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EVALUATION OF CHEMICAL, TOXICOLOGICAL, AND HISTOPATHOLOGIC DATA TO DETERMINE THEIR ROLE IN THE PELAGIC ORGANISM DECLINE

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FORWARD

Monitoring conducted by the Interagency Ecological Program has shown declines in the abundance of four pelagic fish species in the San Francisco Bay /Sacramento - San Joaquin Delta Estuary (Delta), later coined the Pelagic Organism Decline (POD). Three major factors are hypothesized as contributing to the POD: water management operations, contaminants, and invasive species.

The State Water Resources Control Board and Central Valley Regional Water Quality Control Board (the Water Boards) contracted a study with the University of California, Davis to compile and review available data to determine if there are sufficient data to characterize the extent and role of contaminants in the POD. This study used historical water chemistry, toxicity, and histopathological data to attempt an understanding of the effect of contaminants by integrating population ecology and ecotoxicology.

In general, the study found that there was insufficient high quality data available to make conclusions about the potential role of specific contaminants in the POD. Data identified from the legal Delta proved to be very limited, leading to the inclusion of data from tributaries as far as 30 miles outside of the legal Delta. Therefore, and as stated in the conclusions of the report, care should be taken in drawing any specific conclusions about the effect of contaminants on the POD based on the data found and included in the report.

Disclaimer:

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LIST OF ACRONYMS

CDFG	California Department of Fish and Game
COSB	Cooperative Striped Bass
CVRWQCB	Central Valley Regional Water Quality Control Board
DCM	Dichloromethane
DDD	Dichlorodiphenyldichloroethane
DDE	Dichlorodiphenyldichloroethylene
DDT	Dichlorodiphenyltrichloroethane
DOC	Dissolved Organic Carbon
EC	Specific conductance (sometimes Electrical conductivity)
ELISA	Enzyme Linked Immuno-Sorbant Assay
K_{oc}	Organic carbon partitioning coefficient
LOEL	Lowest Observed Effects Level
LC50	Concentration of a contaminant at which 50% of the test organisms die
MDL	Minimum Detection Limit
NOEL	No Observed Effects Level
ND	Non Detect
NPDES	National Pollution Discharge Elimination System
OECD	Organisation for Economic Cooperation and Development
OP	Orthophospate (usually refers to a pesticide)
PAH	Polycyclic Aromatic Hydrocarbon
PBDE	Polybrominated diphenyl ether
PBO	Pipernyl butoxide
PCB	Polychlorinated Biphenyl
pH	power of hydrogen
POD	Pelagic Organism Decline
RL	Reporting Limit
SRCWWTP	Sacramento Regional County Waste Water Treatment Plant
SWAMP	Surface Water Ambient Monitoring Program
SWWTP	Stockton Waste Water Treatment Plant
TIE	Toxicity Identification Evaluation
TRE	Toxicity Reduction Evaluation
US EPA	U.S. Environmental Protection Agency
USGS	United States Geological Survey
UTM	Universal Transverse Mercator
WET	Whole Effluent Test
WWTP	Waste Water Treatment Plant

EXECUTIVE SUMMARY

The rapid decrease in the abundance of the delta smelt, longfin smelt, threadfin shad, and striped bass has been called the Pelagic Organism Decline (POD). One of the hypothesized causes is exposure to contaminants from various sources. The goal of the following review is to determine if sufficient data are available to conclude that contaminants are partially or wholly responsible for the POD. The review examines chemical, toxicity, and histopathology data from monitoring programs and studies conducted on organisms in the Delta.

The review was initially conducted by examining publically available data from databases developed by current or past monitoring programs however was expanded to include both unpublished and published reports. To provide a historical perspective, data analysis and conclusions from the review developed for the California Urban Water Agencies by Fox and Archibald (1997) were included in the following report. The Fox and Archibald (1997) review reported the results of numerous studies performed by state and federal agencies, as well as university research.

After examining POD species' life histories and demography and relying on the Bayesian change point analysis by MacNally *et al.* (in review), it was determined that a step decline did occur between 2000 and 2002 for at least three of the species; delta smelt, threadfin shad, and striped bass. The longfin smelt may not have experienced a steep decline in abundance but did experience a more gradual decline during that period. The life histories of the four POD species suggest that decreased survival of larval and juvenile stages could cause steep declines in abundance. Larval and juveniles of all POD species are found in the Delta during January to June and therefore contaminants in the system could result in decreased survival of larvae and juveniles by either direct or indirect toxicity. Direct toxicity to the larvae and juvenile POD species could be due to contaminant exposure from January to June whereas indirect toxicity may be the result of prey item exposure to contaminants resulting in an indirect affect on the survival of POD species.

Six critical comparisons are made that allow the evaluation of the hypothesis that contaminants are partially or wholly responsible for the decline: 1) concentrations of chemicals, or the amount of toxicity, or the number of lesions in fish from the pre-POD period (prior to 2000) is less than during the POD period, 2) the steepest declines in abundance experienced in 2000-2002 are accompanied by higher concentrations of chemicals, a greater amount of toxicity, or elevated number or severity of lesions than those experienced from 2003-2008, 3) there is

evidence of chemicals present in toxic amounts, demonstrated toxicity, or lesions associated with the January to June period, 4) there is differential sensitivity of POD and non-POD fish species to chemicals present during the POD, 5) there is differential mortality of prey consumed by POD and non-POD species, and 6) threadfin shad are less sensitive to chemicals present during the POD years than the remaining POD species. To conduct the above six comparisons, data was compiled and reviewed for chemistry, toxicity and histopathology relevant to POD species (pre-POD (prior to 2000), POD decline years (2000-2002) and post POD years (2003-2008)).

As part of the review a comprehensive database of water quality results and toxicity data for the Delta for the POD years was developed. The geographic scope of the data included the legal Delta +30 miles. Originally, over 1 million data records were assembled. Data were removed from the analysis if they did not include detection or reporting limits, lacked an identifiable analyte name, were not associated with an identifiable sample site location, and/or units of measure were recorded incorrectly, e.g. chlorpyrifos measured in seconds or nitrate measured in m^3.

Review of the water chemistry data found that there were few chemicals with sufficient data available to draw conclusions about the role of contaminants in the POD. Many chemicals were analyzed with detection limits that were too high resulting in non-detects at levels above toxicologically relevant levels. Other chemicals (e.g. pyrethroids) were not preserved properly leading to the potential for detected concentrations to be biased low. Comparisons with data presented in reports released prior to the POD indicate that chemicals are not found in higher concentrations during the POD years compared to the pre-POD years. Very little data exist to determine if higher concentrations of chemicals occurred during the 2000-2002 step decline period compared to the later POD period. There are too few data to adequately address the January to June concentrations of chemicals with a few exceptions: chlorpyrifos and diazinon. Chlorpyrifos occurred in toxic concentrations in 5.4% of the samples collected and diazinon occurred in toxic concentrations in 4.9% of the samples. There are no toxicity data available to determine if the threadfin shad is relatively less sensitive to chemicals present in the Delta, and the question of why the threadfin shad is increasing in numbers while other POD species are declining is not able to be addressed with chemical data. The cursory review of the relative sensitivity of POD and non-POD species to various chemicals found in the Delta during the POD years does not suggest that POD species are more sensitive. However a more detailed review is being undertaken and the above conclusion is considered preliminary. Striped bass are much more sensitive to chlorpyrifos than the non-POD species reviewed and chlorpyrifos was the one chemical that experienced exceedances of Water Quality Goals for more than 5% of the

samples. Direct toxicity to POD species sufficient to cause the POD is unlikely, but toxicity to prey items could occur given the analysis of the limited data available.

Review of toxicity data indicate that there was as much or more toxicity in water samples collected in the Delta in the pre-POD years compared to the POD years. There appears to be no difference in the percentage of toxic water samples to either *Ceriodaphnia dubia* or *Pimephales promelas* between the 2000-2002 step decline years and the later POD years. The percentage of toxic samples in the January to June period varied between 0% and 7% across years and monitoring programs. Many of these toxic samples were collected from water bodies that are tributaries to the major rivers and it is not clear how transit time and dilution would affect the toxicity of these waters. The percentage of toxic samples collected from Delta waters is slightly lower and less frequent but indicates the potential for toxicity to prey items utilized by POD species. Significant toxicity (50% to 80% of tests performed) in sediment was common throughout the POD period. The significance of sediment toxicity is unknown as it is the interstitial water in the sediment that causes toxicity. Giesy *et al*. (1999) argue that concentrations of chlorpyrifos in sediment interstitial water can not be greater than the concentration in the water column meaning that resuspension of sediments and contaminants would not increase the concentration of chlorpyrifos in the water column or cause additional toxicity.

Review of the histopathology data indicates that there are insufficient data from the pre-POD period to determine if lesions were more or less common or severe prior to the POD years. Ostrach's (2009) report suggests that striped bass have been experiencing reproductive failure due to organochlorine compounds since prior to the POD years. Due to the lack of histopathology studies in the early POD years, there are insufficient data to determine if histopathologies were greater during the 2000-2002 POD period compared to the later POD years. For data collected in the later POD years, overall there is little evidence of major histopathologies in POD species or non-POD species. Some lesions found in delta smelt do suggest exposure to contaminants, although these lesions were not found in every year. Although lesions can take long periods of time to develop, some lesions were described as developing in the few weeks prior to capture of the fish in the fall suggesting exposure to chemicals outside of the period of larval and juvenile development. Stomachs full of food upon capture suggest that delta smelt are not starving. This further suggests that the food supply has not been reduced by exposure to contaminants and can support populations of POD species.

It appears there are insufficient data to parameterize any statistical or physical model that might formally test the hypothesis that contaminants are a cause of the decline. Consequently the results of the six comparisons for chemistry, toxicity and histological data were placed into a weight of evidence context. **The conclusion that is drawn from the analyses is that while contaminants are unlikely to be a major cause of the POD, they cannot be eliminated as a possible contributor to the decline.** Unfortunately, while future research can address our understanding of the relative sensitivity of POD species to various contaminants in the system, it cannot recreate history. It is unlikely that trying to glean data from current monitoring programs will be able to address issues similar to the POD in the future. Current monitoring programs are conducted with a specific purpose and it should not be expected that they can provide information to address issues such as the POD. To avoid the lack of adequate data in the future, it is recommended that a long-term monitoring program including chemical analysis, toxicity testing, and some histopathology analyses be undertaken. These analyses would presumably be combined with monitoring of other indicators such as fish, plankton, invertebrates, and physical parameters. A series of recommendations about this program are provided that address topics from the conceptual development of the program to specific tests to be performed.

ACKNOWLEDGMENTS

Several individuals were instrumental in the development and completion of this review. Mark Gowdy of the State Water Resources Control Board managed the contract and allowed the work to keep moving. Karen Larsen, originally with the Central Valley Regional Water Quality Control Board (CVRWQCB), initiated the project and provided significant guidance in the early stages of the project. Stephanie Fong took over management of the report after Karen's departure for the State Board and guided the project to completion. Chris Foe of the CVRWQCB provided substantive technical input to the project throughout. Additional thanks go to Francisca Johnson for GIS support and Melissa Turner for editing and formatting.

The Expert Panel that reviewed this document included Susan Anderson, Jeff Miller, Debra Denton, and Lisa Thompson. Susan reviewed two drafts of this document and provided exceptionally helpful comments on the organization and the consistency of the key structural elements of the review. The remaining reviewers were able to provide equally helpful reviews in a very short time to allow the delivery of this review to the CVRWQCB. They provided insight and knowledge in their areas of expertise and greatly improved the review. With more time, their comments could have been more fully incorporated into this review and I hope they believe that their comments are adequately reflected in the revisions.

Because the time between the return of the reviews and the delivery of the final report, my coauthors (Werner, Teh, and Loge) have not had a chance to evaluate the revisions and conclusions and may not entirely agree with the final conclusions in this review. I take full responsibility for the current content of the review and all mistakes are my responsibility.

Michael L Johnson

PARTICIPANTS

There were several individuals that were instrumental in the completion of this document. The UC Davis team that developed the review included Michael Johnson, Inge Werner, Swee Teh, and Frank Loge. External reviews were provided by Susan Anderson (independent consultant), Debra Denton (US EPA), Jeff Miller (Aqua-Science), Lisa Thompson (UC Davis Cooperative Extension), Chris Foe (Central Valley Regional Water Quality Control Board), and Stephanie Fong (Central Valley Regional Water Quality Control Board). These individuals are profiled below.

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survival and reproduction of aquatic organisms, and ultimately linking individual health to population decline.

Dr. Frank Loge is a Professor in the Department of Civil and Environmental Engineering at the University of California Davis. He has been actively involved in fisheries research in the Columbia River Basin, San Francisco Bay Delta, and Puget Sound for the past 10 years investigating factors influencing the decline of pelagic and anadromous fish. He has expertise in population lifecycle modeling, biological measures of health and fitness, statistics, and risk assessment.

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Dr. Lisa Thompson is a Fisheries Extension Specialist at UC Davis, in the Wildlife, Fish, and Conservation Biology Department, focusing on the adaptive management of anadromous and inland fish populations. She completed her B.Sc. at the University of Toronto, M.Sc. at McGill University in Montreal, Ph.D. at the University of British Columbia, and did her postdoctoral research at the University of California, Santa Barbara. In California she has conducted studies of fish response to environmental factors such as flow and temperature in the South Fork American River, Cow Creek (Sacramento Basin), the Shasta River (Klamath Basin), the Upper Salinas River Basin, and Pine Creek (Eagle Lake). She is currently involved in projects to: predict the effects of climate change on threatened Butte Creek spring-run Chinook; to restore a naturally spawning population of Eagle Lake rainbow trout; and to study the temperature tolerance and preferences of the hardhead minnow (a California Species of Special Concern). She has been a member of the Shasta-Scott Coho Recovery Team, Southern Steelhead Technical Recovery Team, Spring-run Chinook Workgroup, and the Lower American River FISH Workgroup.

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INTRODUCTION

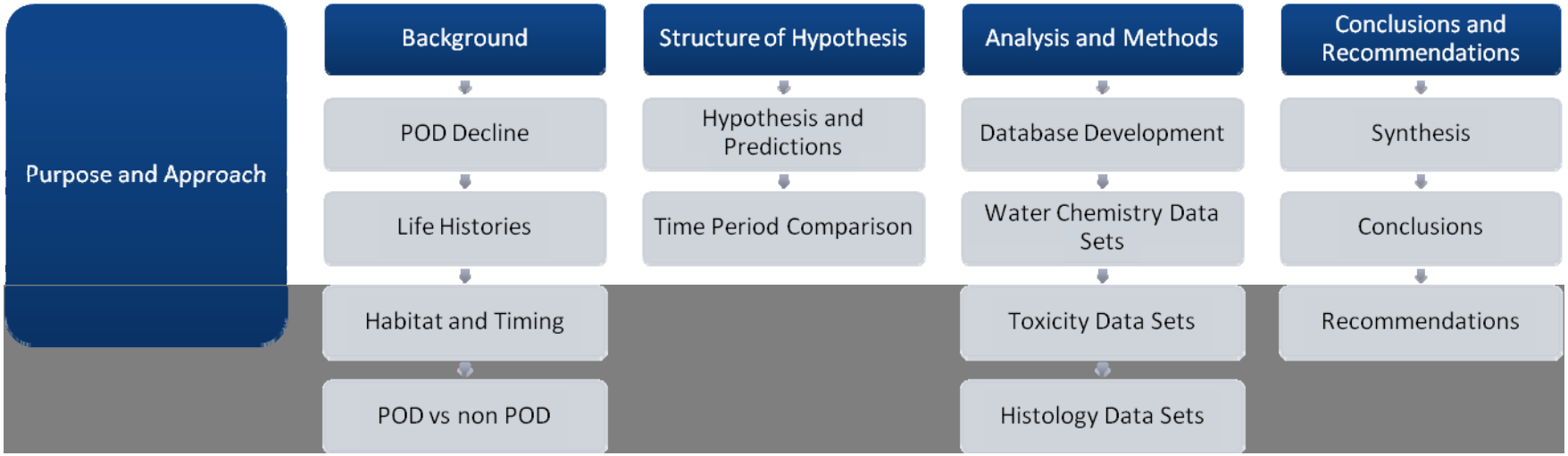
The decline of populations of several species of fish in the Sacramento-San Joaquin River Delta is well documented. Of particular concern is the decline of four species that spend a portion of their life in the Delta; the delta smelt, longfin smelt, threadfin shad, and striped bass. These four species, generally termed the Pelagic Organism Decline (POD) species, are thought to be indicators of the overall health of the Delta ecosystem and consequently, identifying the cause(s) of their decline has become the focus of a large effort by numerous state and federal agencies. One of these agencies, the State Water Resources Control Board through the Central Valley Regional Water Quality Control Board requested a review of the available contaminant data, water and sediment toxicity data, and histopathology data to determine the role of contaminants in the POD. The goal of the review is to determine if sufficient data are available to conclude that contaminants are a partially or wholly responsible for the Pelagic Organism Decline. Specifically, 1) are there sufficient water chemistry data available to indicate the presence of contaminants in the Delta at concentrations necessary to cause sublethal or lethal effects sufficient to cause and/or maintain the POD, 2) are there sufficient toxicity data available to indicate the presence of contaminants in the Delta at concentrations necessary to cause sublethal or lethal effects sufficient to cause and/or maintain the POD, and 3) are there sufficient histopathology data available to indicate that species of fish in the Delta have been exposed to contaminants at concentrations necessary to cause sublethal or lethal effects sufficient to cause and/or maintain the POD? Toxicity could occur directly to POD species or to species in the trophic web that connects the POD species to primary productivity. An ancillary question is; are models of fish population dynamics available that might be used to address questions about the effects of stressors on POD species in the Bay-Delta system. This issue is addressed in Appendix III.

