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The impact of PAH exposure on Gulf toadfish, *Opsanus beta*, on post-stress plasma cortisol, glucose, and liver glycogen levels

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Introduction

- The 2010 *Deepwater Horizon* (*DWH*) disaster exposed marine fish toxic polycyclic aromatic hydrocarbons (PAHs) that are known to inhibit their glucocorticoid stress response.[1]
- Concentrations of stress hormone cortisol have been shown to decrease in PAH-treated fish compared to control fish when toadfish are subjected to crowding stress.[2]
- Metabolic response to cortisol secretion generally includes increased plasma cortisol and glucose levels and a decrease in hepatic glycogen levels.[3,4]
- The presence of environmental pollutants may decrease an organisms' fitness due to the coupled effects of direct toxicity as well as inhibition of stress response, which ultimately impacts populations and marine ecosystems.

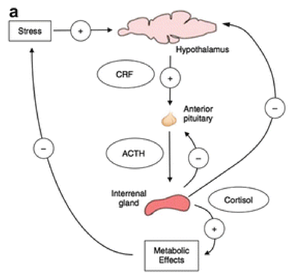

Figure 1: The HPI axis which mediates fish glucocorticoid stress response. [10]

Objective and Hypothesis

- To better understand PAH impact on stress response, we measured plasma cortisol and glucose levels as well as liver glycogen concentrations in PAH-treated fish following exposure to an acute stressor.
- We hypothesized that PAH exposure would result in a downregulation of cortisol release under two different acute stress regimes and would consequently impact blood glucose and liver glycogen levels.

Methods

- Gulf toadfish were obtained from Biscayne Bay shrimpers.
- Toadfish (n = 16) were randomly intraperitoneally (IP) injected with peanut oil alone or peanut oil with either fluorene, phenanthrene, or naphthalene and incubated 72 hrs.
- Toadfish (n=7-8) from each treatment were exposed to either a manual simulated predation chase (*Stress Regime #1*) or a more predictable, automated chase (*Stress Regime #2*).
- Blood samples were taken immediately after stress exposure and later analyzed via colorimetric assay for glucose and radioimmunoassay for cortisol. Liver samples were analyzed via colorimetric assay for glycogen.
- ANOVA and Student's t-test used to find differences between control and treatment groups.

Results

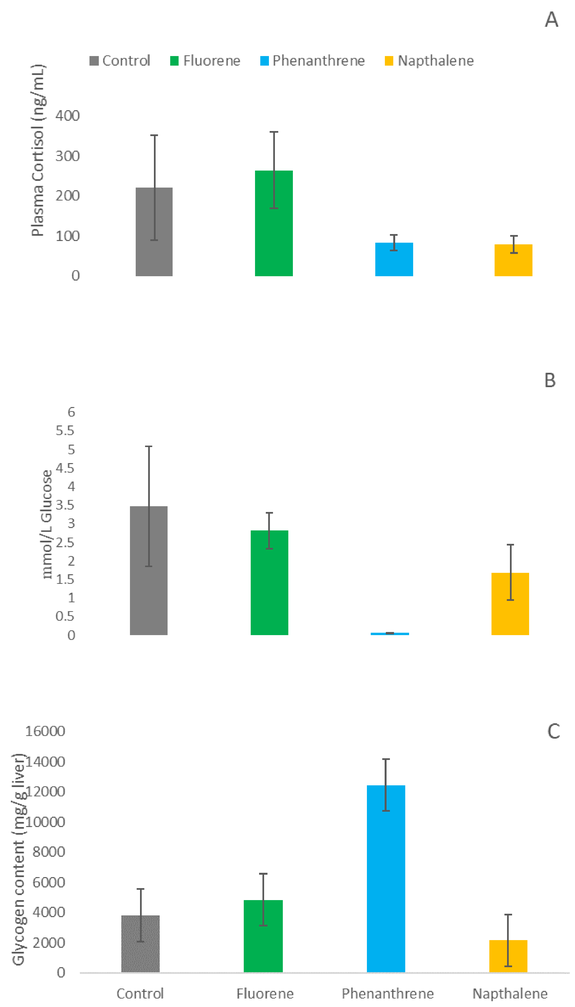

Figure 2: (A) Fish exposed to *Stress Regime #1* and treated with PAHs do not have a significant difference in plasma cortisol compared to control fish, (B) do not have a significant difference in plasma glucose concentration compared to control fish, and (C) do not have a significant difference in liver glycogen concentrations compared to control fish. Values are means ± SEM.

