DISCUSSION QUESTIONS

Prior to the workshop, the CALFED Science Program provided a list of key references and a background paper that surveyed the key literature of ammonia/ammonium in the Sacramento-San Joaquin Delta and Suisun Bay. Workshop participants were asked to submit discussion questions to help guide the facilitated discussion. Workshop participants were asked to submit questions into three general topic areas of 1) sources, fate and transport, 2) food web interactions, and 3) ecotoxicology.

Sources, Fate and Transport

Sources

What are the relative contributions from the sources spatially through the Delta?

What is the likely cause of increased ammonium concentrations in the estuary? Urban wastewater? Some other source? Is there a way to determine whether the source is urban through isotope analysis?

How important is it to characterize atmospheric deposition in west coast estuaries?

What proportion of ammonia in other parts of the Delta-Estuary is from the SRWTP discharge?

What are the relative contributions of non-WWTP sources of ammonia/ammonium to the Delta (e.g., agricultural runoff and excretion from invasive bivalves)?

Do we know how much ammonium is coming into the water column during lower flow months from clam excretion (both Corbula and Corbicula) and the sediments?

Fate

What is the fate of ammonia discharged by the SRWTP at Freeport in terms of concentrations and transformations downstream?

Has the in-situ regeneration rate of ammonium been considered as a supply? Has any one measured in-situ regeneration rates of ammonium in the water column?

How important is it to characterize internal cycling of ammonium from sediment sources in a system like the Delta? In what types of areas is internal cycling likely to play a larger role (i.e. bays, small channels, large rivers, floodplains, etc…).

How well are we examining the dynamics of phytoplankton growth in the waterways from the Lagrangian perspective? Do we know the travel times for phytoplankton in different waterways and seasons and water years? For example: Consider three points in a river A(upstream)→B→C(downstream). Water quality at point "B" reflects phytoplankton and bacterial processing of nutrients between point A and B. And

phytoplankton response to conditions at point B will be reflected by phytoplankton composition and biomass as point C.

Food Web Interactions

Nutrient and Food Web Interactions

In your opinions, what data or evaluations would most conclusively address the hypothesis that ammonia discharges are decreasing phytoplankton productivity in Suisun Bay?

What is the relationship between ammonia and *Microcystis*?

When (at what times of the year) and where does ammonium significantly inhibit algae productivity?

In a nutrient rich system such as the Delta, are ammonium levels likely to influence phytoplankton community composition? Have ammonium levels been linked to harmful algal blooms in other estuaries? If so, how do those ammonium levels compare to levels that occur in the Delta?

Has ammonium been known to cause changes in food web species from those that are high quality food sources to those that are low quality (low energy) or toxic food sources? How would a study be designed to measure the effect of ammonia on base level speciation?

How do ammonia/ammonium concentrations in this estuary compare to those in other estuaries? Have other estuaries documented ecosystem effects? If so, what effects were documented and how did they go about documenting them?

Have reductions in ammonium concentrations in other water bodies led to improvements in ecosystem function.

How do ammonia levels, food and fish abundance throughout the delta and tributaries relate?

What data is available for food abundance and ammonia upstream or in other areas of the Delta? Upstream data appears limited, except for the San Joaquin upstream of Stockton.

Are there specific times of the year when primary food production is low, and how does this relate to ammonia levels? Where is this occurring spatially in the Delta?

Why does the SJR inhibit algae growth in the Dugdale experiments when nitrogen in this river is dominated by nitrate from the Stockton WWTP?

Why does the Dugdale Lab find inhibition of algae growth in grow-out experiments using Sacramento River water from Rio Vista when samples from upstream (at Hood) where ammonium is higher do not show this effect?

Are there periods of the year when ammonium inhibition of diatom production is likely to be the dominant limitation on diatom production? [That is, during the February through May or June period when clam abundance is limited, is the reduction in diatom production likely attributable to ammonium?]

Are areas of the estuary with reduced clam densities more likely to be limited in diatom production by ammonium inhibition (e.g., the confluence)?

Can we discuss the significance of Kimmerer's (2005) work that clam grazing is an important diatom-specific loss factor in the low salinity zone (LSZ)? How well are we partitioning "blame" for diatom losses between clams, ammonia, and other potential loss factors – such as increased settling in deep channels?

