**Creating a Cyanotoxin Target List for the Unregulated Contaminant Monitoring Rule**
May 17-18, 2001
USEPA - Technical Service Center
26 W. Martin Luther King Dr.
Cincinnati, OH 45268

**Meeting Summary**

## Introduction

The Safe Drinking Water Act (SDWA), as amended in 1996, requires the Environmental Protection Agency (EPA) to select a group of known or potential drinking water contaminants every 5 years for regulatory consideration. EPA published its first list of contaminants in 1998. This list, known as the Contaminant Candidate List (CCL), contained many contaminants which lacked critical information (i.e., occurrence data) needed to make regulatory decisions. Such information can be provided by the Unregulated Contaminant Monitoring Regulation (UCMR) program if protocols and analytical methods have been developed and validated.

The 1998 CCL includes cyanobacteria and their toxins as one of the microbial contaminants selected, but it does not specify which toxins should be targeted for study. With over 80 variants of cyanotoxins reported, the scientists from the Office of Ground Water and Drinking Water must review the toxicological, epidemiological and occurrence studies and drinking water treatment literature.

The goal of this meeting was to convene a panel of scientists to assist in identifying a target list of algal biotoxins that are likely to pose a health risk in source and finished waters of the drinking water utilities in the United States. The panel (see Appendix A) consisted of researchers from the drinking water industry, academia, and government agencies with expertise in the area of fresh water algae and their toxins. Toxin selection were based on four criteria: health effects, occurrence in the United States, susceptibility to drinking water treatment, and toxin stability.

The EPA will review the target list and select a final list of toxins to be monitored under the UCMR. Information obtained on the occurrence of these toxins, through one or more UCMR surveys, will be used in making regulatory determinations for algal toxins in drinking water.

## Background
### CCL and UCMR

The 1996 SDWA Amendment requires the EPA to make regulatory decisions on at least 5 contaminants from the CCL every 5 years. Contaminants are listed on the CCL based on their public health significance, likelihood of occurrence in drinking water, waterborne route of transmission, treatment effectiveness, and if they are covered by other proposed or promulgated regulations. The 1998 CCL contains 10 microorganisms, including freshwater algae and their toxins. Regulatory decisions for the contaminants on the CCL require information on their health effects, treatment, and occurrence. Of the 10 CCL microorganisms, 8 were listed as needing more occurrence information. The UCMR, implemented by the 1996 SDWA, and promulgated in 1999, was designed to provide the necessary occurrence information.

The 1999 UCMR is divided into 3 monitoring options: Assessment Monitoring, Screening Survey, and Pre-Screen Testing. The current Assessment Monitoring stage contains unregulated chemical contaminants for which analytical methods were available at the time of promulgation. None of the 1998 CCL microorganisms had methods that could be used for assessment monitoring. All large systems (about 2800) and 800 small systems are required to

perform assessment monitoring for 1 year. The Screening Survey contains a list of chemical contaminants, as well as *Aeromonas*, for which analytical methods needed refinement and will be available at the start of monitoring in 2001 (*Aeromonas* is scheduled to be monitored starting in 2003). Only a select group of large and small water systems are required to conduct monitoring. The Pre-Screen portion of the 1999 UCMR contains the remaining 7 occurrence priority microorganisms from the 1998 CCL for which methods are currently under development. The Pre-Screen Testing will test new methods and target vulnerable water systems to gain information on contaminant occurrence in systems with the greatest likelihood of having detectable levels of the contaminant. This information will help evaluate methods and help design subsequent surveys, if needed. The algal toxins selected by the panel and EPA will be monitored under the UCMR Pre-Screen Testing component when standardized and validated methods are available. If significant occurrence of a toxin is found during Pre-Screen Testing, the contaminant could be monitored in a later screening survey or assessment monitoring.

**Algal Biotoxins**

Most of the world's population relies on surface freshwaters as its primary source for drinking water. The drinking water industry is constantly challenged with surface water contaminants that must be removed to protect public health. Recent reports suggest that toxic cyanobacterial blooms are an emerging issue in the United States because of increased source water nutrient pollution causing eutrophication. A third of the 50 freshwater cyanobacteria genera are capable of producing toxins. The mechanisms of algal biotoxin's toxicity are very diverse, ranging from hepatotoxic, neurotoxic, and dermatotoxic to general inhibition of protein synthesis. The most studied freshwater algal biotoxins are divided into three classes based on chemical structure: cyclic peptides, alkaloids, and lipopolysaccharides.

