particular net confinement (Wendelaar Bonga, 1997) and ACTH injection (Girard, et al.1998).

Tilapia (*Oreochromis mossambicus*) and rainbow trout (*Oncorhynchus mykiss*) were exposed to PCB126 and turbot (*Scophthalmus maximus*) were exposed to Benzo(a)pyrene [B(a)P], via the diet ($50 \mu\text{g} \cdot \text{kg bodyweight}^{-1} \cdot \text{day}^{-1}$). After 5 or 7 days, blood samples were taken at-rest, to obtain basal plasma levels of the investigated parameters, or after net-confinement, testing the responsiveness to a superimposed stressor. Plasma was analysed for ACTH (trout and tilapia), cortisol and glucose (all species) and free-fatty-acids (turbot). Basal hormone and metabolic-fuel levels were unaffected by either toxicant. Confinement

resulted in significant increases of all parameters. However, ACTH levels after confinement were significantly higher in PCB fed fish, cortisol responses to confinement were unaffected by either toxicant, and impaired hyperglycaemic responses to confinement were only observed after PCB-exposure. Turbot were also injected with ACTH (to mimic an activation of the HPI-axis providing a test for ACTH-responsiveness). This resulted in similarly increased cortisol and metabolic-fuel levels in B(a)P and control fed turbot.

Hereafter fish were treated differently: trout and tilapia were starved for 3 weeks to allow for mobilisation of stored PCBs (Carlson, 1980), and were then sampled at-rest or after confinement. Turbot were fed B(a)P for another 3 weeks, then sampled at-rest, after confinement or after ACTH-injection. Despite the different treatments during the second part of the experiments, the results obtained showed certain levels of similarity: Both starvation after previous PCB exposure and continuous exposure to B(a)P resulted in lower metabolic-fuel levels in fish exposed to contaminated diets, but basal hormone levels were unaffected by either toxicant. Confinement resulted in elevated hormone levels but these were significantly lower in toxicant-treated than in control fish. Injecting turbot with ACTH, resulted in significant increases of plasma hormone and metabolic fuel levels in both groups, indicating that ACTH-responsiveness was similar in control and B(a)P-fed fish.

Resting plasma hormone levels, indicative of a primary stress response and metabolic fuel levels, indicative of secondary stress responses (Wendelaar Bonga, 1997), were not influenced by the treatment with either toxicant, indicating that the fish were not stressed by the toxicant treatment itself. However, both toxicants impaired the ability of the fish to respond to an additional (non-toxic) stressor in the same fashion as control fish and this is in agreement with data observed in feral fish caught from sites contaminated with various chemicals including PCBs and PAHs compared to fish captured from control sites (Hontela, 1998). The higher confinement-induced increases in plasma ACTH levels in PCB fed trout and tilapia, seen after 1 week of exposure, are thought to be compensatory for depressed corticosteroid responses. Those only become obvious after toxicant exposures of longer than 1 week and are also apparent when fish were starved after the initial exposure period, resulting in lower confinement-induced plasma cortisol levels in exposed fish after both continuous toxicant exposure or starvation after initial exposure. The absence of the above mentioned compensatory mechanisms at the end of the additional starvation period (increasing exposure to PCB) together with the unaltered ACTH sensitivity even after 4 weeks of continuous exposure to B(a)P, suggest

that dietary exposure to organic toxicants impairs the co-ordination of the stress response in teleost fish at the pituitary level of the HPI axis.