Are the panelists aware of studies documenting ammonium-inhibition of phytoplankton blooms in freshwater systems anywhere else in the world?

Nutrient Dynamics

What are the right levels of nutrients (e.g., ammonia) for the Sacramento River and Sacramento-San Joaquin Delta and Suisun Bay?

How would you describe appropriate levels and balance of nutrients in the Bay-Delta estuary to support a healthy ecosystem? If there is not enough information available currently to describe desirable nutrient levels, what studies are needed to determine desirable nutrient levels?"

Has an ideal range of ammonia needed to support the ecosystem been determined spatially in the Delta?

Why aren't VanNieuwenhuyse's (2007) results suggesting that P supplies are limiting phytoplankton growth getting more traction? Why do we assume that N supplies, per se, govern phytoplankton dynamics, but not N:P ratios and N:Si ratios?

The nitrogen to phosphorus ratio of the Delta is relatively low (about 6). Do the panelists believe that low N:P ratios generally favor blue-green algal growth and if so, could reducing the ammonium-nitrogen output from the Sacramento wastewater treatment plant therefore lead to an increase in blue-green algal blooms downstream?

If treatment plants that currently discharge large ammonia loads were to upgrade to systems that discharge large nitrate loads instead (i.e. not remove the nitrogen, only change its form), what are the likely ecosystem responses? If they were to reduce the total nitrogen load, is it likely that the system would become nitrogen limited (i.e. how much

nitrogen is enough, how much is too much)? Would a low productivity aquatic environment benefit from removing as much ammonia from the system as possible?

Would a cross-systems approach that compares ammonium and chlorophyll concentrations among many systems be useful in evaluating what role if any ammonium concentration may play in determining the trophic state of the Delta?

Food Web Dynamics

What has been the nature of change in species composition of the Delta phytoplankton community in the past decade?

What is the trend in diatom, green algae, flagellates, and cyanobacteria percentages in the Delta phytoplankton community over time?

What has been the nature of change in species composition of the Delta phytoplankton community (e.g., diatom, green algae, flagellates, and cyanobacteria percentages) in the past decade? And, how do these community composition changes vary with respect to location (e.g., Suisun Bay, the upper estuary, and lower Sacramento River locations)?

Need an updated assessment of phytoplankton community composition. Biomass is a general indicator of zooplankton and larval fish food availability; however, species composition of the phytoplankton community is very important as well.

The Dugdale Lab experiments growing algae in cubitainers (mesocosms grow-outs) provide useful information for understanding environmental processes, but are they representative of what is occurring in the environment? For example, grow-outs in the Sacramento River in June (2008) showed greater algae growth at Hood than at Garcia Bend, but ambient samples showed more algae at Garcia Bend than at Hood.

In the recent Dugdale Lab study on the lower Sacramento River (Progress Report distributed to the POD-CWT, Nov-2008), what is the cause of the linear decrease in chl-a (ug/L) from the most upstream site (Hwy 80 bridge – RM 60) to Hood (RM 38)?

Have changes in Delta outflow exacerbated freshwater phytoplankton losses in the low salinity zone (LSZ) from lysing and other salinity-related mortality or morbidity? How do blooms in the LSZ relate temporally and geographically to the transition from freshwater to marine phytoplankton that occurs in the eastern SFE?

Monitoring results for chl.a from Sacramento River reaches during the last 10 years show that during March-May, chlorophyll-a is higher in the reaches downstream from the SRWTP than at Freeport (figure below is one of my discussion slides). These data do not suggest that the river becomes more hostile to phytoplankton growth below Freeport. Instead, they suggest that the river gains chlorophyll-a between Freeport and Suisun Bay. *How do we reconcile these results with Dugdale's group's grow outs at Rio Vista? Is something happening in the river to promote phytoplankton growth that does not happen in the grow outs?* (See Figures 1 and 2)

Despite rising ammonia concentrations in the Sacramento River downstream from the SRWTP, chlorophyll-a levels have been rising over the most recent decade, compared to the preceding decades. This is true generally speaking for the full seasonal cycle, and also true if one examines individual months hypothesized to be important for blooms (e.g. April –see below). *How do we reconcile these data with hypothesized ammonium inhibition in the river? Is something else other than ammonia determining long term patterns of phytoplankton biomass in transport in the River?* (See Figures 1, 3, 4, and 5)

Ecotoxicology

General

If ammonia inhibition is found to be occurring, what is the level of significance?