This review was initially restricted to examining data that are publically available in databases developed by current or past monitoring programs. However, it became clear that there are very few data to address the questions posed above, especially with respect to histopathology. Consequently, the search for data was expanded slightly to include unpublished or published reports where the data will eventually become publically available in a venue such as the California Environmental Data Exchange Network. Also, to provide an historical perspective on the current status of chemical analysis, toxicity testing, and histopathology, the review of Fox and Archibald (1997) was used. That review reported the results of numerous studies performed by state and federal agencies, as well as some research performed at universities in the region. As indicated below, the original reports reviewed in Fox and Archibald were not obtained for the current review. Also, no attempt was made to perform an exhaustive review

of published research since 2000 since the focus of the review was to determine if sufficient data exists to address the questions posed above. Lastly, this review necessarily does not address the complex interactions that may exist between contaminants, invasive species, change in climate, water diversions, or any of the additional myriad of factors that can impact the population dynamics of the POD species. As such, the review can be criticized for being incomplete by not considering all possible indirect interactions. However, the UC Davis team and the panel of expert reviewers have developed the most effective review possible given the constraints on the analysis. Clearly, there is opportunity for additional analyses.

The synthesis is organized in five sections with appendices supporting the text (Figure 1). In addition, an historical perspective is provided for each of the data sets.

Figure 1. Synthesis report structure.



APPROACH

The POD species have experienced a decline in abundance for at least a decade (although see below for the threadfin shad). From 2000-2002, it is thought that the decline involved a dramatic decrease in abundance, followed by a more gradual decline from 2003 to the present. The initial decline is often termed the "step decline". The ability to determine if contaminants are wholly or partially responsible for the step decline and the subsequent continual decline in abundance of the POD species depends on both an understanding of the toxicological evidence (chemistry, toxicity, and histopathology) for effects on POD species, and the life histories of POD and non-POD species. Gaps in our understanding of either of these areas can preclude a conclusion about the effects of contaminants on POD species. Consequently, it is important to provide a brief discussion of the relevant ecological issues and their intersection with the chemical, toxicity, and histopathology evidence available for effects of contaminants on POD species, non-POD species, and the prey species on which both depend. This review uses a combination of basic population biology to understand the potentially sensitive life stages of POD species, along with documenting an exposure pathway(s) which requires an overlap in space and time of the POD species and the contaminants. This integrative approach was broadly applied to the four POD species and other species in the Delta to address the questions posed above.

BACKGROUND

PELAGIC ORGANISM DECLINE

This review is predicated on the assumptions that real step-declines occurred in the early years of this century to all four populations, and that the declines were synchronous (or relatively so). Recently under the auspices of the National Center for Ecological Analysis and Synthesis, a Bayesian change point analysis was performed to determine if these assumptions are correct (MacNally *et al.* in review). The results of that analysis are unpublished but generally confirm that at least three of the four POD species experienced step declines in population abundance (as measured by the numbers caught in the Fall Mid-Water Trawl). Delta smelt experienced a step decline in 2000-2002, threadfin shad experienced a step decline in 2002, and striped bass experienced a step decline in 2002. Longfin smelt demonstrated little evidence of a step decline in those years although longfin smelt have been experiencing a gradual decline since the 1960s with only two short periods of increasing abundance in the 1970s and 1990s. There has been a significant decline in abundance of striped bass for over 30 years, with only one period of slightly increasing abundance in the early 1980s. The abundance of all species demonstrated continuous declines in numbers since the period of the step decline with the exception of the threadfin shad which has experienced a slight increase in abundance since 2002 (MacNally *et al.* in review). Consequently, the assumption that all four species' demographies are responding similarly over the last decade is not met completely. However, given the error involved in the measurements of abundance of the four species and the overall decline of all four species through at least 2002 allows the analysis to move forward.

If contaminants are wholly or partially responsible for the step decline and/or continued reduced population numbers, one issue that must be reconciled is the increase in population growth of other pelagic species in the Delta including the Inland silverside and several species of centrarchids (sunfish and bass). Many of these species use the same general habitat as the POD species and Inland silverside are considered to be potential predators on delta smelt placing them in the same habitat for at least some period of time. Consequently, all pelagic species in the Delta would be expected to be exposed to the same contaminants. If all species are exposed to contaminants, POD species would necessarily be more sensitive and experience population level effects at lower concentrations than non-POD species.

LIFE HISTORIES

The life histories of the POD species are well described and will not be repeated here in any detail. However, for this analysis, the critical aspects of the life cycle include the timing of reproduction, the location where the species spend the various portions of their life, and the length of life. In addition, the life histories and habitat use of other pelagic fish species are additional factors that must be addressed. This section of the synthesis provides:

- An analysis of life histories of POD species to determine the life stage that when impacted by stressors, provides the greatest chance of population declines
- A determination of whether there is overlap in space and time of the most sensitive life stage
- A review of non-POD species' habitat use to address the increase in abundance of other pelagic species
- An analysis of the population dynamics of all POD species

REPRODUCTION

The life history of a species is the description of the allocation of energy to reproduction and survival throughout the life of individuals of that species. Three of the four POD species live one (DS) or two years (TS, LS) and reproduce a single time (Moyle 2002). There is a current hypothesis that some delta smelt females can live two years prior to reproducing and can produce a larger number of eggs (Bennett personal communication) however it appears that the majority of individuals live a single year. Most striped bass can live for perhaps 8-10 years with a maximum age of 30 years, and age at first reproduction for females is usually 4-6 years (Moyle 2002).

POPULATION GROWTH RATES (λ)

The life histories of the POD species all point to reduced juvenile survival as the critical stage causing population declines. The life cycles of the DS, TS, and longfin smelt are very similar and their population growth rate λ, is the product of the survival to age of reproduction and per capita fecundity at reproductive maturity (Crone 2001). Consequently, proportional changes in survival and fecundity affect λ equally. Analyses of life histories across numerous taxa (see Heppell *et al.* 2000 for a review of mammalian life histories, Saether and Bakke 2000 for a

review of avian life histories, and Velez-Espino *et al.* 2006 for a review of fish life histories) indicate that population growth rates of rapidly maturing species respond positively (i.e. increased population growth rates) to improved survival of offspring, while growth rates of later maturing species respond to juvenile and adult survival rates, the relative importance of the two is determined by the amount of time spent in the juvenile and adult age classes.

Striped bass maintain a very different life history in which females mature at 4-6 years and are reproductively active for several years. Evaluating the striped bass life history suggests adult survival has the greatest impact on population growth rate, i.e. reductions in adult survival would have the greatest potential for decreasing λ followed by juvenile survival. Velez-Espino *et al.* (2006) found that for long lived species, as longevity and age at maturity decrease, there is increasing importance of juvenile survival on λ. Based on the analysis of Velez-Espino *et al.* (2006), compared to other North American fishes striped bass are in the category with decreased longevity and decreased age at maturity and it is expected that changes in both adult survival and juvenile survival would have similar effects on λ. Consequently, stressors that decrease juvenile and/or adult survival would reduce λ more rapidly than decreases in reproduction (decreased egg production). Whether reductions in survival are due to direct toxicity or indirect effects is unknown. Indirect effects include toxicity to food items resulting in starvation or decreased condition factor, compromised immune response, and eventually reduced survival.

The conclusion from this discussion is that for all species, rapid population declines can be driven by reduction in early juvenile survival.

HABITAT AND TIMING

The role of contaminants in the POD is strengthened if there is a period of time in which all species are present together as juveniles. However, this is only one of three possibilities for how exposure can occur; 1) all species are found at the same place and time as the contaminant(s), 2) species are exposed in different locations in the Bay-Delta system by the same chemical(s), or 3) species in different locations in the Bay-Delta system are exposed to different chemicals. While it is possible for the same contaminants to be causing toxicity to the POD species in different locations at the same time of the year, or different contaminants to cause similar decreases in survival in different habitats at different times of the year, it is more parsimonious to conclude that similar life history stages are being impacted in the same general

habitat by the same contaminant(s) during the same period of time. This explanation requires that there is a period in which all POD species are located in the same region of the Delta at the same time, or that the chemical(s) to which they are exposed are found widely distributed across the Delta and estuary such that exposure to all species could occur. The only period of time during the year in which all species are located in the same physical location is the period from March through May (with some potential for additional months on either end, the period could be considered January to June) when all species are spawning in the freshwaters of the Delta. Throughout the rest of the year, the species are located in very different habitats including the open ocean by both STRIPED BASS and LS. The period of spatial overlap by the species occurs during spawning and early development when larvae can be susceptible to contaminants. For the POD species, life history theory suggests that the greatest declines in population abundance would be the result of decreases in juvenile survival of all four species. The analysis of the habitat utilization by POD species suggests that the exposure most likely to cause declines in early survival would occur during the winter-spring period when spawning occurs and larval and juvenile fish of all species are present in the Delta (summarized in Table 1). Consequently, a search for the presence of contaminants, evidence of toxicity, or histopathologic markers of exposure is focused first on the same winter-spring period.

Table 1. Life history summary of POD species. Each column provides

POD Species	Distinct Step Decline	Age to Maturity (years)	Life Span (years)	Reproduction	Growth Rate Dependence (Survival)	Acute Toxicity Susceptibility
Delta smelt	Yes	1*	1*	Once	Juvenile	March - May
Longfin smelt	No	2	2	Once	Juvenile	March - May
Threadfin shad	Yes	2	2	Once	Juvenile	March - May
Striped bass	Yes	4-6	8-10	Multiple	Juvenile/Adult	March – May†

*some individuals may possibly live 2 years (Bennett personal communication)

†low salinity zone

POD AND NON-POD SPECIES POPULATION DYNAMICS

Finally, addressing the issue of the population declines of the POD species concurrent with increases in abundance of several other pelagic species is problematic. There are several reasons why different species are experiencing different population dynamics including; 1) although all species are pelagic, there is not sufficient habitat overlap to allow exposure of all species, 2) exposure occurs but direct toxicity is being experienced differentially by POD and non-POD species, and/or 3) toxicity to prey items differentially effects POD species relative to non-POD species. These are enumerated below.

Differential exposure resulting in differential direct toxicity. Species such as the Inland silverside which has experienced explosive population growth may not be exposed to the contaminants at the same sensitive life stages as the POD species. This would require temporal and/or spatial differences in their distribution during periods when exposure of the POD species is occurring. Based on current knowledge, this scenario is unlikely. Species such as the Inland silverside, bluegill sunfish, and smallmouth bass are found in the same habitats during the same periods of the year. Also, they spawn at the same general time and location (Moyle 2002) meaning their pelagic larval and juvenile stages would be in the Delta at the same time as those stages of the POD species. Finally, slight differences in habitat use by POD species and species such as sunfish or smallmouth bass would probably not result in significantly differential exposure as the water would move the contaminants into and out of these different habitats as it passes through the Delta on its way to the pumping plants near Tracy or San Francisco Bay.

Differential sensitivity of POD and non-POD species to the effects of contaminants. It is possible that the POD species are at the extreme end of the gradient of sensitivity to contaminants and species such as the Inland silverside are relatively insensitive. Recent research into the sensitivity of POD species to various contaminants such as ammonia (Werner 2009) suggests that delta smelt are extremely sensitive. However, few data are available to evaluate the sensitivity of POD and non-POD pelagic species to the range of contaminants found in the Bay-Delta system at various times of the year. Currently, there is a suggestion that this hypothesis may be correct, but there are insufficient data to fully assess it. A review of the sensitivity of taxonomically related POD and non-POD species is underway.

Differential toxicity to prey items utilized by POD and non-POD species. If POD and non-POD species utilize different prey items which themselves experience differential survival when

exposed to contaminants, differential mortality of POD and non-POD species could occur. Clearly, as adults the silverside, sunfish, and bass are predatory and would consume different prey than DS, LS, and TS. And, since they live several years, they would be present as adults in the system at the same time as the smelt and shad are present as larval and juvenile stages. As such, they may be less sensitive to the effects of contaminants. However, they also spawn during the same general period as the POD species and their larval and juvenile stages are present as pelagic organisms at the same time as the POD species. Food items during these stages for POD and non-POD species are different (Grimaldo *et al*. 2004). Threadfin shad are filter feeders that remove small cladocerans, copepods, and rotifers through the gill rakers. However, they also consume detritus and phytoplankton (Turner and Kelly 1966). Larval longfin smelt also feed on copepods (The Bay Institute 2007) and larval feed on copepods and cladocerans and juvenile striped bass switch to feeding on mysid shrimp until they are capable of piscivory. Bennett (2005) and Grimaldo *et al.* (2004) indicate the preferred diet item of delta smelt consists of calanoid copepods while the preferred diet item of the early life history stages of sunfish and bass are copepods, cladocerans and other small invertebrates (e.g. chironomids, amphipods) within the macrophyte beds. Larval delta smelt feed on the same food items as adults but larval fish select subadult copepods while adult delta smelt select adult copepods (Nobriga 2002). Grimaldo *et al.* (2004) performed stable isotope analyses and concluded that the open water food web and macrophyte food web have very little overlap. Invertebrate production in the macrophyte beds is most likely fueled by epiphytic algae production on the leaves of the macrophytes and/or detritus produced by the macrophyte bed. Copepod production in the open water is driven by phytoplankton production. There is also evidence that detritus and perhaps dissolved forms of carbon fuel a ciliate pathway for the copepods. Both the copepods and phytoplankton are affected by clam grazing which does not appear to be a factor in the macrophyte beds although clams are found around the edges of the beds (Larry Brown personal communication).

Given that prey items of POD and non-POD species are different, for these indirect effects to fuel the POD, there would need to be either a differential exposure of contaminants to invertebrates in macrophyte beds and in the pelagic zone, or differential sensitivity to the contaminants to which they are both exposed, i.e. those prey consumed by POD species would need to be more sensitive to contaminants than prey consumed by non-POD species. Recent research suggests that there are differences in the sensitivity of different invertebrates to contaminants found in the Bay-Delta ecosystem. Both Weston *et al.* (2004) and Werner (2008) utilized the amphipod *Hyalella azteca* as a test organism when evaluating potential toxicity from pyrethroid pesticides because of its extreme sensitivity relative to species such as the copepod *Ceriodaphnia dubia* (50 fold difference in sensitivity). It is possible that differences in

sensitivity to other contaminants exist among the prey species of POD and non-POD species, but there are no data currently available to evaluate this possibility. Consequently, it is not possible to evaluate the hypothesis that differences in the population dynamics of the POD and non-POD species are driven by differences in the effects of contaminants on food items in their diets during early life stages.

POD POPULATION INCREASES – THREADFIN SHAD PHENOMENON

The final phenomenon that should be explained by any model of contaminant-driven POD is the step decline in the population of threadfin shad followed by the gradual increase in abundance in the subsequent years. The threadfin shad is the only POD species that is found exclusively in the freshwater portions of the Bay-Delta ecosystem year around. STRIPED BASS are also found in the freshwater portions of the Delta year around, but portions of the population do move into Suisun and San Francisco Bays and the Pacific Ocean. Threadfin shad overlap in habitat with all POD species during the late winter-early spring spawning season. One possible explanation is that the step decline was caused by exposure to high concentrations of certain contaminants which caused decreases in survival and the resulting step decline. The threadfin shad would be the least sensitive of the POD species such that a decrease in the concentration of the contaminant(s) would result in minimal effects on threadfin shad but continued decreases in the survival of the remaining POD species. If this is the case, there should be a contaminant in the Delta that was present in higher concentrations during the step decline years and in slightly lower concentrations since that time. An alternative explanation is that the critical period for exposure is not the late winter-early spring when all POD species are present in the Delta, but in several other habitats during the remaining portions of the year. Although this is not the most parsimonious explanation as it requires the same contaminant to be present in several habitats (i.e. open ocean, San Francisco Bay, Suisun Bay) at concentrations sufficient to cause the declines, or different contaminants are present in the different habitats at concentrations sufficient to cause decreases in survival, it remains a possibility.

Summarizing the results of the discussion above:

- Analysis of the life histories of all POD species suggests that contaminant effects on survival, primarily survival of larval and juvenile fish, could be sufficient to trigger the POD and maintain the declines over the subsequent years. Because of the normally very low survival from egg to adult, the reductions in survival would not necessarily need to be dramatic to result in a decline meaning a contaminant-driven decrease in survival could be difficult to detect. Conversely, it appears unlikely that decreases in fecundity driven primarily by decreases in egg production, could be responsible for the POD. Until good estimates of the vital rates for all species are available for use in a formal analysis, these conclusions are only preliminary. To focus the review however, these conclusions were assumed to be correct.

- All POD species are in the Delta during the spring period when spawning is occurring and juvenile fish are present. Consequently, focusing on the presence of chemicals and the occurrence of toxicity in the spring is necessarily a major focus of the analysis.

- Several non-POD species including Inland silverside, various species of sunfish, largemouth bass, and smallmouth bass are present in the Delta at the same time as POD species including overlap in spawning and habitat utilization by larval and juvenile stages. Significant increases in abundance of numerous non-POD species require differential sensitivity to contaminants with POD species being much more sensitive to the effects of contaminants than are non-POD species. Additionally, prey items of the two groups of species are different and it is possible that increased mortality of the prey items of POD species relative to non-POD species are responsible for the decline.

- The combination of a step decline of the threadfin shad with a gradual recovery in abundance is a challenge to explain from a contaminant's perspective. One possible explanation is that the step decline was caused by exposure to high concentrations of certain contaminants which caused decreases in survival. The threadfin shad would be the least sensitive of the POD species such that a decrease in the concentration of the contaminant(s) would result in greatly reduced effects on threadfin shad but continued decreases in the survival of the remaining POD species.