*Cyclic Polypeptides*

Nodularins and microcystins are cyclic peptides containing five and seven amino acids, respectively. Nodularins, with several structural variants, are found predominantly in marine and brackish water, which are not usually used as drinking water sources. Nodularins are produced by the genus *Nodularia*. Microcystins have been found in fresh water throughout the world. They are produced by *Microcystis*, *Anabaena*, *Oscillatoria*, *Nostoc*, *Anabaenopsis*, and terrestrial *Hapalosiphon*. To date, approximately 65 variants of microcystins have been isolated from cyanobacterial blooms and cultures. The most common variant is microcystin-LR. Other common microcystin variants include YR, RR, and LA.

*Alkaloid Toxins*

Alkaloids vary in their chemical structures and stabilities. Depending on the type, it can affect the nervous system, skin, liver, or gastrointestinal tract. Some of the most studied algal toxins belonging to the alkaloid class include: anatoxin-a, anatoxin-a(s), homo-anatoxin-a, saxitoxin, and cylindrospermopsin.

The anatoxins (anatoxin-a, anatoxin-a(s), and homo-anatoxin-a) and saxitoxin are neurotoxic. Anatoxins are low molecular weight, secondary amines. They are produced by *Anabaena*, *Oscillatoria* (*Planktothrix*), *Aphanizomenon*, and *Cylindrospermum*. Anatoxin-a is a nicotinic agonist that binds to nicotinic acetylcholine receptors. Homoanatoxin–a is a potent neuromuscular blocking agent which causes severe paralysis, convulsions and death by respiratory arrest. Anatoxin–a(s) is an organophosphate produced by *Anabaena*. This toxin blocks acetylcholinesterase activity in a manner similar to organophosphate insecticides.

Saxitoxins and their analogues are produced by *Anabaena* and *Aphanizomenon*. Saxitoxins are the cause of paralytic shellfish poisonings (PSP) in people. This toxin acts on the

sodium ion channels of the excitable membranes of the nerves, which leads to paralysis, respiratory depression and respiratory failure. However, to date, there has been no PSP-like illnesses reported in humans from the consumption of drinking water.

Cylindrospermopsin is a hepatotoxic alkaloid that has been isolated from cultures of *Cylindrospermum raciborskii*. Cylindrospermopsin producing species are found in tropical/subtropical climates. This toxin was identified in an outbreak of acute hepato-enteritis and renal damage among an aboriginal population in Australia.

*Lipopolysacharide Toxins (Endotoxins)*

Cyanobacterial lipopolysaccharides (LPS) are cell wall components of gram-negative bacteria. The toxin exhibits both pyrogenic and toxic effects (for additional information, see Weckesser and Drews, 1979). The few studies that have been performed on cyanobacterial LPSs suggest that they are less toxic than other bacterial LPSs, such as those produced by *Salmonella*. Lack of axenic cyanobacterial cultures has limited the progress of research on the structures and toxicity of cyanobacterial LPSs. Circumstantial evidence has implicated cyanobacterial LPSs as the cause of an outbreak in Sewickley, Pennsylvania.

*Other Toxins*

Other less common and studied algal toxins also have been described in locations outside the United States or in non-freshwater habitats. The possibility exists for these toxins to be detected in U.S. freshwaters in the future due to increased salinity of freshwater supplies. This increased salinity is partly the result of salt-water intrusion and salt contamination caused by the mining and commercial industry. The following are some of the lesser known toxins.

*Lyngbyatoxin*

Lyngbyatoxins, a contact irritant, are mainly marine water toxins but their occurrence in freshwaters has not been fully assessed. *Lyngbya*, an aggressive genus capable of widespread growth, has been found in fresh water supplies in the United States. More data is needed to determine its health effects and occurrence in freshwater supplies.

