Impact of chlorine on cyanobacterial neurotoxin β-*N*-methylamino-L-alanine (BMAA): reaction rate and intermediates

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Abstract

β-N-Methylamino-L-alanine (BMAA), has been reported to be biomagnified in the food chain and may cause amyotrophic lateral sclerosis/parkinsonism–dementia complex (ALS/PDC) or Alzheimer's disease. Although many studies have identified that the chemical can be produced by more than 20 genera of cyanobacteria, the removal and fate of BMAA in drinking water has never been reported before. Since chlorine is the most common oxidant/disinfectant used in drnking water treatment process, there is a need to understand the interaction between chlorine and BMAA.

In this study, the reaction pathway of BMAA, the formation of intermediates and their reaction kinetics during chlorination process were elucidated. A liquid chromatography coupled with triple quadrupole mass spectrometry was employed to quantify BMAA and identify its chlorinated intermediates. BMAA was found to react with HOCl to form four different chlorinated-intermediates, with 1 or 2 chlorine atoms. The reaction of BMAA with chlorine follows a second-order reaction, with a high rate constant. Then the chlorinated intermediates further reacted with free chlorine, also following a second-order reaction. While free chlorine was exhausted, the chlorinated intermediates auto-decomposed slowly and follows a first order reaction. The chlorinated-intermediates can be present in the water for more than 6 hrs if the dosage of free chlorine is not sufficient to oxidize intermediates in the system. The intermediates could also be reversed back to BMAA under a reducing condition. The results of this study provide useful insight for understanding the degradation and detection of BMAA in chlorination process for drinking-water systems.

Keywords: Alzheimer’s disease, Chloramines, Chlorination, Cyanotoxin, Drinking Water