Given the growing body of research on sublethal and chronic effects of ammonia and on effects of ammonia in combination with other contaminants and stressors, are EPA acute and chronic criteria adequately protective? If not, on what scientific findings or research would you set new criteria?

Have you seen evidence that site specific criteria for ammonia/ammonium would be appropriate?

Are aquatic species more susceptible to ammonia toxicity if they are stressed by food limitation? How stressed do they need to be to become more susceptible? How could a study be designed to measure increased susceptibility resulting from stress, like that caused by food limitation? Are there other stressors other than food limitation that could increase the susceptibility of an aquatic species to ammonia toxicity?

Does ammonia toxicity to Delta smelt change notably when fish activity level increases or food intake decreases?

What methods are available for measuring effects of life time exposures to low levels of ammonia?

Could continuous exposure to moderate levels of ammonia for many generations have a deleterious impact on a species?

If 8 times ambient ammonia concentrations, and 16 times ambient effluent concentrations (from SRWTP) do not adversely affect juvenile delta smelt survival, then why is the CVRWQCB determined to repeat these acute bioassays when other, more pressing questions regarding stressors in sensitive delta smelt spawning and rearing habitats are not being studied?

Regarding statement on p.7 of the overview report: “Un-ionized ammonia concentrations in the Delta do exceed levels where histopathological effects have been observed (US EPA 1999); however, it is unclear whether these effects translate to effects on survival, growth or reproduction. In addition, there is some evidence that actively swimming and

unfed fish may be several times more sensitive to ambient un-ionized ammonia levels than these laboratory exposures indicate (Eddy 2005)." How widespread is this? Are these large areas or small areas?

On pg. 8 of the overview report – If existing water quality criteria for ammonia or new toxicity levels are exceeded – for how long and for what extent? (Criteria are developed based on exposure periods).

Can the panelists conceive of a scenario in which a gradual increase in ammonium concentrations immediately downstream from the Sacramento wastewater plant could cause a simultaneous, system-wide step-decline in the abundance of four zooplanktivorous fish populations?

What is the current understanding of the relationship between ammonia and zooplankton? Oft cited reports (e.g., Teh et al. 2008 and Werner at al. 2008) have valuable information about ambient conditions, but have not definitively identified ammonia as a toxicity driver.

Molluscs

What is the acute and chronic toxicity of un-ionized ammonia to the early life stages of mollusks that reside in the Delta and Suisin Bay?

To my knowledge, there is very little known on the mechanism of action of ammonia on mollusks. A critical piece of missing information is why are mollusks so susceptible to un-ionized ammonia concentrations?

We have identified very few biomarkers of exposure or effect in mollusks--most studies deal with lethality and a few with growth. What are the sublethal effects of ammonia on mollusks in the Delta?

Similarly, when you experience drought-like conditions (high temperature, low flows), you have the potential for higher ammonia toxicity and these occurrences of "episodic toxicity" may be adversely affecting mollusk populations.

pH and Temperature relationshipsGiven that ammonia concentrations are regulated by pH and temperature (+ salinity in marine systems), are there locations in your system that might typically have higher temperatures and pH that might shift more of the ammonia into the more toxic, un-ionized phase?

How does the panel recommend calculating effective unionized ammonia levels for evaluating the potential for in-stream toxicity in fluctuating temperature and pH environments?

Should pH monitoring be reinstated at the DWR Delta stations so that (1) ammonia/ammonium speciation can be estimated and (2) levels can be compared to water quality standards?

Comments

Some colleague and I have been looking at the effects of un-ionized ammonia on native freshwater mussels for the past 10 years or so. Collectively, we have found that their early life stages (e.g., juveniles) are some of the most sensitive organisms/life stage every tested. In fact, most of the acute and chronic LC50 values for juvenile mussels are well below the U.S. EPA water quality criterion. We have also seen significant reductions in shell growth over both acute and chronic exposures.

