Marine algal toxins and endocrine disrupting chemicals (EDCs) have been reported to cause similar behavioral, morphological, and reproductive effects in marine biota, but it is not yet clear the mechanisms that are related. The responses of anthropogenic EDCs such as pesticides have been well defined and suggested to be based on androgen and estrogen receptor regulation. However, the mechanisms that cause these effects have not been directly studied for marine algal toxins. With the prevalence of HABS potentially increasing on temporal and spatial scales, potential organismal exposure may be important and chronic and sublethal effects of HAB toxins on ED are largely unexplored. In this study, we evaluated the effects of a variety of HAB toxins on androgen transcriptional activity using a cell-based reporter assay for the detection of (anti-)androgenic activity via expression of a luciferase reporter gene from the AR-Ecoscreen cell line. Likewise, estrogen receptor effects were evaluated in vitro using the T47D-KBluc cell line, a breast cancer line, transfected with an estrogen response element, also expressing a luciferase response. Ciguatoxins, maitotoxins, okadaic acid, and a variety of microcystins were evaluated in AR and ER agonist and antagonist assays. While ED effects have been reported in the literature by these compounds, our results showed no effect on AR transcriptional activity from HAB toxins when compared to positive androgen agonist (dihydrotestosterone) and antagonist (hydroxyflutamide). Also, we have not observed any effects on estrogen mediated proliferation when compared to estrogen, 17α-ethinylestradiol. Next steps will involve the evaluation of estrogen receptor specific (anti-) agonistic activity of a variety of phycotoxins as well as combined toxin exposures. Based on these data, we have initiated a controlled in vivo exposure in Fundulus grandis to evaluate other mechanisms and effects of marine toxins on the endocrine system.

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