*Aplysiatoxin*

Aplysiatoxins have low occurrence with evidence suggesting a link to *Lyngbya* blooms. They are marine toxins and contact promotes skin tumors. Further evaluation (i.e., ecological studies) is needed in freshwaters.

*Debromoaplysiatoxin*

Debromoaplysiatoxins are similar to aplysiatoxins in that they have low occurrence in mostly marine waters and contact could promote skin tumors. More research is needed to determine the toxin's occurrence in freshwater.

*Prymnesin*

Prymnesins are produced by eukaryotic algae and are not likely a drinking water issue because they are found mostly in brackish and marine waters. Prymnesin could potentially be of concern in Texas (for example) because of salt-water infusion. Prymnesins are haemolytic cytotoxins and have been implicated in fish kills. Coastal problems need to continue to be tracked.

*Domoic acid*

Diatoms produce Domoic acid. It has been reported to occur in Lake Victoria in Africa.

*Pfiesteria*

Marine and brackish water dioflagellate produce *Pfiesteria* toxins. Investigators have reported a *Pfiesteria* outbreak on the East Coast of the United States.

**Health Effects**

Primary public exposure to algal biotoxins can come from drinking water, recreational water, and dietary supplements. Secondary human exposure may come from algal biotoxin residue on fruit and vegetables from contaminated irrigation water and consumption of animal tissue. As with most drinking water contaminants, children and immunocompromised people are likely at greatest risk of harmful toxin health effects. Lack of markers for toxins have hindered the understanding of algal toxin health effects. Symptoms may be missed, mild cases may be mis-diagnosed, or the causative toxin may be unidentified. The effect of episodic exposure is undetermined.

Microcystins' and nodularins' primary target is the liver. Many of the microcystin structural variants and nodularin are highly toxic with intra-peritoneal mouse LD50s between 50-200 µg/Kg body weight. Microcystins and nodularins inhibit protein phosphatase 1 and 2A. Substances that inhibit protein phosphatase enzymes are considered to be tumor promoters in the liver. Animal and epidemiological studies suggest that low-level chronic exposure to microcystins increase human health risk of carcinogenesis and tumor growth promotion of the liver. Microcystin is retained in the body longer than other algal toxins; intact microcystins have been found in exhumed bodies. The World Health Organization (WHO) guideline of 1 µg/L for microcystin are based on an adult's typical daily intake of water, the individual's body weight, and the toxicity.

Cylindrospermopsin's primary target is the liver, although recent studies have also found it to be carcinogenic and genotoxic. Cylindrospermopsin inhibits glutathione and protein synthesis. In animal studies, the effects of this toxin have been widespread and progressive tissue injury, with cell necrosis in the liver, kidneys, adrenals, lung, heart, spleen and thymus. Nonetheless, cylindrospermopsin is not retained in the body longer than other algal toxins. Cylindrospermopsin has recently been nominated for assessment by the National Toxicology Program.

Anatoxin-a is a cholinergic agonist that binds to neuronal nicotinic acetylcholine receptors. A high dose of anatoxin-a leads to paralysis, asphyxiation, and death. The intra-peritoneal mouse LD50 is 375 µg/Kg body weight. The oral acute toxicity LD50 is greater than 5,000 µg/Kg body weight. Sub-acute toxicity was not reported when anatoxin-a was administered to rats orally in their drinking water at 510 µg/L or 5,100 µg/L suggesting that significant concern with chronic toxicity is unlikely.

Saxitoxin and neosaxitoxin block the inward flow of sodium ions across membrane channels of nerve cells that stimulate muscle cells. The inhibition of impulses in nerve axons by this effect can lead to paralysis of muscles. Only acute effects have been observed. The intra-peritoneal mouse LD50 is 10 µg/Kg, and the oral mouse LD50 is 263 µg/Kg body weight. Neonates are 10 times more sensitive to the effects of saxitoxin and neosaxitoxin than adults, warranting further sensitive subpopulation information collection under the 1996 Safe Drinking Water Act amendments.

Other toxins may have a variety of effects including skin irritation, allergic reactions or neurotoxic effects. However, these toxins have little to no occurrence information available in the United States.