- POD and non-POD species' life histories and habitat utilization do not preclude a mechanism for contaminants being partially or wholly responsible for the POD. The mechanism may be toxicity (either acute or chronic) to POD species directly or mediated through the prey items of each species.

STRUCTURE OF HYPOTHESIS

These conclusions allow the review to move forward to address the three questions posed in the introduction. To be able to definitively state that the answers to the questions are yes, the review must demonstrate the following.

1. There are sufficient water chemistry data available to indicate the presence of contaminants in the Delta at concentrations necessary to cause sublethal or lethal effects sufficient to cause and/or maintain the POD.

    a. *There were/are contaminants present in the Delta during the late winter-early spring period of the POD years allowing exposure of all species simultaneously.*

    b. *POD species in years with greater concentrations of specific contaminants experience greater decreases in abundance.*

2. There are sufficient toxicity data available to indicate the presence of contaminants in the Delta at concentrations necessary to cause sublethal or lethal effects sufficient to cause and/or maintain the POD.

    a. *Evidence of acute or chronic toxicity to POD species most probably during the larval and early juvenile stages should be present.*

    b. *POD species are more sensitive to the effects of contaminants than non-POD species that are not experiencing declines in abundance.*

3. There are sufficient histopathology data available to indicate that species of fish in the Delta have been exposed to contaminants at concentrations necessary to cause sublethal or lethal effects sufficient to cause and/or maintain the POD.

    a. *POD species display more evidence of histopathologies relative to non-POD species that are not experiencing declines in abundance.*

    b. *The histopathologies are known to be associated with exposure to contaminants present in the Delta.*

TIME PERIOD COMPARISON

One way to address the question of sufficient data is to compare abundance of POD species relative to the presence of contaminants from 2000-2006 to the abundance of POD species and the presence of contaminants in years prior to the POD. In the years prior to the POD, some POD species experienced increasing abundance, some decreasing abundance, and some species experienced abundances that varied substantially across the years. The change point analysis suggests that although delta smelt experienced highly variable abundances, there was a period

of gradual increasing numbers from the early 1980s to the period of the step decline in 2002 (MacNally *et al.* in review). The longfin smelt experienced a long period of declining numbers from around 1980 to the mid 1990s followed by a strong increase in abundance in the mid 1990s followed by another period of steadily declining abundance. Striped bass, with the exception of a short period of slightly increasing abundance in the early 1980s, experienced a steady decline in abundance from the 1960s to the present, and threadfin shad experienced a strong increase in abundance from the mid 1980s to 2002 when the species experienced the step decline in abundance. The abundances of all four species are different from each other in the pre-POD years relative to the POD years suggesting that the pre-POD abundance of each species was shaped by different factors. If contaminants were partially or wholly responsible for the POD, it would be expected that more toxic contaminants were present in the water, greater amounts of toxicity were present, and/or histopathologies would be much greater during the POD years than in years preceding the POD. Alternatively, if different types and/or greater concentrations of contaminants, more severe toxicity, and/or more frequent or severe histopathologies were detected in the years preceding the POD, then it is probable that contaminants are not playing a large role in the POD. Unfortunately, because there is not a single set of monitoring locations for contaminants from which data are generated, the conclusion will always be open to debate.

ANALYSIS AND METHODS

Numerous projects were conducted during the period prior to the POD including examination of water chemistry, toxicity, and histopathology of some POD species. Although the analyses of water chemistry suffer from many of the problems that studies from the early POD years suffer (e.g. no reported detection limits, no quality control data), some data are available and useful for comparison to data from the POD period.

DATABASE DEVELOPMENT

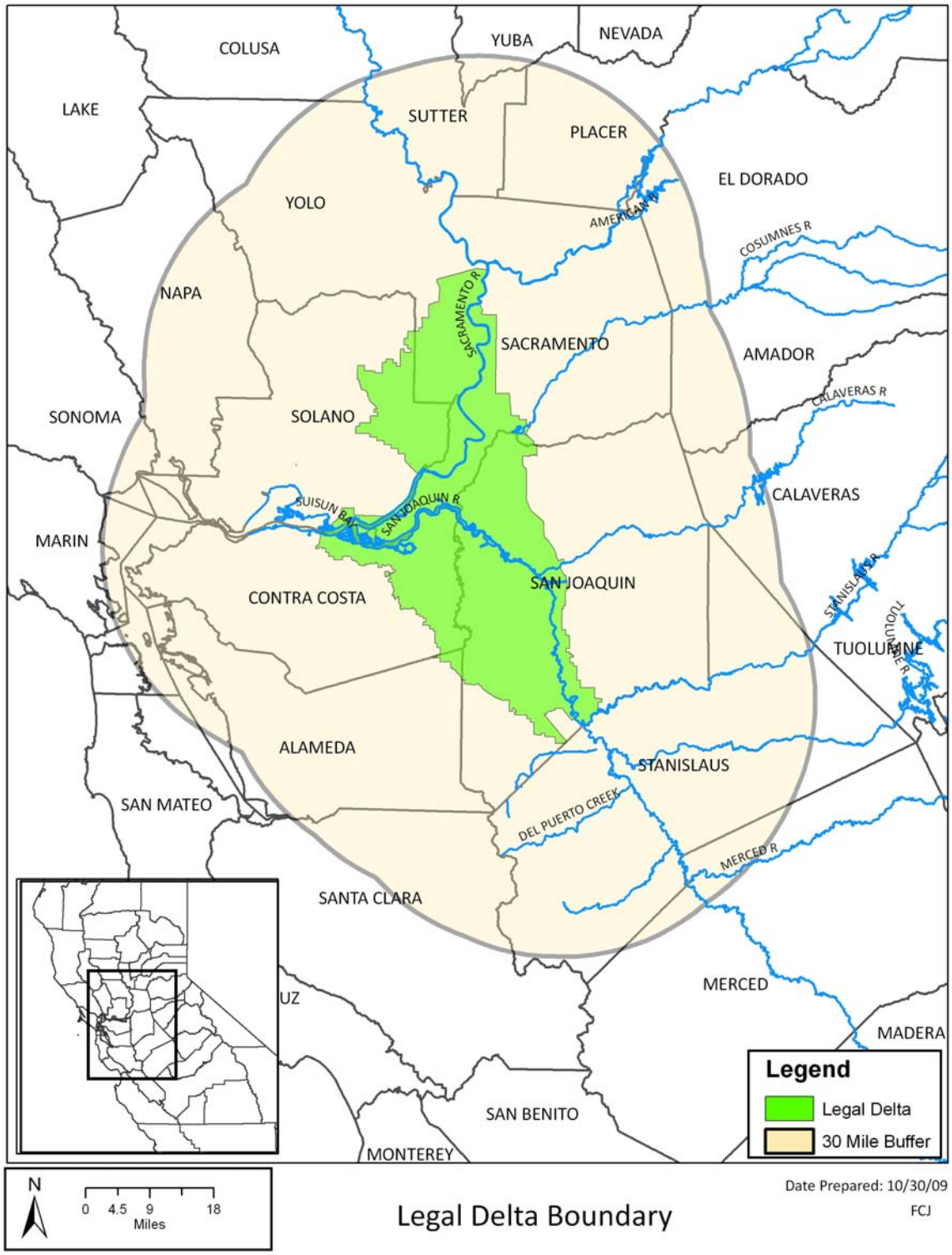
Development of the database used for the analyses occupied a substantial portion of the project's resources but resulted in the most comprehensive database of water quality results and toxicity data for the Delta for the POD years. The geographic scope of the data included the legal Delta +30 miles (Figure 3). The analyses primarily used data from 2000-2006 but if data from prior to 2000 were readily available, those were used. Many data sets (e.g. NPDES compliance data) were available only in hard copy or electronic form that did not allow manipulation. Some of these data sets were transferred into electronic format for manipulation. Originally, over 1 million data records, each with from 3 to 50 individual water quality results, were collected but review by the UC Davis team resulted in a reduction in the size of the data set. The data kept for the POD database included any water quality data with sufficient quality assurance/ quality control data to allow an evaluation of its significance in the POD (see Appendix I for a list of data sets). For example, many data sets contained numerous data entries of ND for non-detect but provided no detection or reporting limits or detection limits that were much higher than those used today. For numerous constituents such as pyrethroids, it is now known that LC_{50} values for various species are much lower (see for example, Amweg *et al.* 2006) than could be measured with detection limits employed in analyses over the last several years. Taken at face value, those entries would suggest that no chemical was present in the system and that pyrethroids could therefore not be a significant contributor to the POD. However, with no detection limits a determination of whether the chemical constituent was actually present was not possible. Many data sets contained sample site locations that were not able to be given map coordinates. In some instances, it was unclear if the data were from samples of effluent discharged to the Delta or ambient waters. Also, many analyte names were not clear, e.g. "unknown hydrocarbon" and it was not possible to identify the type of contaminant. Consequently, data records were excluded from the analysis if:

- They did not include Detection Limits or Reporting Limits

- They did not have an identifiable analyte name
- They did not have identifiable sample site location information as part of the data record
- Units of measure were not possible for the constituent, e.g. chlorpyrifos measured in seconds or nitrate measured in m^3.

The POD chemistry database is a relational database modeled after the Surface Water Ambient Monitoring Project (SWAMP) database format. Related data stored in separate tables can be queried to create a custom data report. For example, metadata (e.g. sample location, sample time, sample collector) for a water chemistry sample is kept in a single table and more detailed information describing the station, project, agencies, and lab results is kept in related sub-tables and LookUp lists. These data are combined through queries defined by the user. Toxicity data are kept in a non-relational toxicity database and may be exported to Excel for ease of viewing. The database is publically available as part of this report.

Figure 2. The legal Delta + 30 miles. The legal Delta is shown in green and the 30 mile buffer is the blue outline. The 30 miles extends into the San Francisco Bay drainage, but only data from water bodies that directly drained to the Delta was used in the analyses.



WATER CHEMISTRY DATA

HISTORICAL PERSPECTIVE

Fox and Archibald (1997) reported on all known water chemistry data on record for the Sacramento River system, the Delta, and the San Joaquin Valley. While too extensive to completely summarize here, there are several chemicals for which there are records of concentrations both before and during the POD. The reader is referred to that document for additional records of chemical concentrations in the vicinity of the Delta. A USGS study of pesticides in the Sacramento River found 7 pesticides in samples collected between 1991 and 1994 (Table 2), and a CVRWQCB study of storm water from Stockton and Sacramento found diazinon in elevated concentrations at several locations (Table 3). Diazinon was found at maximum concentrations that exceeded the current WQG of 0.10 µg/L.

Table 2. Water quality data from a USGS study in the Sacramento River conducted from 1991 – 1994. The original summary is Table 5 of Fox and Archibald (1997). Aquatic Life Benchmark data taken from the Office of Pesticide Programs (OPP) website http://www.epa.gov/oppefed1/ecorisk_ders/aquatic_life_benchmark.htm.

Pesticide	Number of Samples	Number of Detections	Maximum Concentration µg/L	Median Concentration µg/L	Current OPP Aquatic Life Benchmark - Plants[1] µg/L	Current OPP Aquatic Life Chronic Benchmark - Animals µg/L
Atrazine	563	75	0.238	0.016	1	60[2]
Carbofuran	603	139	0.109	0.007	-	0.75[2]
Diazinon	563	214	0.393	0.024	3,700	0.105[3]
Methidathion	563	72	0.212	0.020	-	0.66[2]
Molinate	603	79	1.553	0.213	220	340[2]
Simazine	563	236	0.522	0.75	36	960[3]
Thiobencarb	563	51	0.697	0.007	17	1[2]

[1]Nonvascular plant
[2]Invertebrate
[3]Fish

Table 3. Diazinon concentrations in storm water runoff from Sacramento and Stockton from data generated by V. Connor. Diazinon concentrations were determined by an ELISA test with a detection limit of 0.030 µg/L. The original summary is Table 26 of Fox and Archibald (1997).

City (Sample Date)	Site	Diazinon Concentration, µg/L
Sacramento (1/23/95)		
	Sump 104	>0.5, 1.050
	Sump 111	0.500, 0.450
	Strong Ranch Slough	0.410
	Chicken Ranch Slough	0.625
	Morrison Creek	>0.500, 0.340
	Elder Creek	>0.500, 1.100
	Arcade Creek	0.400
	RD 1000 Drain	0.160
	Natomas East Main Drain	0.260
Stockton (2/6/94, 2/7/94)		
	Mosher Slough	0.900, 0.630
	5 Mile Creek	1.000, >1,000
	Calaveras River	0.380, 0.450
	Mormon Slough	0.320, 0.900
	Lake McLeod	0.200, 0.500
	Turning Basin	0.190, 0.600

In Figure 18 of Fox and Archibald (1997), diazinon concentrations are provided for the San Joaquin River at Vernalis for the month of February, 1993. From February 8 to February 19, concentrations of diazinon in the river varied from a low of 0.15 µg/L to a maximum of approximately 1.10 µg/L and were accompanied by significant mortality in *C. dubia* toxicity tests. The USGS study of pesticides in the San Joaquin River also found numerous pesticides at relatively high concentrations (Table 4).

Table 4. Water quality data from a USGS study in the San Joaquin River conducted from 1991 – 1994. The original summary is Table 50 of Fox and Archibald (1997). Aquatic Life Benchmark data taken from the Office of Pesticide Programs (OPP) website http://www.epa.gov/oppefed1/ecorisk_ders/aquatic_life_benchmark.htm.

Pesticide	Number of Samples	Number of Detections	Maximum Concentration µg/L	Median Concentration µg/L	Current OPP Aquatic Life Benchmark - Plants[1] µg/L	Current OPP Aquatic Life Chronic Benchmark - Animals µg/L
Carbaryl	515	88	0.197	0.018	660	0.5[2]
Carbofuran	640	76	0.100	0.025	-	0.75[2]
Chlorpyrifos	640	40	0.043	0.009	140	0.015[3]
Cyanazine	192	59	0.803	0.150	-	-

Pesticide	Number of Samples	Number of Detections	Maximum Concentration µg/L	Median Concentration µg/L	Current OPP Aquatic Life Benchmark - Plants[1] µg/L	Current OPP Aquatic Life Chronic Benchmark - Animals µg/L
Dacthal	293	111	0.181	0.013	14,300	-
Diazinon	640	447	0.714	0.020	3,700	0.105[3]
Eptam	293	113	0.674	0.021	-	-
Methidathion	515	89	0.802	0.032	-	0.66[2]
Metolachlor	293	129	0.117	0.022	8	1[2]
Molinate	515	7	0.145	0.059	220	340[2]
Pebulate	347	6	1.046	0.458	230	-
Simazine	640	514	1.747	0.072	36	960[2]
Thiobencarb	640	43	0.528	0.011	1	17[2]

[1]Nonvascular plants
[2]Invertebrates
[3]Acute benchmark

All of these data indicate that there were significant quantities of pesticides in the Sacramento and San Joaquin Rivers and the Delta during the period from the late 1980s to the mid 1990s.

WATER CHEMISTRY - CURRENT

There are literally thousands of contaminants entering the Delta every day through discharges originating from the various industries, urban runoff, agricultural practices, and waste water treatment plants in and around the Delta. The initial database was developed using the data sources outlined in Appendix I. To narrow the list of contaminants for the initial review, a primary list of potentially important contaminants was developed from the Relative risk evaluation for pesticides used in the Central Valley Pesticides Basin Plan Amendment project area (Lu 2009, (http://www.waterboards.ca.gov/centralvalley/water_issues/tmdl/central_valley_projects/central_valley_pesticides/risk_evaluation/rre_stff_rpt_feb2009_final.pdf). This evaluation examined 28 high risk and 10 moderate risk pesticides used in the Sacramento River and San Joaquin River watersheds. This list of 38 was narrowed further by examining the database to determine if sufficient data existed to evaluate their role in the POD. Only 10 chemicals were considered to have sufficient data in the database to allow further analysis.

The chemicals initially considered for evaluation included chlorpyrifos, diazinon, diuron, bifenthrin, esfenvalerate/fenvalerate, lambda-cyhalothrin, permethrin, s-metolachlor, propanil, and copper. With the exception of the pyrethroids and copper, these are compounds with moderate to high solubility. The pyrethroids were included because of the recent evidence of their toxicity to aquatic life at extremely low concentrations (e.g. Amweg *et al.* 2006) and the recent increase in their use. Copper was included because of its extremely high use in the Central Valley and its potential adverse effects at several trophic levels. There were essentially no data available for s-metolachlor and propanil.

Data were examined to determine if these contaminants were present in the Delta in the late winter- early spring period at concentrations suspected to be capable of causing toxicity to POD species or prey items consumed by POD species. After the initial review indicated that few records were available for most of the pesticides listed above, the review was expanded to all water quality data available from 2000 to 2008. The data sets used are described in Table 2, Appendix I. The regulatory framework for water quality includes several regulatory targets including objectives, standards, maximum concentration limits, and limits. Each has a specific meaning and not all chemicals have objectives, standards, or limits. In addition, chemicals may also have benchmark values assigned by USEPA's Office of Pesticide Programs. Because there are many applicable categories, the data are described relative to a Water Quality Goal (WQG) which could be an objective, standard, or limit.

This review did examine the potential for non-pesticides (e.g. PCBs, PAHs) to cause the decline but these chemicals did not have sufficient data available to adequately evaluate their role in the POD. Water quality data for these constituents were available primarily through NPDES monitoring conducted by various permitees in the Delta. However, permits require little or no quality assurance/quality control data to be reported and the data were not sufficiently validated to be used. This report also does not include a review of the potential effects of ammonia or cyanobacteria as they are the subjects of other work currently underway.

