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TOXIC *MICROCYSTIS* IN EUTROPHIC LAKES

Ho-Dong Park
Department of Environmental Science, Shinshu University,
Matsumoto 390, JAPAN

The mass development of planktonic cyanobacteria is one of the consequences of accelerating eutrophication in many freshwaters un the world including temperate and subtropical regions. These phenomena are thought to result from increased exogenous nutrient loadings, coupled with temperature and light conditions. Toxic cyanobacterial blooms in eutrophic lakes and reservoirs have been reported in many countries. [Skulberg et al., 1984; Carmichael, 1988; Gorham and Carmichael, 1988] These toxic waterblooms have caused death in livestock and wildlife besides cases of illness in humans. [Billings, 1981; Falconer, 1989] Even though these same toxic cyanobacteria are present in freshwater sources in Japan, the mortality rates of wild or domestic animals have not been reported so far fortunately. The lethal toxicity fo these waterblooms, however was found upon intraperitoneal injection of water extracts from the alga into mice. [Watanabe and Oishi, 1980] Despite the appearance of toxic cyanobacteria in many of the lakes, the research on seasonal variation of toxins is still scarce. [Wicks and Theil, 1990; Lindholm and Meriluoto, 1991; Watanabe et al., 1992; Park et al., 1993; Kotak et al., 1995]

Toxins from freshwater cyanobacteria are classified into two groups, hepatotoxins and neurotoxins. *Microcystis aeruginosa* is the most common toxic cyanobacterium found worldwide, and it produces potent cyclic hepatopeptide toxins, termed microcystins. [Carmichael, 1988, 1989; Ohtake et al., 1989; Carmichael et al., 1990; Harada et al., 1991b; Carmichael, 1992] Microcystins are also found in *Microcystis viridis* [Kusumi et al., 1987; Watanabe et al., 1988], *Anabaena flos-aquae* [Krishnamurthy et al., 1986], *Oscillatoria agardhii* [Meriluoto, et al., 1989], and *Nostoc* sp. [Sivonen, et al., 1990]. The chemical structures of the hepatotoxins contained in *M. aeruginosa* have been elucidated by Botes et al. [1984. 1985], as cyclic heptapeptides. The

toxins are composed of five common amino acids and a pair of L-amino acids as variants. The structural differences among the toxins are related to the remaining two L-amino acids. Recently, desmethyl derivatives have been reported [Harada et al., 1991b], in which methyl groups of *N*-methyldehydroalanine and *N*-methly aspartic acid are replaced by hydrogen atoms. Over 40 microcystins have been isolated. Furthermore, it has been found that microcystins and nodularin inhibit protein phosphatase activity, especially 1 and 2A, in a manner similar to that of okadaic acid. [Mackintosh et al., 1990; Matsushima et al., 1990; Yoshizawa et al., 1990] Microcystins-YR and -LR were purified from a Japanese strain of *Microcystis acruginosa* isolated from Lake Suwa by Kungswan et al., [1987] Thereafter three toxins (microcystins-RR, -YR and -LR) were detected in two strains of *M. aeruginosa* and four strains or *M. viridis* [Watanabe et al., 1988], plus natural samples of *Microcystis* species obtained from lakes in Japan [Watanabe et al., 1989a]

Seasonal change of the amount of toxic heptapeptide microcystin was investigated during the warm season of 1991 and 1994 in eutrophic lake, Lake Suwa. Lake water (ca. 5 liters) was divided between the *Microcystis* cells fraction and the filtrated lake water fraction, and these fractions were analyzed to estimation of the total quantity of microcystin in lake water with HPLC(High Performance Liquid Chromatography), respectively. The high amount of microcystin in cells was estimated during the exponential growth phase of the bloom. The highest concentration of microcystin was 124 μg/l on July 20, 1992. Otherwise, the amount of microcystin in the filtrated lake water was high at the end of the bloom. This amount was very small (under 7 μg/l) during investigation period. More than 20%, however, high percentage of microcystin in the filtrated lake water was showed at the end of bloom. This result suggested that release of microcystin from cells would occur during the decomposition process of *Microcystis* cells.