11TH U.S. SYMPOSIUM ON HARMFUL ALGAE

IT TAKES A COMMUNITY TO TOXIFY: MECHANISMS OF TOXICITY IN FIVE COMMONLY COHABITATING EPI-BENTHIC DINOFLAGELLATES

Ciguatera poisoning (CP) is a global seafood illness caused by the consumption of fish contaminated with algal derived toxins produced by benthic dinoflagellates of the genus Gambierdiscus. Thus far, known toxins produced by these benthic dinoflagellates include ciguatoxins (CTXs) and maitotoxin (MTX). These toxins are known to exert primary toxicity via over-activation and blockage of voltage gated sodium and potassium channels and calcium channels, respectively. However, the symptomology associated with CP is complex and the known activity of CTX and MTX alone may not explain the complex poisoning syndrome. Furthermore, algal extracts have been shown to produce unexplained, non-specific, yet dose dependent activities in sodium channel specific in vitro assays suggesting that other metabolites may be contributing to composite neurotoxicity. The objective of this work was to further examine the bioactivity of five six benthic dinoflagellate species that co-occur in the biosphere of macrophytes in regions where CP is common. We hypothesized that these species contained intracellular secondary metabolites with additional and alternate bioactivity to sodium or potassium channel interactions. Growth rates were characterized for cultures Gambierdiscus belizeanus, G. silvae, Amphidinum sp., Coolia malayensis, and Prorocentrum rhathymum. Growth rates differed across species with Amphidinium sp. exhibiting the highest rate of growth. Bioactivity was evaluated based on hemolysis (mammalian red blood cell lysis) and acetylcholine esterase (AChE) activity. Whole cell extracts of G. belizeanus, G. caribaeus, G. silvae, Amphidinium sp., Coolia malayensis, and Prorocentrum rhathymum caused hemolysis in mammalian red blood cells at ecologically relevant levels. Data associated with AChE activity is currently underway and will be reported and compared across species and strains. Collectively, these data may contribute to our understanding of the complex mechanisms of action and related symptoms associated with CP.

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