The following are some domestic and international guidelines based on health risk for a select group of cyanobacterial toxins:

Anatoxin-a:
- No official guideline in the U.S.
- Australia: 3 µg/L (suggested)

Saxitoxin:
- No official guideline in the U.S.
- Australia: 3 µg/L (suggested)

Microcystin:
- No official guideline in the U.S.
- World Health Organization: 1 µg/L
- Brazil: 1 µg/L (Regulatory Level)
- Australia: 10 µg/L or 1.3 µg/L
- New Zealand: 1 µg/L
- Canada: 1.5 µg/L
- Oregon: 1 µg/g (health food)

Cylindrospermopsin:
- No official guideline in the U.S.
- Australia: 1 - 15 µg/L

**Occurrence**

Several state algal toxin occurrence studies have been performed in United States and Canada.

A recent microcystin study, conducted in the United States and Canada, surveyed 24 public water systems from June 1996 to January 1998. Using ELISA and protein phosphatase assays, 80% of the 677 samples tested positive. Of the positive samples, 4.3% had detections higher than the WHO drinking water guideline of 1 µg/L. Studies of Lakes Onondaga, Oneida, and Champlain in New York have also detected microcystins. The Lake Champlain study found microcystin in 17% of the samples. In Wisconsin, microcystin was detected above 1 µg/L in raw, but not finished water. A survey of an Ohio River *Microcystis* bloom resulted in no detect of microcystins by both ELISA and LC/MS. A Florida survey found microcystin concentrations ranging from 0.1 µg/L to 12 µg/L. Microcystin-LR, the most common variant, is not an unequivocal indicator for the presence of toxins other than microcystin. Collectively, surveys of U.S. waters have detected microcystin, anatoxin-a, and cylindrospermopsin above guideline or proposed guideline values in raw and finished water.

In Canada, HABs occur in all provinces, with the greatest likelihood in shallow and slow-moving bodies of water. In the 1990's, microcystins were detected in multiple lakes, some of which are drinking water sources. Microcystins were also found in Winnepeg's distribution system at 0.45 µg/L (raw water samples) and 0.55 µg/L (tap water samples). Microcystin-LR is the most common hepatotoxin in Canadian water supplies while *Anabaena, Aphanizomenon, Microcystis,* and *Phormidium* have been found to be the most common algal species.

*Cylindrospermopsis* is an emerging and expanding tropic/subtropical genus. It has been observed in the waters of many mid-Atlantic states (Virginia, North Carolina) as well as in Kansas, Oklahoma, and Florida. Florida has detected as much as 90 µg/L of cylindrospermopsin in finished water. Prevalence information is limited because of the lack of chemical standards and methods. The reason behind *Cylindrospermopsis'* sudden emergence is unknown, but recent climatic, environmental, and human habitation changes may be a factor.

The Lake Champlain study detected anatoxin-a in 4% of the samples taken in the summer of 2000. This, along with the symptoms of toxicity, suggest that anatoxin-a was responsible for the deaths of several dogs in the summer of 1999 and 2000. Lake Champlain is used as a drinking water source. In Florida, concentrations as high as 8 µg/L of anatoxin-a has been detected in finished water.

Anatoxin-a(s) occurrence is lower than that of anatoxin-a and saxitoxin. It is a structurally unique toxin because it contains an organophosphate, which may cause long-term health effects. There are currently no standards available for the study of anatoxin-a(s). If ozonation is used as a method of treatment, harmless organo-phosphates could potentially become more toxic.

Saxitoxin occurrence in the waters of the United States is moderate to rare. The samples from Lake Champlain did not contain saxitoxins, however saxitoxins have been reported in New Hampshire, Florida, and Alabama.

Some cyanobacterial toxins and their areas of known occurrence are listed below.

Microcystin:
- United States
- Australia
- Canada
- United Kingdom
- Europe
- China

Anatoxin-a:
- United States
- Canada
- Europe
- China

Anatoxin-a(s):
- United States
- Canada
- Europe
- Anatoxin-a(s) is being detected with greater frequency over time.

