Using Cnidarian Nerve Nets To Visualize the Excitotoxic Effects of BMAA

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Harmful algal blooms on lakes across the globe have become more common as a result of climate change. Cyanobacteria in the blooms produce harmful toxins which have significant consequences to animal and human health. One of these toxins is Beta-Methylamino-L-Alanine (BMAA), a non-proteinogenic amino acid which is structurally similar to glutamate. BMAA has been shown to have neurotoxic effects on cell cultures, rodents, and birds. It has also been implicated in causing an ALS-like condition affecting the Chamaroo people of Guam called ALS-PDC. In this study, I use Nematostella vectensis, a cnidarian with a simple nerve net to understand the effects of BMAA. I exposed Nematostella polyps to various concentrations of BMAA and found that the LD50 for BMAA is 5uM. One explanation for this effect is that BMAA is misincorporated into proteins in place of the amino acid Serine. Therefore, I added 0.1 uM Serine to polyps and incubated with 1uM BMAA and found polyps are protected against BMAA neurotoxicity. Additionally, I found polyps exposed to BMAA exhibit peristaltic rates greater than control animals. This change is correlated with an increase in intracellular calcium levels as shown by the fluorophore, calcium crimson. Taken together, this work shows that Nematostella can be used as a model system and provides a simple screening assay to understand the toxic effects of compounds from cyanobacteria in animals and humans. This is critically important because climate change has increased the prevalence and persistence of harmful algal blooms making these neurotoxins of greater concern.

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