Many of the studies on ammonia and native mussels have been published in the peer-reviewed literature. The first series of papers came out in 2003 in the Environmental Toxicology & Chemistry (volume 22); a second series of papers came out in 2007 in the same journal (volume 26).

Unionized ammonia concentrations are a function of the total ammonia concentration, temperature and pH. Most laboratory ammonia toxicity testing and the U.S. EPA criteria document report fish and invertebrate no and low effect ammonia concentrations as a function of static unionized ammonia levels. Yet pH and temperature change both diurnally and seasonally in many natural systems. This is likely to be particularly important in evaluating the potential for chronic toxicity over a 30 to 60 day time period.

Miscellaneous

How concerned are the panelists about scaling-up the results of micro- and meso-cosm-scale experiments to predict the behavior of whole-systems?

What would be the impact on the estuary if ammonium levels were to double again in the next few decades?

How is the information regarding food and contaminants being pulled together and are there specific questions that this effort (Mike Johnson-UCD) is trying to answer.

Why is so much effort being spent on researching ammonia when relative to the POD other stressors, such as invasive species, chemical stressors, and flows, are known to be a problem, and receive little funding or attention?

What is the level of effort being made for other stressors?

How is the information regarding food, contaminants and invasive species being pulled together?

Is work contemplated to increase the reliability of testing (i.e. higher R^2 factors) with POD or surrogate species?

Questions Related to Silicofluoride and/or Chloramine

Since Sacramento County had started so called ‘fluoridation’ since Sac County voted in year 2000 for it; question is, how many TONS of Silicofluoride, and possibly chloramine

is discharged to the Sacramento River, annually?

Considering there are around 57 ‘registered’ Waste Water Treatment Plants, or around 30 cities surrounding the SF-Bay discharging waste water to the SF-Bay; has a study been done to determine if each WWTP is accomplishing 100% DECHLORAMIZATION [e.g. removal of Ammonia] before pumping waste water in the SF-Bay?

Are there now ‘waivers’ reluctantly allowing residual Chloramine to be discharged to the SF-Bay?

What studies have been done by SFPUC-Water Dept. to determine if Chloramine and Silicofluoride drinking/tap water treatment has increased the LEAD intake of school Children from school drinking fountains? This has been a major problem for schools in Washington DC, and Seattle, specifically.

Since Silicofluoride [EPA ‘regulated pollutant’ classified by ATSDR as ‘Hazardous Waste’] used to treat Sacramento & San Francisco plus 29 other cities drinking/tap waters surrounding the SF-Bay drinking waters with it’s inherent ‘trace toxics’ [e.g. Arsenic, Lead, radionuclide’s, …has anyone in California government studied whether residuals of Silicofluoride and/or Chloramine discharged by WWTP’s into our SF-Bay and incoming Rivers affected our SF-Bay and San Joaquin Delta.

Suggestion: In conjunction with CA Fish & Wildlife, hire SF-Bay Scientists at USGS located in Menlo Park to do studies: A ‘material balance study’ to determine the Annual TOXIC loading of residual Chloramine & Silicofluoride ‘toxics’ by 57 WWTP’s around SF-Bay, and Sacramento River. Find out how much TONNAGE in these chemicals are purchased per year, how much is used by the water treatment plants annually.

In field sample in SF-Bay estuary and Sacramento River WWTP discharge points for Ammonia ‘water & vegetation evidence’, and fish collapse evidence. Report to be issued by USGS with meaning scientific conclusions!