Saxitoxin:
- United States
- Australia
- Brazil
- Europe (Portugal)

Cylindrospermopsin:

- United States (Florida, Kansas, North Carolina, Virginia, Oklahoma)
- Australia
- Brazil
- Japan
- Israel
- Europe

Lyngbyatoxin:

- United States (Hawaii)
- Australia
- Florida
- Pacific Islands

Lyngbyatoxins are predominantly found in marine bodies of water.

The frequency and duration of Harmful Algal Blooms (HABs) have been on the rise. Most occurrences of cyanobacterial toxins are caused by poor water quality (i.e., nutrient over-enrichment). Urban, agricultural, and industrial expansion have all contributed to such water quality deterioration. An effective way to control HABs is to manage the nitrogen/phosphate ratio. The management of both nutrients, rather than just one, is a more effective long-term solution. Other options are to manipulate water residence time, temperature, irradiation, and to mix waters to create undesirable growing conditions. The observed changes in occurrence patterns of algae and toxins suggest that the final target list needs to be flexible to allow new toxins to be added in response to invasions of new species of algae in the United States.

**Algae and Algal Toxin Treatment**

Treatment options to remove cyanobacteria and toxins are:

- Enhanced Coagulation
- Ballasted Flocculation
- Conventional Treatment (Coagulation/Sedimentation)
- Slow Sand Filtration*
- Granular Activated Carbon (GAC)*
- Biologically Activated Carbon (BAC)*
- Filtration*
- Microfiltration*
- Non-chlorine Oxidants*

*These processes are more effective after coagulation/sedimentation processes.

Coagulation/sedimentation/filtration process has been reported to be between 90 and 99.9% successful at removing algae, but it is not effective at removing dissolved toxins. Physical removal of cells may be effective for toxins that tend to be retained in healthy cells such as microcystin, but would be less effective for toxins that are released by healthy cells such as cylindrospermopsin.

Treatment options for specific toxins:

Anatoxin-a and Anatoxin-a(s):

- Carbon (PAC, GAC) (adsorption)
- Ozone (anatoxin-a)
- Chlorine (anatoxin-a(s))

Saxitoxin:
- Carbon (PAC, GAC)
- Boiling
- Ozone
- Chlorine (less effective)

Cylindrospermopsin:
- Filtration
- Carbon (PAC, GAC)
- Chlorine
- Ozone

Microcystin:
- Chlorine
- Filtration
- Ozone
- Carbon (PAC, GAC)

Nodularin:
- Chlorine
- Filtration
- Ozone
- Carbon (PAC, GAC)

Activated carbon will remove biotoxins but its effectiveness is site-dependent because the amount of dissolved organic carbon in the source water will affect its efficiency. The more dissolved organic carbon in the water, the less efficient GAC will perform. While chemical oxidation may inactivate the biotoxins, studies need to be conducted to ensure that more potent toxins aren't being created. It is most likely that all algal biotoxins will be removed nano- and RO filtration. However, these processes are very expensive and the concentrated waste stream would be extremely toxic. The effectiveness of each treatment also depends heavily on the proper operation of the treatment plant.

**Stability of toxins**

The stability of toxins in natural and finished waters is a factor in their potential health risk. Microcystins are considered to be the most stable toxin in both natural and finished waters. Being a cyclic polypeptide, microcystin is resistant to heat, hydrolysis, and oxidation. Studies suggest that microcystin breaks down more rapidly in the presence of sunlight and dissolved organic carbon concentration between 2 and 16 mg/L. Anatoxin-a is more stable in the dark and degradation is accelerated by alkaline conditions. Anatoxin-a decomposes rapidly in high pH solutions. Saxitoxins and cylindrospermopsin are relatively stable but will breakdown in the presence of cell pigments. More specific information is needed on the stability of toxins in natural and finished waters. Since most of these toxins breakdown more slowly in the dark, this information would be particularly useful to states considering artificial recharge of aquifers.

**Analytical Methods**

Below is a list of analytical method types currently available for specific algal toxin detection, though many still need further validation studies.

