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## **A SURVEY OF 50 NH LAKES FOR MICROCYSTINS**

*Principal Investigators: Dr. James Haney, Miyoshi Ikawa*

*Descriptors: biotoxins, microcystins, Microcystis, cyanobacteria, algae, eutrophication, lakes, water quality*

### *Problem and Research Objectives:*

Cyanobacteria blooms pose a potential threat to the use of lakes for both recreation and drinking water supplies. There are increasing reports of health problems associated with toxic cyanobacteria such as *Microcystis* in many parts of the world. Preliminary investigations in New Hampshire lakes indicated the presence of the hepatotoxin microcystin in lakes of varying trophic status. The major objective of this study is to conduct a survey of New Hampshire's lakes to determine which lakes contain toxic cyanobacteria that produce microcystins and evaluate whether there is a direct relationship between the presence of the cyanobacteria toxins and the trophic condition of the lakes.

### *Principal Findings and Significance:*

Results from this study indicate the presence of microcystin toxins in all of the lakes examined. These findings shift the emphasis from asking, "which lakes have toxic cyanobacteria?" to "what controls the level of cyanobacteria toxins in lakes?" By measuring the MC levels in the lake water as well as the weight-specific concentrations in the plankton we were able to demonstrate that some lakes have very small amounts of plankton that are relatively toxic and similarly, some lakes with large quantities of plankton with low specific toxicity.

Although eutrophication has been linked with problems of toxic cyanobacteria, the focus of most previous studies has been on "problem" lakes that exhibit blooms of cyanobacteria. We have demonstrated that microcystin toxicity parallels the relationship between nutrients and phytoplankton biomass (chlorophyll a) and extends from ultra-oligotrophic to eutrophic lake conditions. This allows for a quantitative forecasting of the impact of nutrient enrichment on lake toxicity, which could be important for the management of surface water supplies for drinking water and recreation.

Because of the emphasis on phosphorus as a limiting factor for phytoplankton growth in lakes, we initially tested our microcystin-eutrophication hypothesis using total phosphorus as the driving nutrient. Surprisingly, nitrogen (total nitrogen) provided a better predictor of toxin concentration than phosphorus, suggesting future lake monitoring and research should also include testing for total nitrogen.

The NH microcystin survey also demonstrated that microcystin toxin concentrations are correlated with other parameters commonly measured in lake monitoring programs, such as chlorophyll a, Secchi disk depth and acid neutralizing capacity. This is a significant finding in that it indicates the results from lake monitoring surveys can be applied to predict the likelihood of toxicity problems in a lake.

Microcystins were detected in an extremely broad range of concentrations in the net phytoplankton and zooplankton of all the lakes tested. Overall, the zooplankton contained approximately 20% of the phytoplankton microcystin content, indicating considerable amount of this toxin are passing into the lake food web and possibly being bioaccumulated by other lake biota such as fish and benthic consumers. Some of the study lakes had high levels of microcystins in the zooplankton, compared to

other lakes with comparable nutrient levels, raising questions concerning the role of the composition of the lake food web in the transfer of toxins. The strong positive correlation between zooplankton MC and the % *Daphnia* in the lakes suggests the species composition of the zooplankton grazers may influence the transfer of microcystins. Likewise, it is likely that the degree of fish planktivory in the lake may indirectly impact the efficiency of movement of MC from the phytoplankton to the zooplankton grazer community. Clearly, investigations of microcystin transfer through lake food webs are needed to better understand these processes.

Our data support the model that, in general, nutrients promote the development of microcystin toxicity in lakes. The utility of these models, however, is limited by the high lake-to-lake variability. Other factors must clearly be included in future models to be useful for forecasting the effects of nutrient enrichment on lake toxicity. We have identified lake mean depth as an important variable, along with factors such as the buffering capacity or ANC of the water. Long-term studies should be undertaken on a subset of lakes to incorporate the influence of light and temperature. Such models would have greater predictive power for specific lakes and also permit long-range forecasting of the effects of global climate change on lake toxicity.