FIGURES

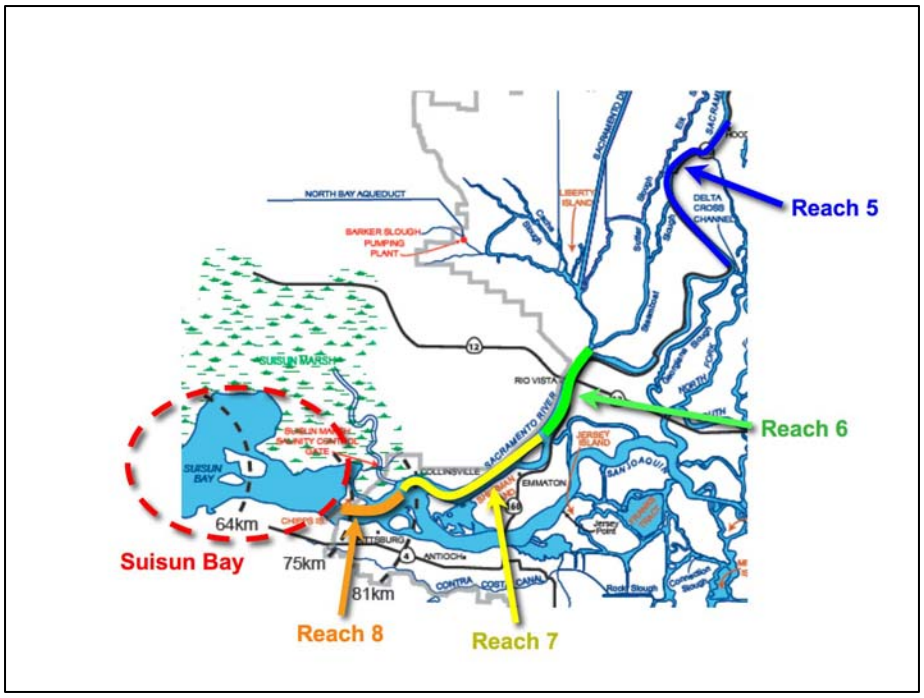


Figure 1: Sacramento River Reaches

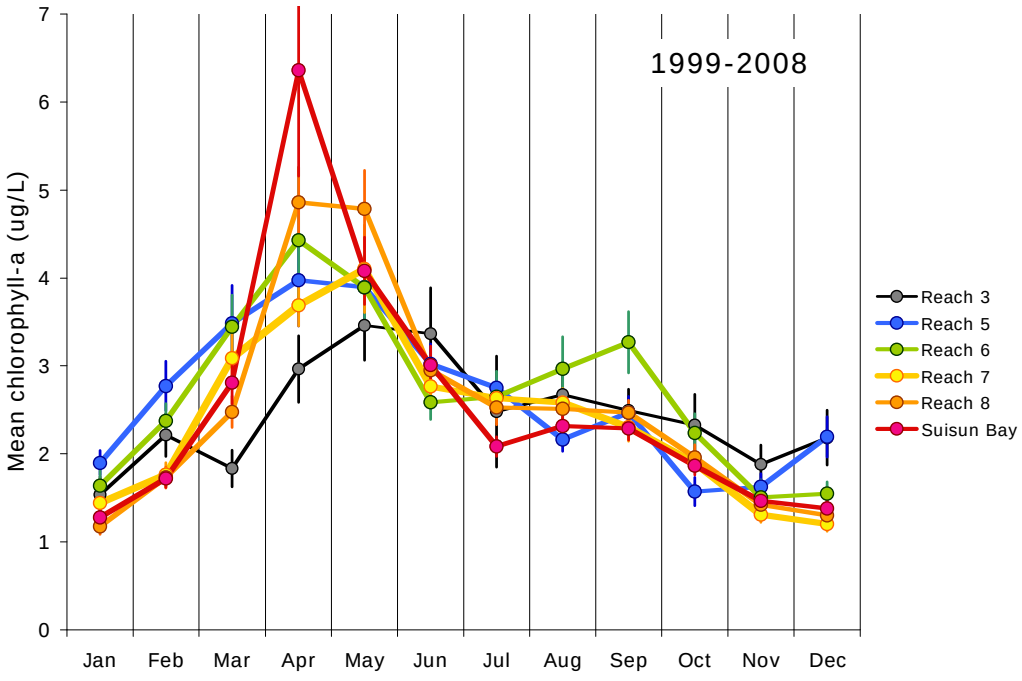
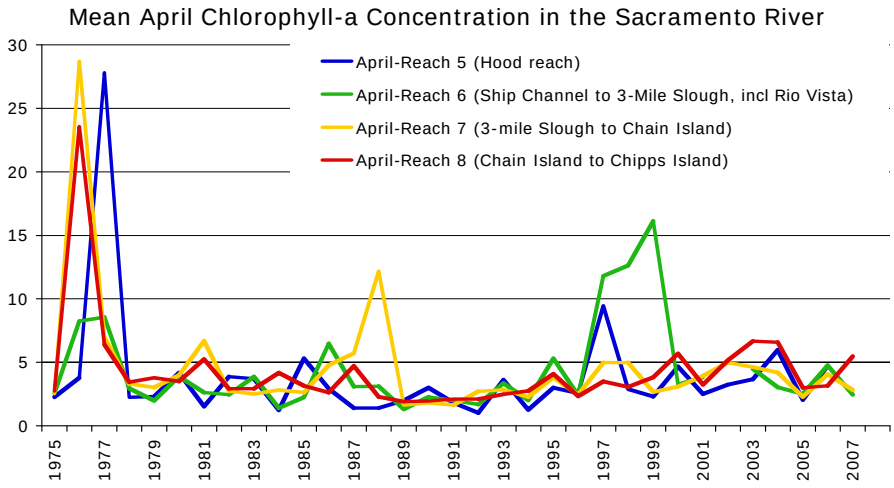