Anatoxin-a:
- Bioassay
- HPLC
- LC/MS
- GC-ECD
- GC/MS

Anatoxin-a(s):
- Mouse bioassay
- Enzyme assay
- LC/MS
- HPLC

Saxitoxin:
- Mouse bioassay
- Immunoassay
- Cell receptor assay
- HPLC-FL
- LC/MS

Cylindrospermopsin:
- Bioassay (difficult to perform and interpret)
- HPLC-UV
- LC/MS

Microcystin:
- Immunoassay
- Bioassay
- Protein phosphatase assay
- HPLC
- LC/MS

Lyngbyatoxin and Aplysiatoxins:
- None available

There is currently no one universal analytical method for algal toxin detection. One reason is due to the structural diversity of the differing toxins and their variants. The lack of standards and low specificity of current assays need to be addressed in considering method development.

**Toxin Selection**

Based on the available information, the panel grouped the cyanobacterial toxins into four different priority categories.

Highest Priority:
- Microcystin
- Cylindrospermopsin
- Anatoxin-a

Medium to High Priority:
- Saxitoxin
- Anatoxin-a(s)

Further Study Needed:
- Nodularin
- Lyngbyatoxin
- Aplysiatoxin
- Debromoaplysiatoxin
- Prymnesin
- Domoic acid

Not a Drinking Water Issue At This Time:
- LPS Endotoxin

**Other Issues**

- Analytical standards are needed for all algal toxins, except saxitoxins which are already available through FDA.
- Epidemiological studies are needed for saxitoxins.
- Anatoxin-a, saxitoxins, and microcystins are commercially available but there is need for the other toxins to become commercially available.
- ELISA assays are needed for cylindrospermopsin and anatoxin-a. There is some international advancement on these kits. An Australian team is working on cylindrospermopsin ELISA kit and a Canadian team is working on anatoxin-a ELISA kit.
- Molecular probes are needed.
- Analytical methods need to be made into standard methods.
- Acute and chronic effects of algal toxicity need to be studied.
- Sampling should take place in raw water, finished water, and storage reservoirs.
- Nodularins and microcystins can be tested at the same time.
- Low and high level chronic biotoxin studies need to be performed.

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# Appendix A

## Creating a Cyanotoxin Target List for the UCMR
## May 17-18, 2001

### Participants

| Name | Contact Information |
|---|---|
| ***Meeting Leaders*** | |
| Jim Sinclair | USEPA<br>TSC (MS-140)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268<br>(513) 569-7970 |
| Judy Westrick | Northern Kentucky University<br>Dept. of Biological Sciences<br>Nunn Dr.<br>Highland Heights, KY 41099<br>(859) 572-5297<br>Effective 8/01:<br>Lake Superior State University<br>Assistant Professor of Chemistry<br>650 W. Easterday Ave., Crawford Hall<br>Sault Ste. Marie, MI 49783<br>(906) 635-2165 |
| ***Meeting Support*** | |
| George Hallberg | The Cadmus Group, Inc.<br>135 Beaver St.<br>Waltham, MA 02452<br>(781) 434-2537 |
| Ashton Koo | The Cadmus Group, Inc.<br>135 Beaver St.<br>Waltham, MA 02452<br>(781) 434-2503 |
| ***Presenters*** | |
| George Izaguirre | Water Quality Lab<br>MWDSC<br>700 Moreno Ave.<br>LaVerne, CA 91750<br>fax: 909-392-5246<br>909-392-5127<br>gizaguirre@mwd.dst.ca.us |