References

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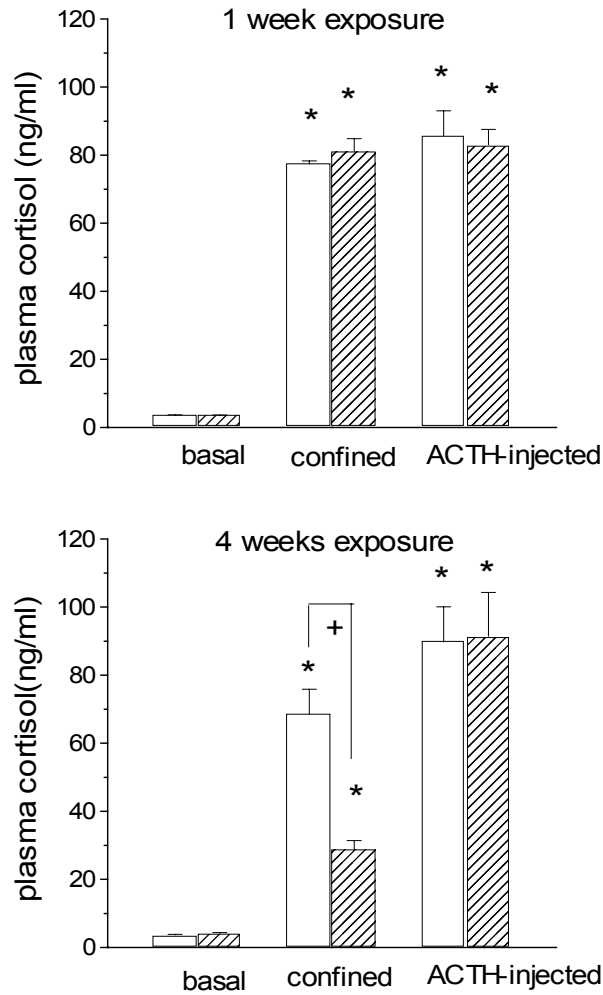


Figure 1: Effect of dietary B(a)P on the primary stress response in turbot. Data for trout and tilapia are published (Quabius et al. 2000) Environmental Toxicology and Chemistry Vol 12 p 2892 - 2899. Open bars represent control fish and hatched bars represent fish receiving the B(a)P containing diet (n=8 +/- SEM). Asterisks indicate significant differences from basal sample, and pluses indicate significant differences due to the B(a)P exposure.

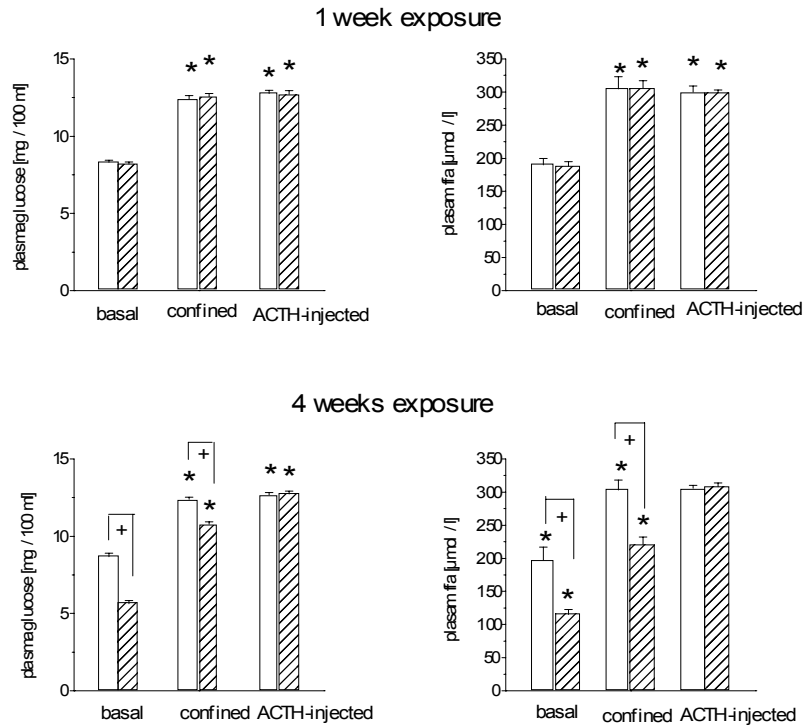


Figure 2: Effect of dietary B(a)P on secondary stress responses in turbot. Data for trout and tilapia are published (Quabius et al. 2000) Environmental Toxicology and Chemistry Vol 12 p 2892 - 2899. Open bars represent control fish and hatched bars represent fish receiving the B(a)P containing diet (n=8 +/- SEM). Asterisks indicate significant differences from basal sample, and pluses indicate significant differences due to the B(a)P exposure.

