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Cyanobacterial blooms and the occurrence of the neurotoxin, beta-N-methylamino-L-alanine (BMAA), in South Florida aquatic food webs

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ABSTRACT

Recent studies demonstrate that most cyanobacteria produce the neurotoxin beta-N-methylamino-L-alanine (BMAA) and that it can biomagnify in at least one terrestrial food chain. BMAA has been implicated as a significant environmental risk in the development of neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and Amyotrophic Lateral Sclerosis (ALS). We examined several blooms of cyanobacteria in South Florida, and the BMAA content of resident animals, including species used as human food. A wide range of BMAA concentrations were found, ranging from below assay detection limits to approximately 7000 μg/g, a concentration associated with a potential long-term human health hazard.

1. Introduction

There is a general consensus that Harmful Algal Blooms (HABs) are increasing worldwide (Smayda, 1990; Hallegraeff, 1993; Anderson et al., 2002; Glibert et al., 2005). This is primarily the result of an increasing human population generating increasing nutrient runoff from fertilizer, animal waste, sewage, and soil erosion (Nixon, 1995; Richardson and Jorgensen, 1996; Moffat, 1998; Heisler et al., 2008; Anderson et al., 2002, 2008). The increased mobilization of nutrients has led to the eutrophication, first of small water bodies such as ponds, lakes and rivers (Vollenweider, 1992); then of estuaries such as Chesapeake Bay (Cooper and Brush, 1991; Harding and Perry, 1997); and more recently of large seas such as the Black Sea (Bodeanu, 1992; Mee, 1992; Cociasu et al., 1996), Baltic Sea (Larsson et al., 1985; Nehring, 1992), and Adriatic Sea (Vollenweider et al., 1992; Justic et al., 1995); and of continental shelf areas such as the Mississippi River delta (Turner and Rabalais, 1991, 1994; Justic et al., 1995).

Blooms of cyanobacteria appear to also be increasing worldwide in response to increased nutrient inputs (Chorus and Bartram, 1999; Paerl, 2008). Cyanobacteria have the ability to produce a wide array of secondary metabolites (Moore, 1996; Welker and von Dohren, 2006; Sivonen and Borner, 2008), many of which are noxious or toxic to animals and/or humans (Carmichael and Falconer, 1993; Chorus and Bartram, 1999; Carmichael, 2001; Carmichael et al., 2001; Ibelings et al., 2008; Falconer, 2008; Pilotto, 2008; Stewart et al., 2008). Many of these toxins are dermatoxins, hepatotoxins, or neurotoxins, but only a few species of cyanobacteria are known to produce each of these toxins. Recently Cox et al. (2005) have produced laboratory results that suggest that virtually all cyanobacteria species produce the neurotoxin beta-N-methylamino-L-alanine (BMAA). The data demonstrate a 500-fold variation in the amount of BMAA produced among the cyanobacteria species examined, but how much of this variation is genetic and how much is environmental–physiological is not known at the present time. Metcalf et al. (2008) and Esterhuizen and Downing (2008) have also presented data suggesting that most cyanobacteria produce BMAA in the environment.

In vitro studies have shown that BMAA is toxic to neurons at concentrations as low as 10–30 nM (Weiss and Choi, 1988; Weiss et al., 1989; Rao et al., 2006; Lobner et al., 2007). BMAA is structurally similar to glutamate and binds to glutamate receptors (Richter and Mena, 1989; Copani et al., 1990; Smith and Meldrum, 1990; Allen et al., 1995; Mash, 2008). Abnormal stimulation of these receptors may play a role in neurodegenerative diseases such as Alzheimer's disease, Parkinson's disease, and Amyotrophic Lateral Sclerosis (Takahashi et al., 1997). BMAA has also been shown to be neurotoxic in a variety of animal models (Spencer et al., 1987; Karamyan and Speth, 2008).

BMAA, as an amino acid, is water soluble, not lipid soluble, and thus has a low octanol–water partition coefficient (Yunger and Cramer, 1981). It therefore would not be expected to biomagnify