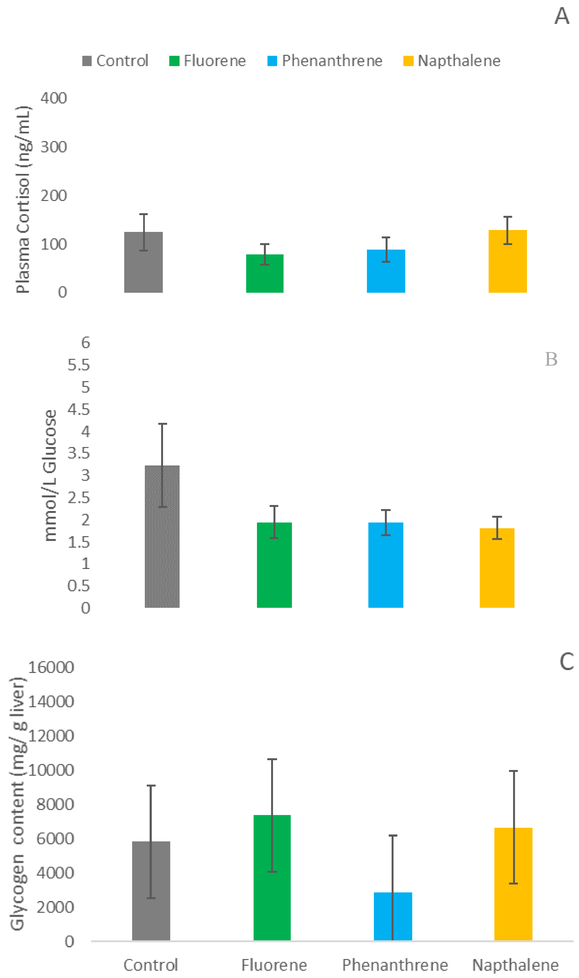

Figure 3: (A) Fish exposed to *Stress Regime #2* and treated with PAHs do not have a significant difference in plasma cortisol concentrations compared to control fish, (B) do not have a significant difference in glucose concentrations compared to control fish, and (C) do not have a significant difference in liver glycogen concentrations compared to control fish. Values are means ± SEM.

Discussion

- *Stress Regime #1* elicited a two-fold stronger stress response in control fish compared to levels achieved in *Stress Regime #2*; automated stress may be a more consistent acute stressor that allows fish to habituate.
- Phenanthrene-treated fish saw reduced cortisol release and lower plasma glucose and higher liver glycogen in response to *Stress Regime #1*, when compared to control fish; this was not true for *Stress Regime #2*, which suggests that there may be a "threshold" at which stress response downregulation effects become apparent. [5]
- Our trends for phenanthrene-treated fish were in line with other studies on toadfish; this suggests that it stimulates cortisol elevation initially, but phenanthrene exposure may cause exhaustion of the stress response, via kidney Melanocortin 2 receptor (MC2R) downregulation, when fish are subjected to additional stress. [2]
- PAHs may also have direct toxicity effects on cortisol synthesis via the aryl hydrocarbon receptor (AhR), which induces biotransformation and maybe oxidative stress. [6]

Conclusions

The HPI axis is a multifaceted cascade of signals that may each be susceptible to downregulation in MC2R receptor due to exhaustion, AhR-mediated oxidative stress, or their combined effects on cortisol synthesis.

Limitations of the study include neglecting to measure resting cortisol levels and coupling cortisol downregulation with glucose and glycogen changes.

Additional studies may elucidate if MC2R downregulation can be linked to antioxidant exhaustion at the kidney.

PAHs and other environmental pollutants compromise the integrity of the fight-or-flight response, which can have far-reaching effects on populations of organisms within the marine ecosystem.

Acknowledgements

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References

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