Potential toxicity was based on the presence of chemicals at concentrations greater than the Water Quality Limits (action level) for aquatic life used in CVRWQCB regulatory programs (Table 5). In some instances, these are established values in the Basin Plan based on numerous scientific studies, in other instances they are 1/10 of selected LC_{50} values. Virtually no data are available for toxicity of contaminants in the Delta to the POD species so it was not possible to select 1/10 of LC_{50} values for POD species. Herbicides were compared to the currently available

Water Quality Goals which are generally 1/10 of the EC_{50} values for reduced algal growth. The use of these values represents the most conservative approach to determining if contaminants are present in sufficient concentrations to cause toxicity.

For the analysis, a cumulative frequency distribution of sample concentrations was developed. The distribution was generated based on the assumption that the concentrations in the database are a sample of all concentrations that could be obtained if a very large number of samples were collected. I.e. concentrations in the database were treated as a random sample of all possible concentrations and the sample data were used to generate a cumulative frequency distribution. All environmental concentrations were fit to a lognormal distribution as this has been established as an appropriate distribution to represent environmental data by both the US EPA (Fisher and Burton 2003) and the OECD (Wagner and Løkke 1991). If samples were listed as Non Detect in the database, a value of one-half the detection limit or the reporting limit was assigned as the concentration to allow a cumulative frequency distribution to be developed. In all cases, the analysis was limited to those samples whose reporting limit was below the Water Quality Goal (see description under chlorpyrifos and diazinon below). The cumulative frequency distribution was compared to the Water Quality Limit (Table 5) to estimate the proportion of samples from the Delta that would exceed that limit. This proportion was used as an indicator of the potential for contaminants to cause toxicity to POD species or their prey items.

Table 5. Water Quality Goals and half life for pesticides used in the cumulative frequency analysis.

Pesticide	Water Quality Goal	Half-Life
Diazinon	0.10 µg/L	70 hrs – 12 weeks
Chlorpyrifos	0.015 µg/L	3 – 4 weeks
Bifenthrin	None	NA
Lambda-cyhalothrin	None	NA
Permethrin	None	NA
Esfenvalerate/Fenvalerate	None	NA
Diuron	2 µg/L	6 weeks – 6 years
Dissolved Copper	2 µg/L	NA[1]

[1]Copper may complex with different ligands and reenter the dissolved form, but does not break down.

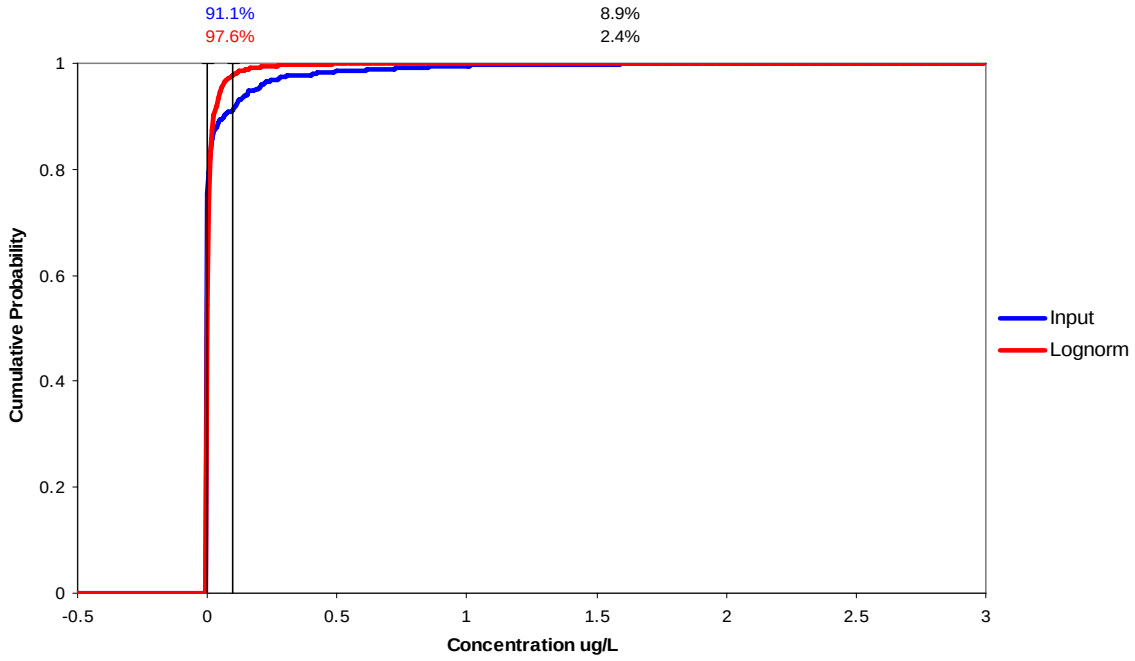
ORGANOPHOSPHOROUS PESTICIDES (OP) – CHLORPYRIFOS AND DIAZINON

Diazinon had the greatest number of samples of any pesticide examined for this review with 930 samples collected between 1999 and 2008 (not including laboratory Quality Control samples). Of those, 225 samples contained measureable concentrations of diazinon. Minimum detection limits (MDL) ranged from 0.003 µg/L to 0.05 µg/L for those years with no discernable pattern across time. Throughout the POD period, both low and elevated MDL were used depending on the sampling program.

The location of the stations from which samples were collected place the diazinon both inside and outside of the legal Delta. Using a radius of 30 miles from the Delta places many of the sampling locations in tributaries to the Sacramento River (e.g. Colusa Basin Drain, Gilsizer Slough), San Joaquin River (e.g. Orestimba Creek, Stevinson Lower Lateral, Pixley Slough), or to the Delta (e.g. Mokelumne River, Calaveras River, Duck Creek, Mosher Slough) itself. However, a few of the stations are from within the Delta in the Sacramento River, or from interior drain channels within the Delta islands (e.g. Drain to Grant Line Canal off of Wing Levee Rd, Terminous Tract @ Glascock Rd). Not all stations had measureable concentrations of diazinon, including many from the Delta islands. From within the Delta itself, only the water from the drain channel to Grant Line Canal off Wing Levee Rd had measureable amounts of diazinon. Flows in the water bodies outside of the Delta would be expected to move the diazinon to the Delta within a day, especially during the winter and spring period, but the actual fate and transport of diazinon is unknown and could vary considerably across the year. Further empirical studies or modeling of the system would be necessary to understand the travel time to the Delta and the concentration of diazinon reaching Delta waters. Movement of diazinon from the interior of the Delta islands to Delta waters is determined by the pumping of the water off of the islands. Obtaining pumping information was also beyond the scope of this project but an understanding of the concentration of diazinon in the drain channel when the pumps are activated is critical to understanding the load of diazinon (and all other pesticides) moved from the islands to the waters of the Delta.

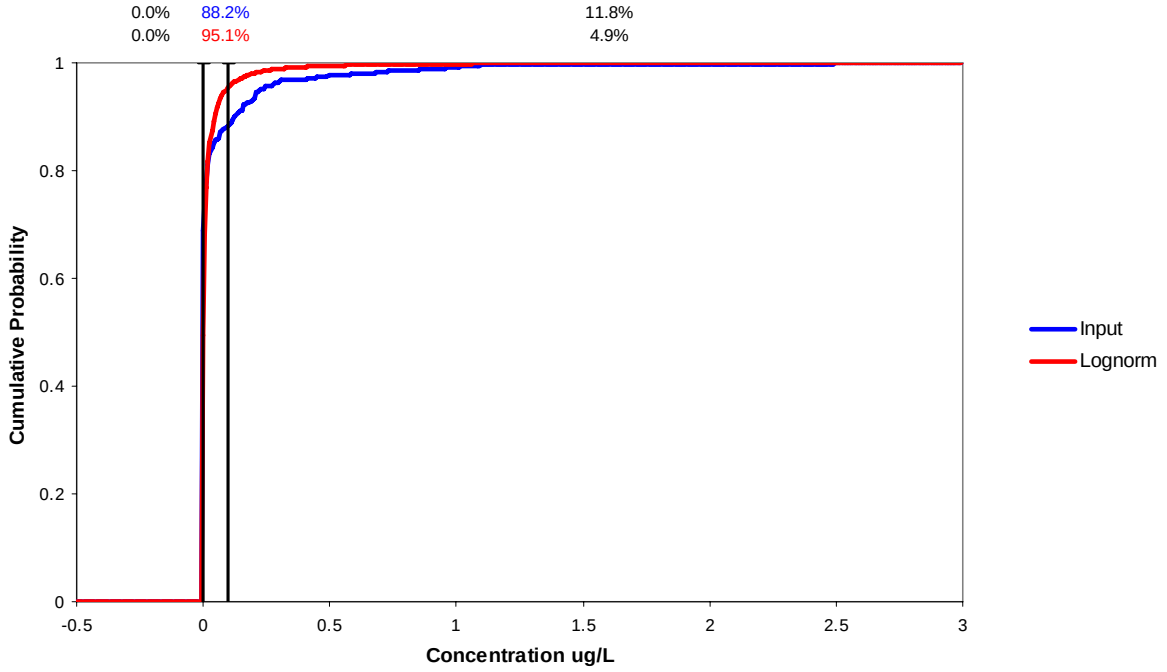
The Water Quality Goal for diazinon is 0.1 µg/L. The cumulative frequency distribution indicated that 8.9% of samples would be above the Water Quality Goal for diazinon (Figure 3). The long half life suggests that diazinon would remain toxic from the time runoff enters tributaries until it reached the Delta and once in the Delta it would remain toxic for the entire period of residence in the Delta.

Figure 3. Cumulative frequency distribution for concentration of diazinon in surface water from 2000-2008. The red line is the fitted lognormal distribution, and the blue line is the cumulative distribution for the data. The vertical line is a concentration of 0.1 µg/L, the current WQG. The numbers above the graph are the percentages of the distribution above and below 0.1 µg/L, i.e. 97.6% of all samples are expected to fall below 0.1 µg/L. The input distribution is the raw data and the lognorm distribution is a lognormal distribution fit to the data.



There were 527 data records for the months of January to June in the years 2000-2008. However, there were only 21 records for 2000-2002, of which 19 were non-detects. Consequently, a cumulative distribution function was generated for the entire 2000-2007 period (Figure 4). The two cumulative distribution functions are nearly identical. Including data from the entire year results in 9% of the samples exceeding the WQG and if the analysis is restricted to the January to June period, nearly 12% of the samples exceed the WQG.

Figure 4. Cumulative frequency distribution of diazinon concentration for water samples collected during the January to June period from 2000-2008. The vertical line to the right of 0 is the WQG of 0.10 µg/L.



There is also a large amount of monitoring data available for chlorpyrifos throughout the Valley, with 1212 records available for analysis (after excluding quality control analyses). However, only 948 samples were analyzed with MDLs sufficiently low to measure chlorpyrifos at the WQG of 0.015 µg/L. Chlorpyrifos was one of the few pesticides for which records were available for the 2000-2002 step decline period. The most elevated concentrations of chlorpyrifos were detected during the 1999-2000 period with lower concentrations detected in the 2005-2006 period. Between March 2002 and December 2002, numerous samples were collected but no chlorpyrifos was detected. The MDL for the analyses performed during 2002 were either 0.05 or 0.02 µg/L and all reporting limits (RL) were 0.05 µg/L, all above the Water Quality Goal of 0.015 µg/L. The MDL dropped to 0.00259 µg/L starting in early 2005 as the chlorpyrifos WQG dropped to 0.015 µg/L and analytical techniques improved dramatically to accommodate the need for measuring chlorpyrifos in samples at low concentrations. When the MDL were lowered, 48 of 323 samples (15%) had concentrations between 0.015 µg/L and 0.05 µg/L. If the same percentage is applied to the period between 1999 and 2005 when most MDL were 0.05 µg/L, an additional 15% of the samples could have had concentrations of chlorpyrifos between 0.015 µg/L and 0.05 µg/L. Assigning a value of one-half the detection limit for all values of non-detect when the detection limit was 0.05 would automatically place all samples above the WQG

of 0.015µg/L. While this technique is appropriate for generating non-zero concentrations when the detection limits are below the WQG, it is not appropriate when the detection limits are above the WQG as it inflates the percentage of samples expected to be above the Goal in the cumulative distribution function. Consequently, only samples analyzed with detection limits below the WQG were used in the cumulative distribution function analysis. Unfortunately, this limited the analysis to samples analyzed in 2005 or later and limits the usefulness of the results.

There were numerous records for chlorpyrifos from the January – June period for 2000-2008 suggesting that the pesticide was in the system during the period when exposure to the POD species was most likely to occur. The cumulative distribution functions for the entire data set and the samples collected from January to June were similar (Figures 5 and 7). Over the entire POD period, there was just over 8% probability of exceeding the WQG for chlorpyrifos (Figure 5). Because there are very few data from 1999-2002, when the analysis was restricted to 20003-2008, the probability of exceeding the WQG was also 8%. If the analysis is restricted to the months of January to June for the 2003-2008 period, only data are available for the years 2005-2008. During these years, there is just over a 5% probability that the concentration of a sample exceeds the WQG (Figure 7).

Figure 5. Cumulative frequency distribution for chlorpyrifos concentrations in the Delta 2000-2008. The vertical line to the right of 0 is WQG of 0.015 µg/L. In this case, based on the fitted lognormal distribution, 8% of the samples are expected to exceed the WQG.

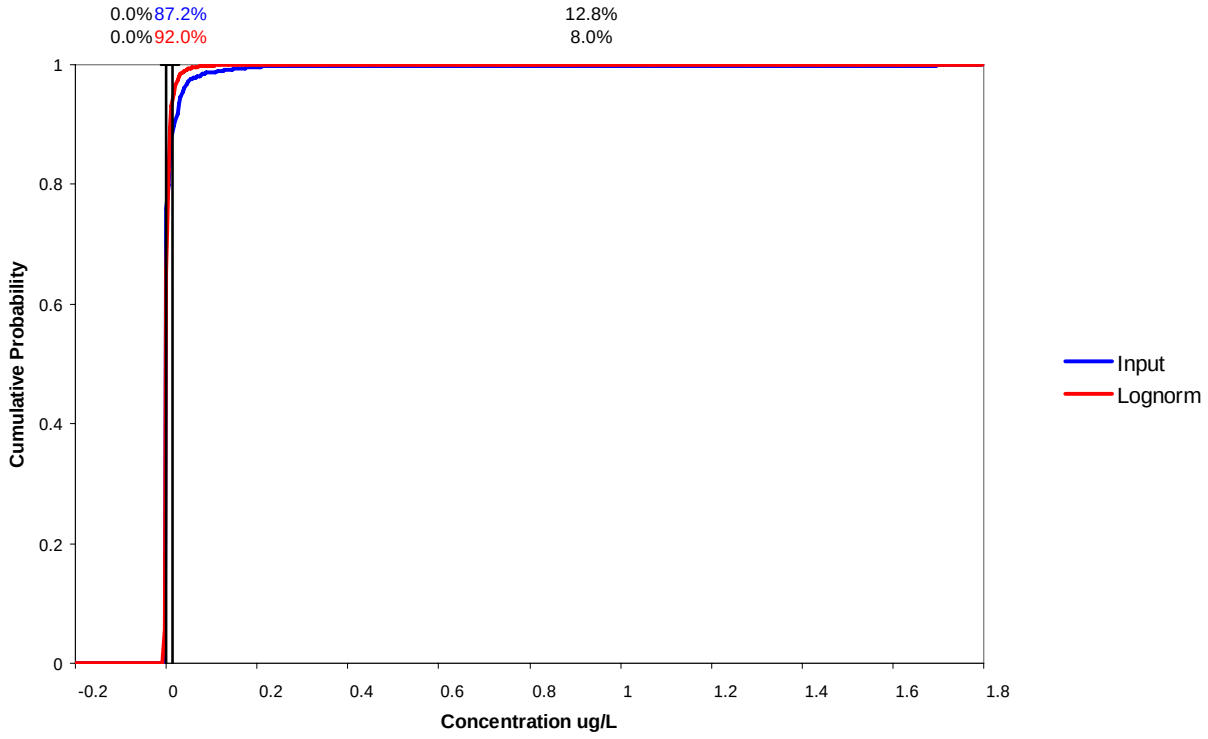
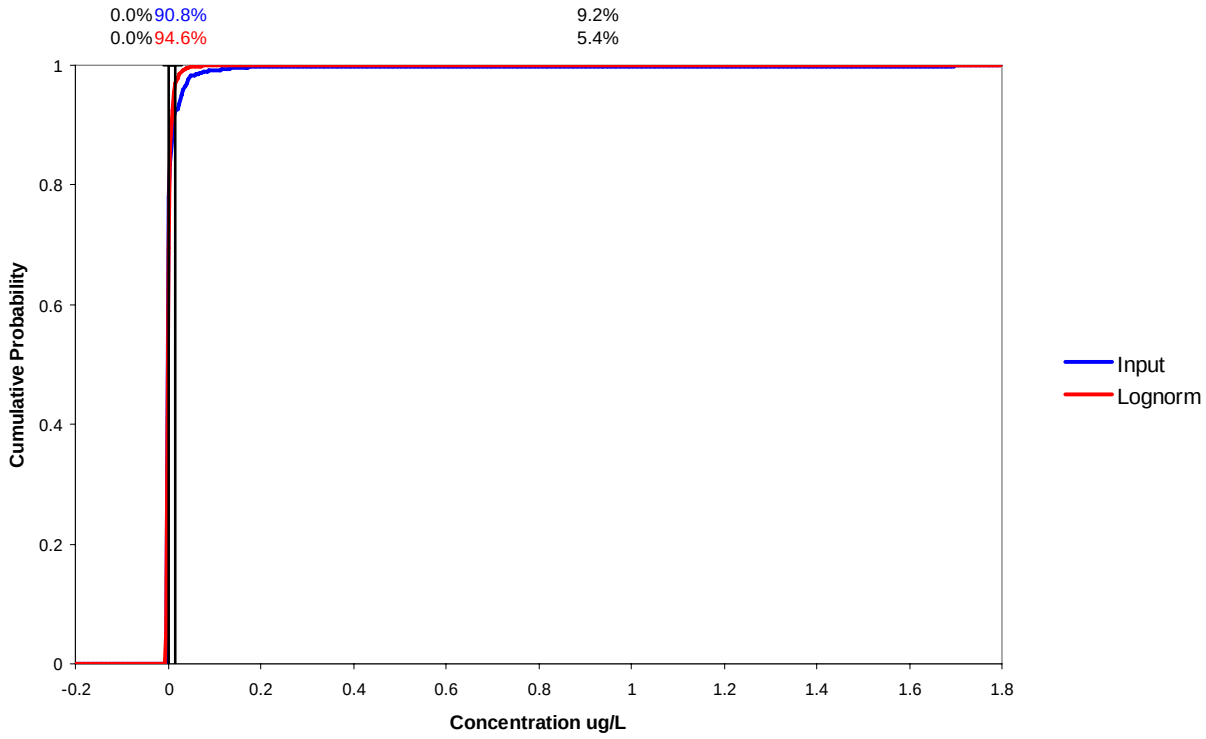


Figure 6. Cumulative frequency distribution for chlorpyrifos concentration in water samples collected from the Delta in the months of January to June 2003-2008. The vertical line to the right of 0 is the WQG of 0.015 µg/L.