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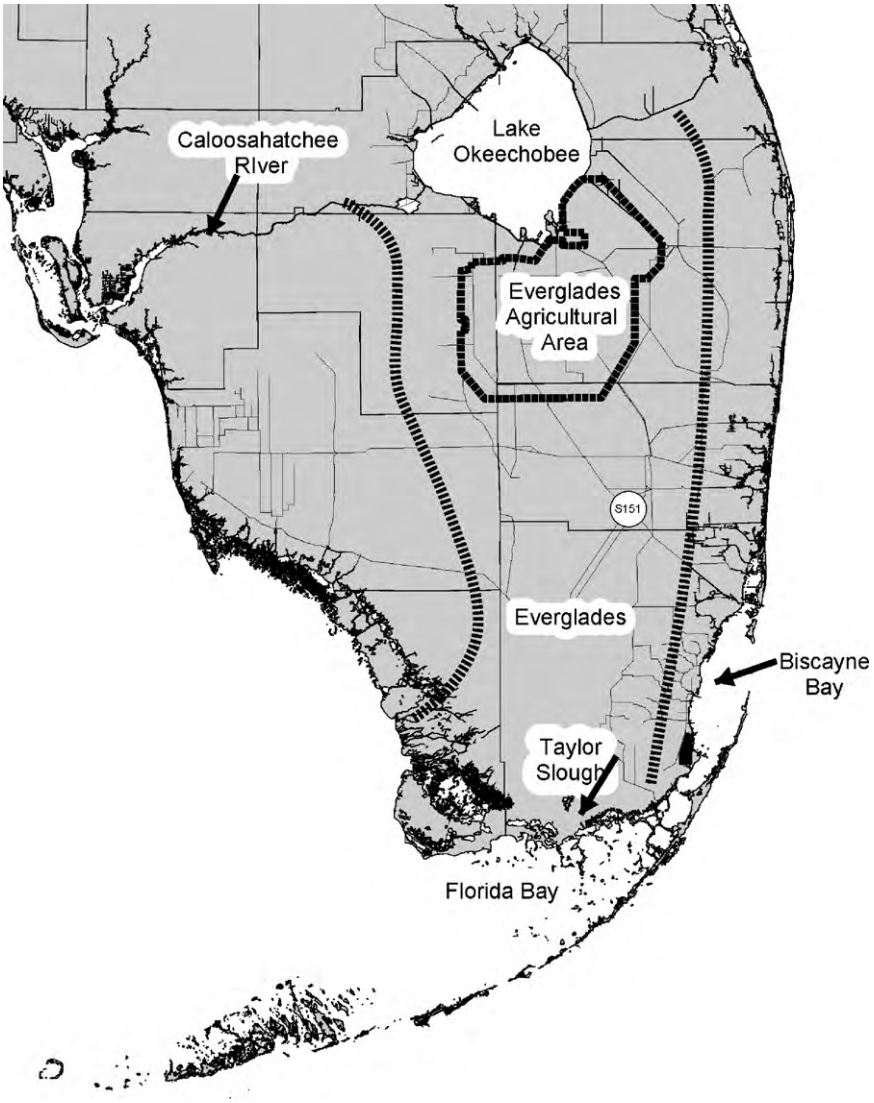

Fig. 1. Map of major water bodies in South Florida. Lines of parallel bars indicate general watershed boundaries of the Everglades.

up the food chain (Mackay, 1982; Connolly and Pedersen, 1988; Arnot and Gobas, 2006; Kelly et al., 2007). However, unlike the more moderate bioaccumulation observed with most water soluble cyanobacteria toxins (Xie et al., 2005; Ibelings and Chorus, 2007; Ibelings and Havens, 2008; Funari and Testai, 2008), Cox et al. (2003), Banack and Cox (2003), Murch et al. (2004a,b), and Banack et al. (2006) have demonstrated a 10,000-fold biomagnification of free BMAA and 50-fold biomagnification of total BMAA in a food chain in Guam from symbiotic cyanobacteria to cycads to fruit bats (Pteropids, also known as "flying foxes"). Cox and Sacks (2002) hypothesized that the increased consumption of these fruit bats by one ethnic group of people in Guam, the Chamorros, in the 1940s led to a 100-fold increase in the development of Amyotrophic Lateral Sclerosis (ALS)–Parkinsonism dementia complex in the Chamorros living in Guam. The observation of high concentrations of BMAA in the autopsied brains of Chamorros who died of these neurodegenerative diseases and the absence of BMAA in the age-matched non-neurological control brains of Canadians who died of other causes suggested a possible link between BMAA and neurodegenerative diseases (Murch et al., 2004a,b). Recently Pablo et al. (2009) have demonstrated high concentrations of BMAA in Americans who died of Alzheimer's disease or ALS, but little or no BMAA in the brains of age-matched non-neurological controls, or in cases of Huntington's disease, a genetic disorder. These data suggest that the unusual Guam situation is not unique, and that BMAA may biomagnify in other food chains, enter the human diet, and potentially trigger neurodegenerative disease.

Together, these recent studies suggest that BMAA from cyanobacteria could be involved in neurodegenerative diseases. As BMAA is non-lipophilic, it would not be expected to biomagnify, yet the indirect evidence suggests that it can biomagnify in at least certain circumstances. To determine if BMAA is present and can biomagnify in aquatic food chains, BMAA concentrations were analyzed in animals collected from water bodies in South Florida (Fig. 1) known to have blooms of cyanobacteria.

2. Methods

2.1. Sampling methods and sites

Water samples were collected on a roughly monthly basis at numerous stations in Florida Bay (Fig. 2), Biscayne Bay (Fig. 3), and the Caloosahatchee River (Fig. 4). Sample tissue to be analyzed for BMAA content were collected from various frozen animals that had been collected for a variety of other research projects in these water bodies.