Abstract

An unprecedented bloom of the cyanobacterium Microcystis aeruginosa Kütz. occurred in the St. Lucie Estuary, FL in the summer of 2005. Samples were analyzed for toxicity by ELISA and by use of the polymerase chain reaction (PCR) with specific oligonucleotide primers for the mcyB gene that has previously been correlated with the biosynthesis of toxic microcystins. Despite the fact that secreted toxin levels were relatively low in dense natural assemblages (3.5 microg l(-1)), detectable toxin levels increased by 90% when M. aeruginosa was stressed by an increase in salinity, physical injury, application of the chemical herbicide paraquat, or UV irradiation. The application of the same stressors caused a three-fold increase in the production of H(2)O(2) when compared to non-stressed cells. The application of micromolar concentrations of H(2)O(2) induced programmed cell death (PCD) as measured by a caspase protease assay. Catalase was capable of inhibiting PCD, implicating H(2)O(2) as the inducing oxidative species. Our results indicate that physical stressors induce oxidative stress, which results in PCD and a concomitant release of toxin into the surrounding media. Remediation strategies that induce cellular stress should be approached with caution since these protocols are capable of releasing elevated levels of microcystins into the environment.