| Name | Contact Information |
|---|---|
| Wayne Carmichael | Department of Biological Sciences<br>Wright State University<br>Dayton, OH 45434<br>937-775-3173<br>wayne.carmichael@wright.edu |
| John Burns | CyanoLab<br>205 Veagler Dr.<br>Suite 302<br>Palatka, FL 32177<br>386-328-0882<br>cyanolab@bellsouth.net |
| Hans W. Paerl | Institute of Marine Sciences<br>University of North Carolina at Chapel Hill<br>3431 Arendell Street<br>Morehead City, NC 28557 USA<br>phone: (252) 726-6841, Ext. 133 fax: (252) 726-2426<br>hpaerl@email.unc.edu |
| Lorrie Backer | National Center for Environmental Health<br>Centers for Disease Control and Prevention<br>1600 Clifton Rd., NE<br>MS E-23<br>Atlanta, GA 30333 |
| Michele Giddings | Water Quality & Microbiology Division<br>Health Canada<br>5th Floor., MacDonald Building<br>123 Slater Street, A.L. 3505A<br>Ottawa, Ontario, Canada<br>K1A OK9<br>613-952-2594 |
| John Standridge | Wisconsin State Laboratory of Hygiene<br>2601 Agriculture Dr.<br>P.O. Box 7996<br>Madison, WI 53707-7996<br>jhs@mail.slh.wisc.edu<br>ph. 608-224-6209<br>fax. 608-224-6213 |
| Greg Boyer | SUNY-CESF<br>Syracuse, NY 13210<br>glboyer@esf.edu<br>315-470-6825 |
| Bob MacPhail | US EPA<br>MD-74B<br>US EPA Mailroom<br>Research Triangle Park, NC 27711<br>919-541-7833<br>macphail.robert@epa.gov |

| Name | Contact Information |
|---|---|
| ***Participants*** | |
| Carrie Moulton | Colorado State University<br>Biology Dept<br>Ft. Collins, CO 80525<br>970-377-2370. |
| Colleen McDermott | Dept. Biology and Microbiology<br>University of Wisconsin-Oshkosh<br>Oshkosh, WI 54901<br>(920)424-7090<br>(920)424-1101 (FAX)<br>mcdermot@uwosh.edu |
| Betsy Hilborn | US EPA/NHEERL<br>Human Studies Facility<br>104 Mason Farm Road, Room 350<br>Chapel Hill, NC 27599<br>(919) 966-0658<br>(919) 966-0655 fax<br>Hilborn.E@epamail.epa.gov |
| Donna N Myers | USGS<br>6480 Doubletree Ave<br>Columbus, OH 43229<br>dnmyers@usgs.gov<br>614-430-7715 |
| Misha Hasan | AWWARF<br>6666 W. Quincy Ave.<br>Denver, CO 80235<br>(303) 734-3425<br>mhasan@awwarf.com<br>(303)347-6122 |
| Bruce Mintz | EPA ORD/NERL (MD-75)<br>Catawba Bldg., Rm. 208<br>3210 Hwy 54<br>RTP, NC 27709<br>919-541-0272<br>919-541-0445 (fax)<br>mintz.bruce@epa.gov |
| Richard Eaton | USEPA<br>Gulf Ecology Division<br>1 Sabine Island Dr.<br>Gulf Breeze, FL 32561<br>850-934-9345 |

| Name | Contact Information |
|---|---|
| Dr. Kathy Echols | U.S. Geological Survey<br>Columbia Environmental Research Center<br>4200 New Haven Road<br>Columbia, MO 65201<br>573/875.5399 X1838 |
| Dave Munch | US EPA<br>TSC (MS-140)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268 |
| Greg Carroll | US EPA<br>TSC (MS-140)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268 |
| Ron Stetler | US EPA<br>NERL (MS-320)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268<br>stetler.ron@epa.gov<br>513-569-7928 |
| Mila Maizels | US EPA<br>NERL (MS-564)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268<br>(513) 569-7814<br>maizels.mila@epamail.epa.gov |
| Miriam Steinitz-Kannan | Northern Kentucky University<br>Dept. of Biological Sciences<br>Nunn Dr.<br>Highland Heights, KY 41099 |
| Armah A. del la Cruz | US EPA<br>NERL (MS-314)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268-1316<br>(513) 569-7224<br>fax: (513) 569-7170 |
| Susan Cormier | US EPA<br>NERL (MS-642)<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268 |
| Michael C. Miller | Department of Biological Sciences<br>University of Cincinnati<br>Cincinnati, OH 45221-0006<br>(513)556-9751<br>millermc@email.uc.edu |

| Name | Contact Information |
|---|---|
| Steve Vesper | US EPA<br>NERL<br>26 West Martin Luther King Drive<br>Cincinnati, OH 45268 |
| Bridgett Steele | Northern Kentucky University<br>steeleb@nku.edu |
| Barry Pepich | IT Corp<br>pepich.barry@epa.gov |
| Margie Bassett | IT Corp<br>bassett.margie@epa.gov |