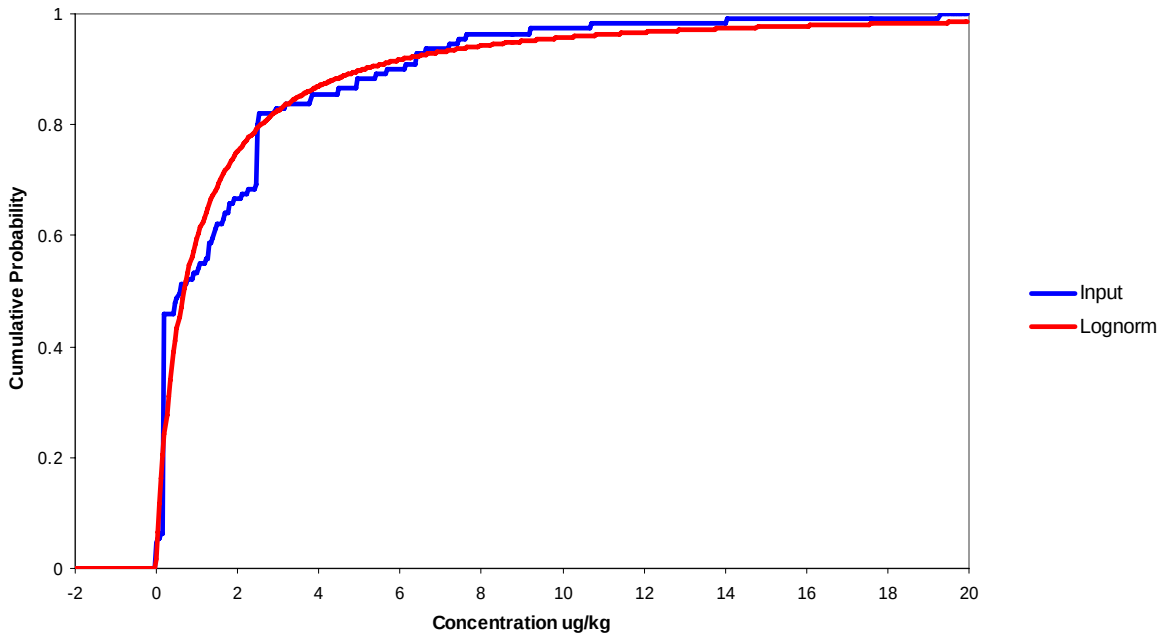


The locations of the sample sites are tributaries to the Sacramento River, San Joaquin River (e.g. Orestimba Creek), or to the Delta (e.g. Mokelumne River, Calaveras River, Duck Creek, Mosher Slough) and within the Delta. Flows in these water bodies would be expected to move the chlorpyrifos to the Delta within a day, but the actual fate and transport is unknown and could vary considerably across the year depending on flow. Similar to diazinon, empirical studies or modeling of the system would be necessary to understand the travel time to the Delta and the concentration of chlorpyrifos reaching Delta waters.

There were a significant number of samples analyzed for chlorpyrifos in sediment. The cumulative distribution function indicates that approximately 80% of the samples would have concentrations below a concentration of 2 µg/L, but the remaining 20% of the samples could have chlorpyrifos in sediments at concentrations reaching 20 µg/L (Figure 7). There is currently

no WQG for chlorpyrifos in sediment and it is difficult to place the results into a context where they can be interpreted. The LC_{50} for chlorpyrifos in sediment for *H. azteca* is 399 µg/kg and the LC_{50} for *Chironomous tentans* (a midge) is 383 µg/kg suggesting that the concentrations in sediment are not sufficiently elevated to cause toxicity to benthic organisms. D. Weston in studies of sediment toxicity has found that chlorpyrifos is rarely the cause of toxicity and that pyrethroids are generally more often the cause (http://ucce.ucdavis.edu/files/filelibrary/1598/34128.pdf). The relevance of chlorpyrifos in sediment to the POD is unknown because 1) the link between contaminated sediment and pelagic organisms is unclear, and 2) the locations from which the samples were collected were not in the Delta waterways but rather from tributaries to the Delta and the interior Delta island drain channels. Movement of sediment from the interior drain channels of the Delta islands or tributaries to the San Joaquin River to the Delta is questionable, but it is possible that a major storm event could result in the transport of sediment downstream to the Delta. Resuspension of sediments in the Delta could result in the movement of chlorpyrifos into the pelagic zone but it is not known how sediment-bound chlorpyrifos would impact pelagic organisms.

Figure 7. Cumulative frequency distribution of chlorpyrifos concentration in sediment. There are no vertical lines because there is no WQG for chlorpyrifos in sediment.



PYRETHROID PESTICIDES

Numerous pyrethroids were detected in the water and sediment during the POD years, but there were no results from 2000 to 2003. There were samples analyzed for lambda-cyhalothrin in 2003, and samples were analyzed for the remaining pyrethroids from 2004 through 2007. No comment was found in the database as to whether the samples were preserved with dichloromethane. If samples were not preserved, the concentrations found in the water or sediment could be lower than the concentration in the ambient media. As might be expected from the high K_{oc} values, most of the analyses were for pyrethroids in sediment. Although the relevance of sediment-bound pyrethroids to pelagic species is unknown, because of the recent focus and conclusions that pyrethroids could be responsible for the POD, data are presented for all common pyrethroids in the database. Water column data were available for several pyrethroids but the number of samples with measureable concentrations of pyrethroids in water is small. Recent work by Weston indicates that LC_{50}s for *H. azteca* in the water column range from 0.002 – 0.020 µg/L for several pyrethroids (Weston in press) indicating that previous water quality analyses for pyrethroids have been performed with detection limits above those necessary to detect potential toxicity.

BIFENTHRIN

Of all pyrethroids, the largest number of samples (563) were collected and analyzed for bifenthrin. The first water column sample was analyzed in July 2004, after the step decline period of the POD species. There were 8 samples with measureable concentrations of bifenthrin in the water column and only 2 of those were from the January to June period.

Sediment samples were primarily collected from numerous tributaries to the San Joaquin River. Of the 96 samples analyzed, 49 contained measureable concentrations of bifenthrin in the sediment. The largest concentrations detected were in 2005 and early 2006 with extremely high concentrations present in the sediment. However, all measurements were from a single water body, Del Puerto Creek.

PERMETHRIN

During the POD years, 547 water column samples were collected and analyzed for permethrin. Of those, only three water column samples contained measureable concentrations of permethrin; 0.036 µg/L in May 2005 from Kellogg Creek @ Highway 4, 0.023 µg/L in March 2005 from Marsh Creek @ Balfour Rd, and 0.006 µg/L in August 2007 from Ulatis Creek @ Brown Ave. All three sample locations are downstream of urban areas. An additional 62 sediment samples were analyzed for permethrin and 10 samples collected from 2003 to 2005 contained measureable amounts.

ESFENVALERATE/FENVALERATE

No water column samples analyzed for esfenvalerate/fenvalerate from 2000-2008. There were 49 environmental sediment samples analyzed for esfenvalerate/fenvalerate; of those 9 had measureable concentrations. All were collected in April 2003 or September and October 2004.

LAMBDA-CYHALOTHRIN

No water column data were available for lambda-cyhalothrin. From sediment, 49 samples were analyzed and 23 samples contained measureable concentrations of lambda-cyhalothrin. The 23 samples were collected on 6 dates from 2003 to 2005.

CYPERMETHRIN

No water column data were available for cypermethrin. From sediment, 44 samples were analyzed and 19 contained measureable concentrations of cypermethrin. All measureable samples were collected in 2004 and 2005.

DIURON

Water column data are available for 537 diuron samples collected between 2004 and 2008, of which 145 had measureable concentrations of diuron. Detections occurred in most months of the year, the exceptions being November and December. The most elevated concentrations

were associated with the winter-spring period with presumably greater runoff potential. There was a wide variance in observed concentrations from less than 1 µg/L to 180 µg/L. The WQG for diuron is 2 µg/L and the cumulative frequency distribution (Figure 8) indicates that only 7.4% of the samples are expected to be above the WQG. For the January to June period, 269 records were used in the cumulative frequency distribution analysis (Figure 9). A slightly greater probability of exceeding the 2 µg/L WQG occurs during the January to June period with 10% of the samples expected to exceed the WQG. The toxicity of diuron to invertebrates and vertebrates is low; consequently any effect of diuron on the POD species would be indirect through toxicity to phytoplankton and aquatic vegetation. There is a slight trend for increasing concentrations in the more recent years although the trend is not significant.

Figure 8. Cumulative frequency distribution of diuron in samples collected from 2004 – 2008. The vertical line to the right of the distribution functions is the WQG of 2.0 µg/L.

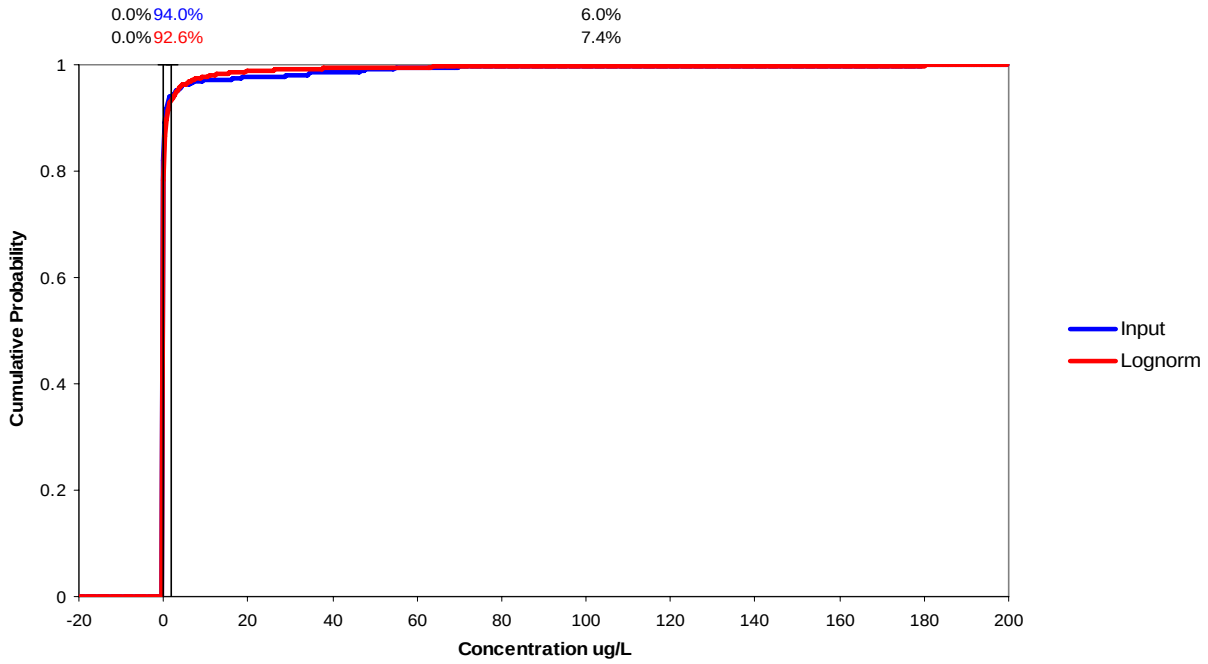
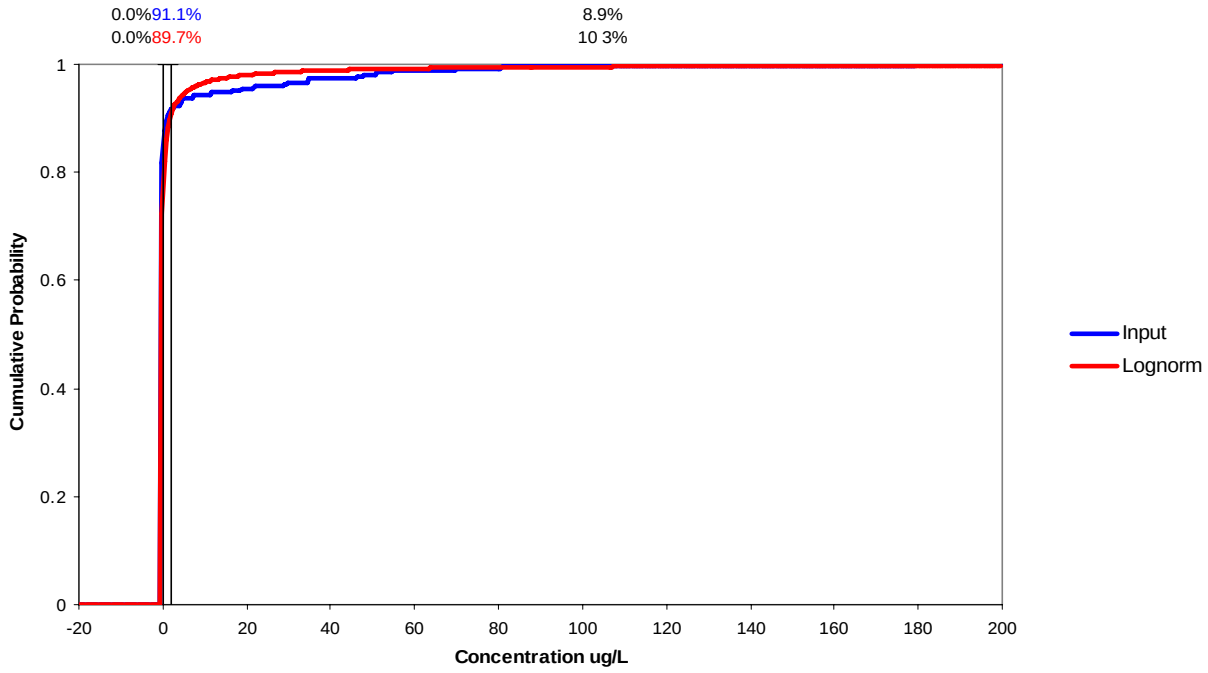


Figure 9. Cumulative frequency distribution for diuron for the January to June period. The vertical line to the right of the frequency distributions is the WQG of 2.0 µg/L.



COPPER

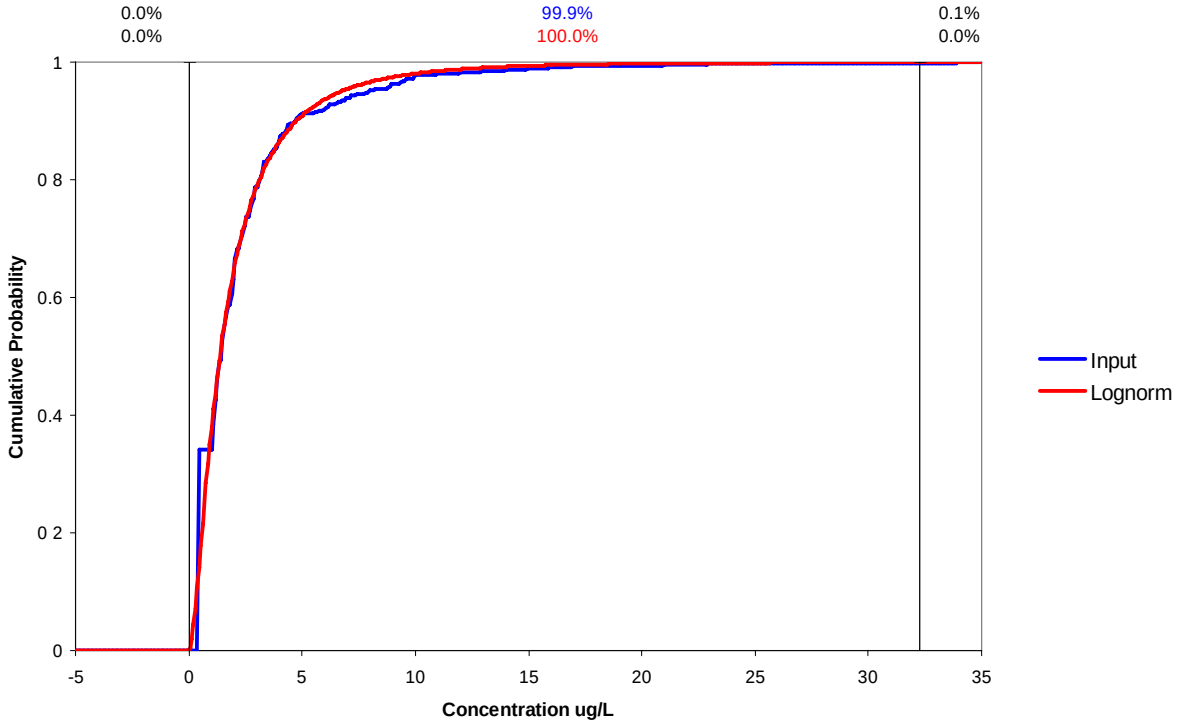
Copper is one of the most heavily applied pesticides in the Central Valley and there is a large amount of monitoring data for both total and dissolved copper available from 1995-2008. In addition, copper has been mined in the mountains around the Central Valley and could be entering surface waters from mine tailings. Copper was detected in every month of the year, although not in every month in every year. Total copper was found in concentrations reaching as high as 98 µg/L. Dissolved copper concentrations reached 34 µg/L. There are three entries in the database with concentrations of total copper above 39,800 µg/L, which were assumed to be in error. These data were from the Regional Board monitoring program and were obtained from sites UnknownRB5 141 (station code 535STC501), UnknownRB5 156 (station code 535STC030), and UnknownRB5 173 (station code 535STC509). Without the original data, it was impossible to confirm the values and they were excluded from consideration.