Figure 2: Temporal and Spatial Distribution of chlorophyll-a

Figure 3:



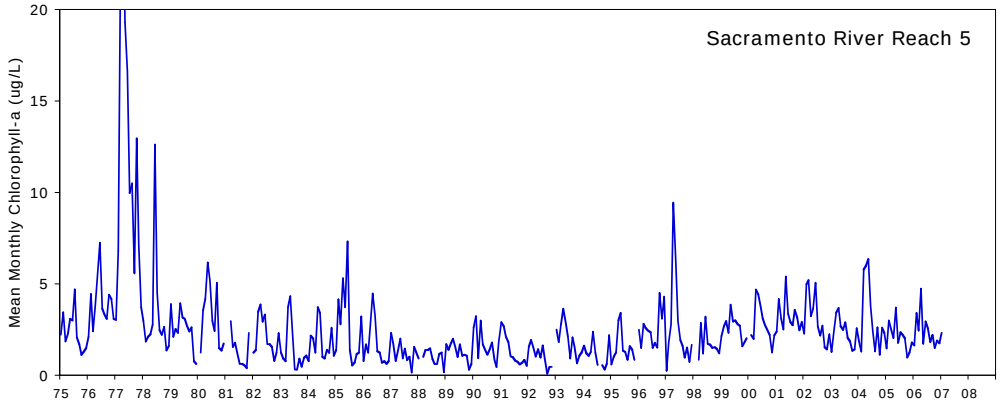


Figure 4: Mean Monthly Chlorophyll-a in the Sacramento River Reach 5, includes Hood and Greene's Landing

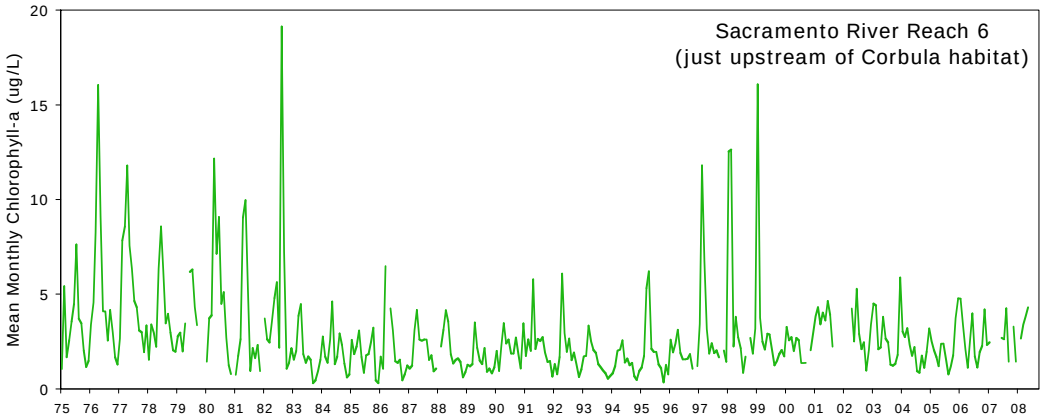


Figure 5: Mean Chlorophyll-a in the Sacramento River Reach 6