There were 716 data records for dissolved copper and the analysis focused on the dissolved form because it is the bioavailable form. Dissolved copper complexes with dissolved organic carbon and consequently, the toxicity of samples due to dissolved copper would depend on the organic carbon in the water at the time. Toxicity of dissolved copper also varies with hardness and pH. Unfortunately, there were very few data with a combination of dissolved copper, dissolved organic carbon (DOC), hardness, and pH and the toxicity of the dissolved copper was

adjusted for the average DOC, hardness, and pH during the POD period. There are limited measurements of DOC in the database and the average DOC during the POD years is 6.2 mg/L and all measurements were made during the January to June period. The average hardness for the POD years was 228 mg/L. Both hardness and DOC had relatively small ranges and the Coefficient of Variation for DOC was 70% and for hardness was 35% indicating that DOC tended to be about twice as variable as hardness. pH was the least variable of all parameters with a mean of 7.6 and a CV of 6.5%. A linear interpolation was performed to arrive at a criterion value to compare to the environmental concentrations and the cumulative frequency distribution. A pH of 7.5 was used, and the average hardness and DOC values from the Delta during the POD years were compared to the values in Appendix G of the EPA criteria document (US EPA 2007). Linear interpolations were performed for both the hardness criterion method and the Biotic Ligand Model (BLM) method and the lowest value was generated from the BLM. The interpolation resulted in a criterion of 32.75 µg/L dissolved copper. The values between which the interpolated value was developed were relatively close and even if a nonlinear interpolation would be more correct, the error introduced by using a linear interpolation is minimal.

The cumulative frequency distribution indicates that no samples are expected to fall above the WQG of 32.75 µg/L (Figure 11). Caution must be used when interpreting these results as several assumptions and data interpolations were used to derive the WQG. However, the results indicate that even if the WQG was dropped by one-half, there would be less than 1% of the samples with concentrations above the WQG.

Figure 10. Cumulative frequency distribution of dissolved copper in the Delta. The vertical line to the right side of the plot is the WQG of 32.75.



For the target chemicals discussed above, there are few data on which one can draw a conclusion about their role in the POD. Pyrethroids were not the focus of analyses until after the step decline period (2000-2002) and sufficiently low detection limits were not commonly used until 2005, precluding an understanding of their role during the early POD years. Diuron was also not the target of analyses until well into the POD years. Although there are more records for diazinon and chlorpyrifos, there are few data available for chlorpyrifos during the POD years, especially during the step decline period. The cumulative frequency distribution analysis suggests that a very large proportion of the samples in the Delta during the POD years were at concentrations above the current WQG. It is unknown if these concentrations would be found in the 2000-2002 period but one way to indirectly evaluate this hypothesis is to examine Pesticide Use Reports for the use of chlorpyrifos in the Sacramento River basin, the Delta, and the San Joaquin River basin to determine if applications remained constant through the POD period. Such an analysis is beyond the scope of this project. The largest amount of data is available for diazinon although few data are available during the January to June periods of each year, and those data that are available indicate that diazinon was found in toxic concentrations approximately 10% of the time. A thorough review of historic data for diazinon was beyond the scope of this project, but the high frequency of detection from the study in the

early 1990s suggests that diazinon was not a new contaminant in the system starting around the POD. However, it is not possible to draw any conclusions about the relative concentrations of diazinon in surface waters in and around the Delta before the POD years.

Because of the limited data for the target chemicals the database was examined for those chemicals for which data do exist for the POD period. All physical parameters (e.g. flow, velocity, temperature, specific conductance, turbidity) and miscellaneous non-chemical parameters (e.g. *E. coli*, *Streptococcus*, COD, all BOD, chlorophyll a and phaeophytin a) were removed from the database prior to analysis. During the POD period, 366 chemicals were measured from water column samples in 67,823 analyses. The chemicals included PAHs such as Benz(a)anthracene, legacy pesticides such as DDT and its breakdown products DDD, and DDE, metals and metalloids such as antimony, arsenic, and methyl mercury, halogenated compounds such as chloroform, and pesticides such as azinphos methyl and dimethoate. The greatest numbers of data records were for selenium (12,395), boron (11,872), copper (2,408), ammonia (2,081), phosphorus as P (1,642), ammonia as N (1,582), zinc (1,503), sulfate (1,472), chromium (1,282), diazinon (1,108), Total Kjeldahl Nitrogen (1,096), and chlorpyrifos (1,032). By comparison, 127 chemicals had 10 or fewer measurements. Numerous chemicals had as few as 5 measurements including several PCB congeners and PAHs. Attempts were made to evaluate the data for PAHs, chlorinated hydrocarbons, and PCB/dioxin compounds. However, the fraction from which the samples were analyzed (i.e. total, dissolved, total dissolved, total carbonaceous, particulate, total suspended, etc) was not recorded so it is not possible to determine if the PAHs were bioavailable. Data were available for fifty PCB congers (200 records total) but again, the fraction was not specified making the data unusable. Additionally, a large proportion of the data records for most constituents were non-detects leaving few data to analyze.

When the analysis focused on contaminants in sediment only 2471 records for 160 different contaminants were found. Bifenthrin, alpha chlordane, gamma chlordane, chlorpyrifos, DDD, DDE, DDT, dieldrin, total permethrin, permethrin-1, and permethrin-2 all had 37 data records. Fifty-five contaminants had 10 or fewer records. For most of the contaminants with the exception of the pyrethroids, most data records were non-detects.

To summarize the chemistry results, there are very few data records for the entire period of the POD period, and almost no records for chemical analyses during the 2000-2002 step decline years (Appendix I). No data are available for the pre-POD years and historical comparisons can be made only using reports from that period. Despite the inability to create frequency distributions for the pre-POD period, it appears that the concentrations of many chemicals were similar or greater during the pre-POD period compared to the POD period. Diazinon and

chlorpyrifos concentrations were similar in both periods, and many pesticides such as molinate and thiobencarb are found in much lower concentrations during the POD years than in the years immediately preceding the POD.

From 2000-2003, very few samples were analyzed with detection limits low enough to determine whether chemicals were present in the water. There were sufficient data available for diazinon, chlorpyrifos, and copper from the 2003-2008 POD period, and the analyses indicate that only a small percentage of samples would have diazinon or dissolved copper at concentrations expected to cause toxicity. However, between 5% and 10% of the chlorpyrifos samples were at toxic concentrations. Water column analyses were conducted across the year providing numerous samples during the January to June period. The results of these analyses are similar to those from the entire year. Dissolved copper was unlikely to have caused significant toxicity to POD species or their prey items. Chlorpyrifos was found at toxic concentrations in just over 5% of the samples during January to June, and diazinon was found in toxic concentrations in just under 5% of the samples.

To address the relative sensitivities of the POD and non-POD species, the USEPA ECOTOX database was queried for POD and non-POD species' LC_{50} values of several chemicals examined above including chlorpyrifos, diazinon, dieldrin, parathion, carbofuran, permethrin, esfenvalerate/fenvalerate, lambda-cyhalothrin, cypermethrin, and bifenthrin. Of this set of chemicals, data were available for striped bass only for chlorpyrifos, parathion, carbofuran, dieldrin, and two values for esfenvalerate (Table 11). All tests were from 1 to 4 days in length, although no attempt was made to review the individual studies. There are no toxicity data in the database for delta smelt, longfin smelt, or threadfin shad; there are data for striped bass for several chemicals. Consequently, there is no way to address the issue of differential sensitivity of threadfin shad and the rest of the POD species to chemicals present in the Delta during the POD that could account for the increase in abundance of the shad. Of the 5 chemicals for which data were examined, striped bass had the lowest LC_{50} value for chlorpyrifos and carbofuran and the highest value for dieldrin and esfenvalerate. A more thorough review of the sensitivities of various species is currently underway, and this analysis cannot be taken as definitive. However, currently there are no data to suggest that striped bass are more sensitive to contaminants than non-POD species. Their long life span and position at the top of the trophic web place them in a position to bioaccumulate lipophilic contaminants potentially causing problems with reproduction (see Histopathology section below).

Table 6. LC50 values for striped bass, a POD species, and four non-POD species that are found in the Delta in the same general habitats as the POD species.

Chemical	Striped bass LC_{50} µg/L	Largemouth bass LC_{50} µg/L	Bluegill sunfish LC_{50} µg/L	Green sunfish LC_{50} µg/L	Inland silverside LC_{50} µg/L
Chlorpyrifos	0.58	-	13.5	47.1	**7.2**
Parathion	1703	900	2568	1048	-
Carbofuran	207	2021	595	213	-
Dieldrin	227	6	41	116	-
Esfenvalerate	**2.17**	-	**0.39**	-	-

These data along with the cumulative frequency distributions above suggest that although toxicity to fish species could occur, it is likely to be rare. Toxicity to prey species could be more frequent.

Conclusions

- Chemicals are not found in higher concentrations during the POD years compared to the pre-POD years.
- Very little data exist to determine if higher concentrations of chemicals occurred during the 2000-2002 step decline period compared to the later POD period.
- There are too few data to adequately address the January to June concentrations of chemicals with a few exceptions. Of those exceptions, chlorpyrifos occurred in toxic concentrations in over 5% of the samples collected and diazinon occurred in toxic concentrations in just under 5% of the samples.
- There are no toxicity data available to determine if the threadfin shad is relatively less sensitive to chemicals present in the Delta, and the question of why the threadfin shad is increasing in numbers while other POD species are declining is not able to be addressed with chemical data.
- The cursory review of the relative sensitivity of POD and non-POD species to various chemicals found in the Delta during the POD years does not suggest that POD species are more sensitive. However a more detailed review is being undertaken and the

conclusion is preliminary. Striped bass are much more sensitive to chlorpyrifos than the non-POD species listed in Table 6, and chlorpyrifos was the one chemical that experienced exceedances above 5%.

- Direct toxicity to POD species is unlikely, but toxicity to prey items could occur given the analysis of the limited data available.

TOXICITY DATA SETS – HISTORICAL PERSPECTIVE

An understanding of the levels of toxicity in the pre-POD years compared to the POD years allows an understanding of whether toxicity increased sufficiently during the POD years to indicate a role for contaminants in the POD decline. If increased levels of contaminants in the Delta and increased levels of toxicity were responsible for the decline, it would be expected that the levels of toxicity would be greater during the POD years compared to the pre-POD years. A historical perspective of toxicity testing, water chemistry, and histopathology data from 1965 to 1996 was provided by Fox and Archibald (1997) who reviewed a number of reports produced between the 1960s and the 1990s by various federal, state and local agencies and the University of California. Although some of the literature cited is in peer-reviewed journals, the majority of these reports are no longer readily available. The Fox and Archibald (1997) review provides a baseline for comparison with the results of toxicity monitoring programs conducted from 2000 to the present. The Fox and Archibald (1997) review did not address the levels of toxicity experienced in the tests but did report the percentages of tests with significant toxicity.

Using statistics provided by the California Department of Fish and Game, Fox and Archibald (1997) found that fish kills in the Central Valley had declined from the 1960s to 1990s. In the 1960s, tens of thousands of fish were killed by pesticides on an almost yearly basis. Many pesticides were listed as unknown, but other pesticides responsible for the fish kills include DDT, toxaphene, parathion, xylene, chlorazine, disyston, acrolein, hydrothal, thiodan, chloradane, azinphos-methyl, molinate, copper sulfate, metam-sodium, carbofuran, and endosulfan. By the 1990s, the maximum number of fish killed as a result of exposure to pesticides was around 200 in the entire Sacramento and San Joaquin River basins in a few years but generally no fish were reported killed as a result of exposure to pesticides. The most recent largest fish kills occurred in 1991 with 1000+ and 7000 fish reported killed in the Sacramento River basin and the San Joaquin River basin respectively (Table ES-1 of Fox and Archibald 1997). The major 1991 Sacramento River fish kill was associated with the Cantara Loop train derailment that spilled metam-sodium into the upper Sacramento River and the San Joaquin River fish kills involved 4000 crayfish and 3000 fish deaths from carbofuran in San Joaquin County.

Toxicity of water samples to all test species was relatively high during all years for which data were available (Table 6). Although not always available, the data indicate that the percentage of toxic tests was higher during the January to June period than during July to December of the

tested years. Sediment toxicity testing was infrequent but toxicity was found in areas near Sacramento including an urban sump in Sacramento and Sacramento Slough. In the San Joaquin River basin, sediment was toxic to *H. azteca* in samples collected at TID Lateral #5 (Harding Drain) and Orestimba Creek, and toxic to *C. dubia* in samples from Mud Slough and Orestimba Creek. Fox and Archibald (1997) noted that most of the toxicity was found in back sloughs and small upland drainages and toxicity was more frequent during rainfall events. Between May 1991 and April 1994, the USGS conducted daily monitoring for 21 pesticides in the Sacramento River found simazine, diazinon, and carbofuran present in 42%, 38%, and 23% of the samples respectively. Other pesticides found included molinate, thiobencarb, and methidathion. None except for diazinon were found in concentrations expected to be toxic to aquatic life. Simazine and diazinon were found in daily samples collected at the San Joaquin River at Vernalis in 70% and 80% of the samples respectively, and metolachlor, dacthal, eptam, and cyanazine were detected in between 30 and 45% of the samples.

In the Delta, toxicity to *P. promelas* and *C. dubia* did occur, but at a lower level compared to the Sacramento or San Joaquin River sites. There were also lesions in the livers of up to 30% of striped bass larvae collected between 1988 and 1990. Mortality to *C. dubia* was attributed to OP and carbamate pesticides including chlorpyrifos, diazinon, and carbofuran according to Toxicity Identification Evaluations (TIE) (USEPA1989a, 1989b, 1991, 1992, 1993a, 1993b, Norberg-King *et al.* 2005). It was not noted if the results of the TIE analyses were confirmed by chemical analysis.

Table 7. Summary of results presented in review of toxicity and water chemistry results performed by Fox and Archibald (1997). Additional data are provided in Fox and Archibald.

Location	Years	Species	% of Acutely Toxic Tests	% of Toxic Tests (January – June)
Sacramento River basin (40 sites)	1988 - 94	*P. promelas*	15	18.8
Sacramento River basin (40 sites)	1988 - 94	*C. dubia*	27	32.1 (35.3 from April to June)[1]
Sacramento River basin (40 sites)	1988 - 94	*S. capricornutum*	17.9	9.7
San Joaquin River basin	1988 – 92	*P. promelas*	18	NA
San Joaquin River basin	1988 – 92	C. dubia	27	26 (21 from April to June)
Delta	1987	P. promelas	30 (100% of tests exhibited reduced growth)	NA
Delta	1988	P. promelas	8	NA
Delta	1991 - 95	P. promelas	0	NA
Lower Sacramento River	1987 - 92	*C. dubia*	20	NA
Lower Sacramento River	1994 - 95	*C. dubia*	9	NA
Sacramento River at Freeport Marina	1990 - 96	*P. promelas*	50	NA
Colusa Drain, Sacramento Slough, Butte Slough	Mid 1980s	*C. dubia*	57	NA
Colusa Drain	1989	*Neomysis*	27 - 78	NA

Location	Years	Species	% of Acutely Toxic Tests	% of Toxic Tests (January – June)
Colusa Basin Drain	1988-90	*M. saxatilis* larvae	67 - 100	NA
Colusa Basin Drain	1991	*M. saxatilis* larvae	40	NA
Colusa Basin Drain	1992	*M. saxatilis* larvae	12	NA
Sacramento River at Colusa, Walnut Grove, and Rio Vista	1988-89	*M. saxatilis* larvae	13 - 50	NA

[1]The percentage of toxic samples in the April to June period varies across year and individual study with toxicity to *C. dubia* as high as 88% occurring in 1989.
NA – no data available for this specific time period

Fox and Archibald (1997) concluded that toxicity was ubiquitous and that pesticides such as atrazine, simazine, diuron, carbofuran, diazinon, chlorpyrifos, methidathion, molinate, thiobencarb, and methyl parathion were commonly found in samples collected throughout the Central Valley at concentrations sufficient to cause toxicity. Results of the TIEs indicated that these same compounds were responsible for toxicity observed in the samples. In addition, tissue analyses of striped bass livers found organochlorine compounds such as DDT and arachlor 1260 throughout the 1980s and into the early 1990s.

The Fox and Archibald (1997) review clearly establishes that for the years in which data are available, there were a substantial percentage of acutely toxic samples in the major rivers in and around the Delta, primarily in the April – June period, as well as frequent detections of pesticides in the water at concentrations known to be toxic to aquatic life, and the presence of organochlorine compounds in fish tissue. Toxicity occurred to a variety of species including striped bass, one of the POD species.

POD YEARS SINGLE-SPECIES TOXICITY TESTS - BACKGROUND

Based on the stated goals of the Clean Water Act (CWA), the EPA and individual States implement three approaches to protect water quality. These approaches include chemical-specific control, toxicity testing control, and biological criteria/bioassessments (USEPA 1991). Each of the three control approaches has advantages and limitations.

The chemical-specific approach involves the development of water quality criteria (WQC) for chemicals as expressed in terms of the acute criterion and the chronic criterion. These criteria are developed following EPA water quality guidelines. EPA has developed water quality criteria for the 126 priority pollutants as required under CWA Section 308. These WQC are based on minimum data requirements that include both acute and chronic toxicity tests with the specified numbers and types of aquatic species. WQC are intended to protect most of the tested species, most of the time. The chemical-specific approach can allow prediction of

ecological impacts before they occur. It also considers bioaccumulation and human health impacts. A limitation of the chemical-specific approach is that not all toxicants in wastewaters or aqueous samples may be known, and therefore, control requirements can only be established for those that are known. For mixtures of chemicals with unknown interactions or for chemicals having no chemical-specific criteria, sole use of chemical-specific criteria to safeguard aquatic resources would not be protective.

Toxicity testing is needed because the chemical-specific approach only addresses individual chemicals and does not address chemical interactions or chemicals that are not known to be in the effluent. In addition, criteria have been developed for only a limited universe of chemicals. This is why the toxicity testing and bioassessment approaches for protecting aquatic life are also critical components for protection of aquatic resources. The primary advantage of using the toxicity testing approach is that this tool can be used to assess toxic effects (acute and chronic) of all the chemicals in aqueous samples of effluent, receiving water, or stormwater. This allows the effect of the aqueous mixture to be evaluated, rather than the toxic responses to individual chemicals. Some advantages of WET testing include the toxicity of effluent or ambient water is measured directly for the species tested; the aggregate toxicity of all constituents in a complex effluent is measured; and ecological impacts can be predicted before they occur. Toxicity tests can be used to assess ambient waterbodies (i.e., receiving water) making these tools effective in the assessment of small and large watersheds (de Vlaming et al. 2000). This has been demonstrated by the State of California which has successfully used an ambient toxicity testing approach to identify and regulate frequently occurring toxic chemicals. This approach includes pinpointing critical sampling locations for collecting the ambient waters to be assessed using acute and chronic toxicity tests. If toxicity is detected, then additional samples are collected to determine the spatial and temporal toxicity patterns. Subsequently, EPA's Toxicity Identification Evaluation (TIE) procedures are used to identify the causative toxicant(s). The goal of the TIE is to identify the chemical(s) causing toxicity in an aqueous sample. This ambient toxicity testing approach has led to the 303(d) listing of chemicals beyond the 126 priority pollutants commonly tested; one such listing is the pesticide diazinon, which is not a priority pollutant (SWRCB 2003). In addition, the approach of toxicity testing in conjunction with TIE analysis may be used to determine chemical interactions. These interactions can be additive, synergistic, or antagonistic. Lydy et al. (2004) provides a synthesis review of challenges in regulating pesticide mixtures and pesticide toxicity to aquatic organisms. Limitations of WET are that it directly measures only the immediate bioavailability of a toxicant(s) in the aqueous sample, and the long-term cumulative toxicity of a compound is not measured.

Acute toxicity tests are used to determine the concentration of effluent or ambient water that results in mortality within a group of test organisms during a 24-, 48- or 96-hour exposure. In an acute toxicity test, an effluent sample is collected, diluted, and placed in test chambers with the chosen test species. After 24, 48 or 96 hours, the number of live organisms remaining in each test concentration and in a control is recorded. The standard EPA acute test methods for freshwater and estuarine/marine invertebrate and vertebrates are delineated in USEPA (2002a).

A chronic toxicity test is defined as a short-term test in which sublethal effects, such as fertilization, growth or reproduction, are measured in addition to lethality (in some tests). Traditionally, chronic tests are full life-cycle tests or shortened tests (approximately 30 days) known as early life stage tests. Measuring the chronic toxicity of effluents is difficult because of the potential for effluent toxicity to change over time. Thus, even a shortened chronic early life stage test conducted in one month would have to be repeated at intervals to ensure that process or receiving water changes were not altering toxicity in ambient waters. In addition, toxicity spikes occurring during any one portion of a 30-day test could produce a different level of toxic response than an identical spike occurring during a different time of the test. The duration of chronic toxicity tests precludes the use of a single effluent sample due to probable reduction in toxicity with storage and requires extensive logistical arrangements for sampling and handling of effluent. Chronic toxicity test methods of 7 days duration require a minimum of three samples. As a result of such considerations EPA has developed a suite of shorter toxicity tests (short-term chronic tests) that aim to detect toxicity at chemical concentrations near those that produce chronic toxicity in longer term tests. The short-term chronic tests were developed and selected based on characteristics such as sensitive species, sensitive life-stages and endpoints, taxonomic and ecological diversity, short duration, availability of organisms for testing, and low volume requirements for test solutions. These resulting tests have typical durations of 40 minutes to 9 days, enabling tests to be run with effluent or receiving water samples at lower costs and increased test frequency. The chronic test methods are delineated in USEPA test methods (USEPA 1995, 2002b, 2002c).

Monitoring in freshwater ecosystems typically employs EPA three-species toxicity tests with freshwater algae (*Selenastrum capricornutum*), the copepod (*Ceriodaphnia dubia*), and the fathead minnow (*Pimephales promelas*) (USEPA 2002b). There are numerous advantages in using established WET test species for ambient monitoring including well understood life

history and husbandry of the test organism, and established test protocols with a robust statistical basis for endpoint interpretation. Depending on site-specific water quality conditions, it may be appropriate to utilize other species. For example, standard WET species may not tolerate high TDS waters characteristic of some ambient and storm waters. In cases where water quality characteristics are not compatible with standard test species, the permitting authority should use best scientific judgment within local and state agencies and EPA to select alternate species and/or testing approaches.

For testing of estuarine environments, EPA has published short-term chronic toxicity test methods for several West Coast species which could be used for environmental monitoring in estuarine and marine environments (USEPA 1995). The estuarine species include topsmelt (*Atherinops affinis*) and mysid (*Holmesimysis costata*). For testing marine waters, protocols for Pacific oyster (*Crassostrea gigas*), mussel (*Mytilus* sp.), red abalone (*Haliotis rufescens*), giant kelp (*Macrocystis pyrifera*), sea urchin (*Strongylocentrotus purpuratus*), and sand dollar (*Dendraster excentricus*) are available. Monitoring programs may be conducted in areas that contain species of special concern. EPA has provided guidance on selection of standard test organisms that would predict responses of species that are threatened or endangered (USEPA 2003).

The same USEPA WET test methods are used as the primary tool for stormwater and ambient monitoring, the conditions under which the procedures are used are generally different from monitoring continuous effluent discharges. Procedures which should be considered include:

- Experimental design – sample collection location, single vs. multiple concentrations
- Sampling – frequency, volume, container material, holding time
- Toxicity test method – organism selection, renewal frequency

Toxicity testing procedures that are typically used in WET testing NDPES compliance, coupled with other biological assessments, have become increasingly important tools for identification of waterbodies which fail to meet goals of the CWA. In general the same organisms, testing protocols and sampling methods used in WET testing can be used in stormwater and ambient

water monitoring. However, stormwater and ambient water study designs may need to incorporate different test organisms and sampling strategies to meet the goals of the study.

Stormwater monitoring for toxicity is really a special case of effluent monitoring, the main difference being that stormwater is episodic. There are special conditions associated with stormwater monitoring in cities and towns where collected stormwater is conveyed through separate storm sewer systems or through combined sewers to a treatment plant prior to discharge. In most cases, stormwater is directly discharged to the receiving system without treatment. Ultimately, a successful stormwater program minimizes the level of contaminants in the stormwater. The most severe receiving water problems due to wet weather flows are likely associated with chronic exposures to contaminated sediment and to habitat destruction.

The receiving waters of either an effluent or stormwater discharge are monitored to achieve a greater understanding of the potential effects of the discharge. Standard effluent monitoring tools, such as toxicity testing and water chemistry are used gather data on receiving water impacts, but other tools include in situ toxicity tests, bioassessments, and sediment toxicity testing. The experimental design of the ambient monitoring study will be based on the study questions and the tools that are chosen. Water column toxicity tests will pick up more ephemeral toxicity, and therefore should be used in fewer places, but perhaps more often. In situ water column toxicity tests can integrate toxicity over time, and could probably be used more sparingly, at least temporally. Sediment acts as a sink for many chemicals, particularly hydrophobic contaminants, and sediment toxicity testing tends to monitor the potential for longer term effects. Sediment toxicity tests could be used less often temporally, but over a wider spatial range.

REVIEW OF TOXICITY TESTING RESULTS, 2000-2008

Data were assembled from the sources listed in Appendix I. Maps for the various programs discussed in this section are provided in Appendix II. In some programs, such as the Irrigated Lands Regulatory Program (ILRP), very little sampling was conducted in Delta waters directly. Monitoring was performed occasionally on the major tributary rivers to the San Joaquin River or Delta (e.g. Mokelumne, Stanislaus Rivers), and primarily on tributaries to the major rivers entering the Delta (e.g. Calaveras River, Mokelumne River), small creeks and sloughs entering the Delta (e.g. Lone Tree Creek, French Camp Slough), and agricultural drains within Delta Islands (e.g. Roberts Island, Terminous Tract). It is assumed that all of these waters do reach

the Delta reasonably close to the time they were sampled for toxicity. If toxicity was a result of the chemicals discussed above, the half-lives of the chemicals would be sufficiently long to continue to cause toxicity. Some data are from ambient sampling from NPDES facilities discharging into the Delta and Delta tributaries. For both of these types of programs, the amount of dilution of the water entering the Delta is unknown and it is not known if the toxicity would remain. Finally, some monitoring occurred in Delta waters directly as part of the POD monitoring program. The following chapter summarizes results of these data sets. A general summary of available monitoring data is presented first followed by results from each monitoring program. Monitoring data were not available for all programs and consequently data are summarized by program as well.

From October 2001 to January 2008 data were available for 2522 toxicity tests performed on samples collected from several programs (Irrigated Lands Regulatory Program, Sacramento River Watershed Program, Interagency Ecological Program, Surface Water Ambient Monitoring Program, POD Toxicity Monitoring Program, Sediment Quality Objectives Program, and NPDES monitoring programs, see Individual Monitoring Programs below for a description of each) from water bodies that drain to the Delta, San Joaquin River or the Suisun Bay – San Pablo Bay area (Table 7). The results indicated low levels of toxicity compared to the toxicity experienced in the 1980s and 1990s (Table 2) with the exception of sediment where almost one-half of the tests performed resulted in toxicity. Toxicity results were broken down into 3 categories; significance not applicable to sample, statistically significant at $p = 0.05$ but survival above the threshold, and statistically significant at $p = 0.05$ and survival below the threshold. The meaning of the first category is not known because the database contains no notes to explain the designation, but this category includes only two tests and both tests were performed out of hold time according to notes in the database. Tests in the second category may not be biologically relevant because although the test is statistically significant, survival of the test organisms is above a threshold, usually 80%. The threshold has been used by programs such as the Surface Water Ambient Monitoring Program to establish a biologically meaningful reduction in survival as opposed to a statistically significant reduction that may be the result of complete survival in the controls and only limited mortality in the treatments. The third category is considered both statistically and biologically significant. For *C. dubia*, all tests were statistically significant and below the evaluation threshold although 5 test results were not formally compared to the evaluation threshold when originally entered into the database. For *P. promelas*, 7 tests were statistically significant and above the threshold and 8 tests were statistically significant and below the threshold. For *H. azteca*, 2 test results were in the first category, 88 in the second category, and 399 in the latter category.

Table 8. Summary of test results for toxicity testing in the Sacramento River basin, Delta, and San Joaquin River basin for 2001-2008. Category III tests are considered to be both statistically and biologically significant. The summary does not include test results from the Pelagic Organism Decline (POD) Ambient Toxicity Monitoring Project as those data are not available.

Test Organism	Test Matrix	Number of Tests	Number Toxic	Percent Toxic	Number of Category III tests	Percentage of Category III tests
Ceriodaphnia dubia	Water	922	46	5	46	5
Pimephales promelas	Water	612	15	2.5	8	1
Hyalella azteca	Sediment	1001	489	49	399	40

Table 9. Summary of test results for toxicity testing in the Sacramento River basin, Delta, and San Joaquin River basin for 2001-2002, and 2003 – 2008 for the January to June period. All tests were performed between January and June. No tests in 2001 were performed during the January to June period.

Years	Test Organism	Test Matrix	Number of tests	Number toxic	Percent Toxic
2002	*Ceriodaphnia dubia*	Water	50	0	0
2002	*Pimephales promelas*	Water	20	0	0
2002	*Hyalella azteca*	Sediment	120	8	7
2003 – 2008	*Ceriodaphnia dubia*	Water	852	71	8
2003 – 2008	*Pimephales promelas*	Water	572	15	3
2003 – 2008	*Hyalella azteca*	Sediment	665	385	58

Table 10. Toxicity test results on a year by year basis for the San Joaquin River and Delta for the 2002-2008 period. All toxicity testing on samples collected in 2001 resulted in no toxicity (see text). Results are not shown for tests on samples collected outside the Delta. All test on *P. promelas* and *C. dubia* were performed on water column samples; all tests on *H. azteca* were performed on sediment samples. See text for details.

Year	Months	Test Organism	Number of Tests	Number Toxic	Percent Toxic
2002	April - June	*P. promelas*	20	0	0
2002	April - June	*C. dubia*	50	0	0
2002	July - Dec	*H. azteca*	144	64	57
2003	Jan – June	*C. dubia*	40	1	2.5
2003	Jan - June	*H. azteca*	112	72	64
2003	July – Dec	*C. dubia*	81	4	5
2004	July – Oct	*P. promelas*	48	0	0
2004	Jul - Oct	*C. dubia*	50	2	4
2004	Aug – Nov	*H. azteca*	216	153	71
2005	Jan - June	*P. promelas*	146	3	2
2005	Jan – June	*C. dubia*	153	11	7
2005	Jan – June	*H. azteca*	137	37	27
2006	Jan - June	*P. promelas*	65	2[1]	3
2006	Jan – June	*C. dubia*	72	11	15
2006	Jan – June	*H. azteca*	14	8	57
2006	Jul – Dec	*P. promelas*	45	0	0
2006	Jul – Dec	*C. dubia*	48	3	6
2006	Jul – Dec	*H. azteca*	15	4	27
2007	Jan – June	*P. promelas*	183	5	3
2007	Jan – June	*C. dubia*	192	9	5

Year	Months	Test Organism	Number of Tests	Number Toxic	Percent Toxic
2007	Jan – June	*H. azteca*	21	11	52
2008	January	*P. promelas*	18	1[1]	5.5
2008	January	*C. dubia*	20	3	15

[1]Toxicity in one sample attributed to discharge from dairy lagoon

There were 434 toxicity tests performed during the 2001-2002 period of the pelagic organism step decline. None of the samples in 2001 were from the January – June period; 190 samples were collected between April and June 2002 (Table 8). There were 2088 tests performed during the 2003-2008 period. Tests for toxicity to *C. dubia* accounted for 852 tests of which 71 (8.3%) were statistically significant and below the evaluation threshold. There were 572 toxicity tests performed on *P. promelas*, of which 7 tests were statistically significant and above the threshold and 8 tests were statistically significant and below the threshold. Of the 665 *H. azteca* sediment tests performed, 385 (57.8%) were statistically significant. Two tests fell into the first category, 72 in the second category, and 311 in the third category.

Test results for the January to June period for 2002 – 08 are summarized in Table 8 and Table 9. Compared to test results summarized in Fox and Archibald (1997), there was a lower percentage of toxic samples for all species except *H. azteca*. Of all toxicity tests performed in the region, a large percentage were performed on small urban creeks in South San Francisco Bay during the months of January to June and are probably not relevant to the early survival of the POD species as it is unlikely that POD species would be exposed to water or sediment from these sources during that period. Consequently, these tests are not included in Table 8.

It was not possible to use water quality data to explain the results of the toxicity tests. Toxicity Identification Evaluations were performed only for a small portion of the tests and the cause of toxicity in many samples is unknown. In many programs, water chemistry was not performed on water collected at the same time as the samples for toxicity. The toxicity that was found was often restricted to locations in the San Joaquin Valley and was not widely distributed. For example, in 2002, all toxic sediment samples were collected from Orestimba Creek, Ingram Creek, Del Puerto Creek, and Grayson Drain, water bodies located west of the San Joaquin River and south of the Delta. In 2004, 314 samples were collected from water bodies that drain to the Delta and San Joaquin River including numerous sites along 3 urban creeks in the Sacramento metropolitan area. In that year, only 16 sediment samples were collected for *H. azteca* from outside the 3 urban creeks in Sacramento/Roseville. Of those 16 samples, 3 were toxic. Of the 200 samples collected from the urban creeks, 160 were toxic (80%). This was the

highest fraction of toxic tests from any general location sampled over the period for which data were available. All sediment samples were collected from August to November when most POD species are outside the Delta.

INDIVIDUAL MONITORING PROGRAMS

Throughout the course of the POD period, several monitoring programs have conducted toxicity testing. These programs are the Irrigated Lands Regulatory Program (ILRP), Sacramento River Watershed Program (SRWP), Interagency Ecological Program (IEP), Surface Water Ambient Monitoring Program (SWAMP), POD Toxicity Monitoring Program, Sediment Quality Objectives Program (SQO), and NPDES monitoring programs. These programs are addressed individually below. For some programs, data were available for review, for others, program reports were used to generate the discussion. It should be noted that the intent of these programs varied from compliance monitoring to characterization of surface water quality. Reporting requirements and data quality objectives vary across programs, and monitoring data or project reports are used in the current review for a purpose not originally intended by the individual programs.

IRRIGATED LANDS REGULATORY PROGRAM

Investigations of aquatic toxicity in agricultural drains performed by the Irrigated Lands Regulatory Program (2003-2008) focus on the impact of discharges from agriculture on water quality. Test frequency varied across the years with testing generally occurring monthly at fixed stations. Additional testing may have been performed as a result of significant toxicity in previous tests. No testing was performed on surface waters in the North Delta, although limited testing was performed on surface waters that eventually reach the North Delta. Results show widespread acute water column toxicity to *C. dubia* and the unicellular green alga, *Selenastrum capricornutum*, as well as instances of acute toxicity to *P. promelas* in the Central, South and West Delta (Appendix II, Tables 3-5). Toxicity was present throughout the sampling period, during winter months as well as during spring and summer, and site-specific patterns were apparent. Of the 36 *Ceriodaphnia* tests in which significant toxicity occurred, almost half (17) had samples causing complete mortality. Twenty-five of the 36 tests indicating toxicity occurred during the January – June period in which POD species would be present in the Delta. Although there is some variability across years, from 2005 – January 2008, about 6 tests per year indicated acute toxicity. Most of the toxicity (18 of 25 tests) was associated with storm events in the months of January – March of all years.

In >50% of water samples for which *C. dubia* TIE results were reported, the evidence suggested that metabolically activated, non-polar compounds, i.e. OP insecticides, were the cause of toxicity. Chlorpyrifos was shown to be present at up to 5.8 toxic units (TU) in a single sample[1]. Toxicity of several samples was traced to carbamate insecticides as the likely toxicants. "Labile, hydrophobic" non-polar organic compounds contributed to toxicity in other samples, and PBO-synergized toxicity suggests that pyrethroid insecticides were present at toxic concentrations in a up to 3 samples collected from tributaries to the Central and South Delta. Two samples from Lone Tree Creek contained concentrations of ammonia that were toxic to *C. dubia*.

Only 8 samples were toxic to *P. promelas* from 2004 through January 2008. Lone Tree Creek had 3 of the toxic samples and two of those were attributed to ammonia and discharges from dairies. No cause was assigned to the third toxic sample. All three toxic samples were associated with storm events during the January-February period, and the three toxic samples occurred in 2005, 2006, and 2008. Overall, 6 of the 8 toxic samples occurred during the January – June period when POD species are present in the Delta.

Toxicity to *Selenastrum* occurred more frequently than other species with 40 samples causing significantly reduced growth. Twenty-nine of the samples were collected between January and June. TIEs were performed on only 10 samples and the causes of toxicity included cationic metals (2 samples), nonpolar organic compounds (2 samples), or the combination of the two (5 samples). Toxicity was not persistent in one TIE and the results were inconclusive.

Sediment toxicity was more localized than water column toxicity (Table 7) and occurred in 45 samples collected. Sediments collected from Del Puerto Creek, Grant Line Canal, Hatch Drain, Marsh Creek, Kellogg Creek, Roberts Island Drain and Sand Creek were highly toxic, and toxicity was demonstrated over several months and in multiple years at most sites. Toxicity was present but less severe in sediments from Delta Drain (Terminous Tract), Littlejohns Creek, Lone Tree Creek, Unnamed Drain to Lone Tree Creek (Temple Creek) and Pixley Slough. Some samples caused significantly reduced growth while others caused significantly lower survival. The relevance of toxicity to the pelagic organism decline is unclear as it is unknown how much

[1] A Toxic Unit is the amount of a chemical that kills one-half of the test organisms in a toxicity test.

of the sediment from each of these sites is transported to the Delta, where within the Delta it is deposited, how that sediment impacts the pelagic food chain, and how much contaminant leaches from sediment into waters flowing into the Delta.

There are no data available from the ILRP for the step-decline period so no conclusions could be drawn about the impact of toxicity on POD species from the sample sites monitored. Although there are a few months of data available from the summer of 2004 (outside the window of time when POD species are expected to co-occur in the Delta), the bulk of the monitoring for this program occurred from 2005 to the present. Monitoring data suggest that some toxicity was present and often resulted in 0% survival of the test organisms. The North Delta was underrepresented in the sampling with data available from only two stations. As a result, it is difficult to determine from ILRP data whether toxicity was sufficient to contribute partially or wholly to the POD.

SURFACE WATER AMBIENT MONITORING PROGRAM (SWAMP)

Data were available at 119 sites for the period from October 2000 – November 2005. During the review period, toxicity monitoring within this program occurred on the San Joaquin River and its tributaries, many tributaries of the Delta, and numerous creeks draining to San Francisco Bay (Appendix II, Table 6 a, b).

Overall, acute toxicity to larval fathead minnow was detected in 24 of 252 samples (9.5%) collected from the Delta and its tributaries. All tests were four-day acute tests. Four of these tests were performed in the October 2000 – November 2002 period of the step decline, and all occurred during the January – June period. No TIE results were available.

Acute (96 h survival) and/or chronic (7-d growth and survival) *P. promelas* toxicity was seen in the mainstem San Joaquin River (at Crows Landing, Patterson, Hills Ferry, Lander Ave, Airport Way) in June 2001, November and December 2004, and January, February, March, May and September 2005. Eight of the 9 water samples collected at the Tuolumne River @ Shilo were toxic to fish in January 2002, December 2004, February, March, May, June, August and September 2005. Fish toxicity was also repeatedly detected in the Cosumnes (10 of 26 samples), Stanislaus (3 of 9), Merced (3 of 9), and Mokelumne (2 of 8) Rivers during 2004 and 2005 (Appendix II, Tables 9-11). Seasonal patterns were not apparent.

Acute toxicity to *C. dubia* was rarely seen in the tributaries or the mainstem San Joaquin River with only one sample each from the New Jerusalem Tile Drain in 2001, Hatch Drain and Hospital Creek in January 2003, and two samples from the Mokelumne River on the same date in September 2003 having acute toxicity. Chronic toxicity (reduced reproduction) was commonly detected at sites tested including the San Joaquin River @ Patterson (4 of 11 samples), and San Joaquin River @ Airport Way (6 of 11 samples). Several of these samples were collected during the January – June period in 2005.

Twenty-seven samples were toxic to *Selenastrum* between September 2001 and February 2006 (2001 – 2, 2002 – 11, 2003 – 11, 2005 – 2, 2006 – 1). With the exception of the 2001 samples, all were collected within the January – June period. Of the 27 toxic samples, 10 were from water bodies that were tributaries of the San Joaquin River (4 from Del Puerto Creek) or emptied directly into the west Delta (6). Three of the four samples from Del Puerto Creek were collected at different stations within a short distance of each other on the same day in 2002. The remaining 17 toxic samples were collected from water bodies that emptied into San Francisco Bay or the Pacific Ocean.

Sediment toxicity testing using *H. azteca* resulted in 26 tests with significantly reduced survival and 19 tests with significantly reduced growth. Seven of the tests with organisms with reduced growth were from samples in which survival was also significantly reduced (Appendix II, Tables 12-15). Only 8 tests were performed on sediment collected in the Delta or its tributaries while the remaining tests were performed on sediment collected in urban watersheds in the Sacramento and San Francisco Bay areas. Because of the nature of sediment tests, sampling is much less frequent and the applicability of the test results to areas immediately outside the location of sample collection is unknown.

PELAGIC ORGANISM DECLINE (POD) AMBIENT TOXICITY MONITORING; INTERAGENCY ECOLOGICAL PROGRAM

Monitoring sites for this program were selected from among the California Department of Fish and Game Townet Survey stations, and in accordance with the prevalent distribution of POD

species. Sample locations were primarily in main channels of the Delta away from shore (Appendix II, Table 16). The following is a summary from Werner *et al.* (2005, 2008a).

Water samples were collected every two weeks in June-September 2005, and throughout 2006-2008, and tested for toxicity using the amphipod *H. azteca* and *C. dubia* (2005 only). As of January 2006, routine partial toxicity identification evaluation (TIE) tests were conducted with *H. azteca* on all water samples with piperonyl-butoxide (PBO), a chemical synergist/antagonist, to provide early evidence for the presence of two classes of toxic insecticides, organophosphates and pyrethroids. If toxicity (<50% survival within 7 days) was observed in a water sample, TIEs were initiated immediately to identify the causative agents. Water samples were submitted for chemical analyses whenever significant acute or chronic toxicity was observed. Toxicity tests with juvenile delta smelt and juvenile striped bass were performed on samples from select Delta sites in July and August 2005. In subsequent years, acute and chronic toxicity tests with larval delta smelt were performed during late spring/early summer.

INVERTEBRATE TOXICITY

In 2005, significant acute and/or chronic toxicity to amphipods was detected in 6 of 131 (4.6%) total samples in the Napa River (site 340), the Old River (sites 902, 915), the San Joaquin River (sites 910, 804), and the Sacramento River (site 711). Water collected from site 804 (September 27, 2005) also caused significant mortality of copepods (*Pseudodiaptomus forbesi*), which were used as test organism in only one set of tests (Sept. 2005). No significant toxicity to *C. dubia* was observed during the 2005 sampling period, and therefore *H. azteca* was used in all subsequent testing. During 2006-2007, fifteen samples (2.2%) were acutely toxic. Only 1% of samples tested during the first six months of 2008 caused acute amphipod toxicity. Most acutely toxic samples were collected from sites in the lower Sacramento River (Hood, site 711), the Deep Water Shipping Channel (Light 55) and site 405 (Benicia). Only one sample collected from site 602 (Suisun Bay) and one from site 323 (San Pablo Bay) were acutely toxic. To date, the observed pattern suggests compromised water quality in the lower Sacramento River/Deep Water Shipping Channel, lower Napa River and Carquinez Strait near Benicia.

During 2006-2007, 36 (or 5.8 %) of 623 ambient water samples were acutely toxic to *H. azteca*. A more conservative statistical method (Tukey) resulted in 15 (or 2.4%) acutely toxic samples. These numbers include 5 samples, where survival in ambient water with PBO was significantly lower than in the respective PBO control, but no statistical difference was seen between

ambient sample and respective control. The percentage of toxic samples in 2007, a relatively dry year, was far higher than in 2006, a year with unusually high river flows. In 2006, only 1.7% (USEPA or Tukey statistics) or 0.3% (Tukey statistics only) of 353 samples tested exhibited acute toxicity, while 8.8 % (USEPA or Tukey statistics) or 4.1% (Tukey statistics) of 340 samples tested in 2007 were toxic. Most of the acutely toxic samples were collected from sites in the lower Sacramento River (Hood, 711), the Deep Water Ship Channel (Light 55) and Carquinez Strait near Benicia (405).

PIPERONYL-BUTOXIDE EFFECT

PBO is often used to distinguish between toxicity due to pyrethroids and OP pesticides. If the addition of PBO to the sample water enhances the toxicity found in the sample, the putative cause is pyrethroid pesticides. If PBO reduces toxicity in the sample, the putative cause is OP pesticides. During 2006-2007, PBO synergized or reduced toxicity in six (1%) of the samples tested. These came from the lower Sacramento River, Deep Water Shipping Channel, Napa River, Benicia and upper San Pablo Bay. The observed decreased toxicity suggests the presence of OP pesticides in samples collected from Hood and Light 55 (both in October 2007), and the increased toxicity suggests the presence of pyrethroid insecticides in samples collected from Napa River, San Pablo Bay and Benicia. TIEs performed on toxic samples from sites 323 and 405 indicated that non-polar organic chemicals contributed to the observed toxic effects, while toxicants were labile and could not be identified in samples from sites 711 and Hood. In 2008, PBO synergized toxicity of two samples from Cache Slough near the mouth of Ulatis Creek (1/31/08, 2/28/08). A pyrethroid-focused TIE (1/31/08) showed that pyrethroid insecticides caused the observed effects.

Amphipod growth relative to controls was not a sensitive indicator of toxicity, partially due to the variable size of the organisms at test initiation, and – more importantly - the variability in food content of ambient water samples from different sites. Final dry weight of *H. azteca* exposed to laboratory control water was generally lower than in ambient samples. Within-laboratory tests demonstrated that the reduced growth was due to the lack of particulate organic matter (POM) naturally present in ambient water samples, which *H. azteca* used as a supplemental food source.

Effects of PBO on *H. azteca* growth: Addition of PBO to the ambient sample resulted in a significantly different final amphipod weight relative to the corresponding ambient treatment in 73 water samples (10.5% of samples tested; Appendix II, Tables 5 a, b). Of these, 28 (4.0% of samples tested) were significantly different based on two statistical approaches. Amphipods exposed to 22 PBO-treated samples were heavier than their counterparts (only 3 based on Tukey's statistics) suggesting OP toxicity, while weight was lower in 51 PBO-treated samples (24 based on Tukey's statistics) suggesting pyrethroid toxicity. Final amphipod weight in PBO-treated water samples was significantly lower than the PBO control in 9 of these samples. Sites in the South-Eastern Delta (902, 910, 915), Montezuma Slough (609) and the Lower Sacramento River (711) had the highest number of samples showing PBO effects on growth, and patterns where several neighboring sites sampled on the same date showed similar organism responses were seen repeatedly. For example, PBO addition resulted in an increase in growth in samples collected from sites 902, 910 and 812 on June 6, 2007. Most samples where addition of PBO modified amphipod growth were collected during winter/early spring or late summer.

Toxicity Identification Evaluations: There were few samples that caused reduced survival to the extent required for TIE procedures (beyond the addition of PBO with the initial screening) to be successful. In all 4 samples tested, toxicity was lost by the time TIE procedures were performed.

FISH TOXICITY

Fish toxicity: No significant acute or chronic (growth) toxicity to juvenile (3-months old) striped bass was observed in July 27/28, 2005 samples from sites 340, 711, 910 and 915. No significant toxicity to juvenile (3-months old) delta smelt was observed in August 30/31, 2005 samples from DFG stations 340, 711, 910 and 915.

Tests with larval delta smelt on water samples from sites 711, 910, 915, 609, 504 and 340 (2006), or 711, Hood, 915, Vernalis, 609, 504 and 340 (2007) showed that survival was significantly reduced in samples from Hood (collected June 6, 2007) and site 711 (July 26, 2007), both in the lower Sacramento River. Although electrical conductivity (EC) and turbidity were low at these sites, the reduced survival could not be explained by these factors alone when compared to the low EC and low turbidity controls. In 2008, two of four samples from the

Sacramento River at Hood (collected 4/22/2008 and 5/20/2008) caused a significant reduction in delta smelt survival compared to the appropriate controls.

EFFECT OF AMMONIA ON INVERTEBRATES AND FISH

Although ammonia was not the focus of this review because an intense synthesis of ammonia data and literature was concurrently being assembled. However, analysis of 2006-2007 *H. azteca* data revealed that ammonia-N and unionized ammonia had significant effects on amphipod growth. When analyzed by site, total ammonia-N concentrations were negatively correlated to survival at Light 55. Ammonia-N and un-ionized ammonia concentrations were negatively correlated to *H. azteca* growth at sites 323, 812 and Light 55. Analysis across sites for different seasons determined that survival and growth during the winter of 2007 was negatively associated with levels of ammonia-N and un-ionized ammonia. For delta smelt, there was no correlation of larval 7-d survival with NH_3 concentration, where maximum unionized ammonia concentrations were <0.016 mg/L.

CHEMICALS DETECTED

Several samples contained detectable amounts of pyrethroid pesticides: Site 902 sampled on 8/22/06 contained 5 ng/L cyfluthrin and 24 ng/L permethrin; site 340 sampled 2/13/07 contained 63 ng/L cyfluthrin, and sites 915 and 508 sampled on 2/28/07 and 3/1/07, respectively, contained 2 and 3 ng/L lambda-cyhalothrin. A sample from Light 55 collected 2/1/07 contained 6 ng/L of diazinon. The pyrethroids bifenthrin and/or lambda-cyhalothrin were also detected in 2008 at sites Cache-Ulatis (2/28/008), 711 (4/9/08 and 4/23/08), Hood (4/22/08), and Cache-Lindsey (4/23/08). The herbicide diuron was detected in all samples where it was analyzed at concentrations up to 0.086 µg/L (sites 711, Cache-Lindsey, Hood, 902) sampled 4/9/08, 4/22/08 and 5/12/08. The water quality goal for diuron is 2 µg/L. PAHs were detected at Hood and Cache-Lindsey. Analysis results of total and dissolved metals suggest that metals are unlikely to be the dominant toxicants in these water samples, although a more detailed analysis of available LC_{50} and effect concentrations as well as mixture effects is needed. A sample collected from Suisun Slough on 5/21/08 contained relatively high concentrations of dissolved copper, chromium, cadmium, arsenic and aluminum.

Nine water samples analyzed during the reporting period contained detectable concentrations of insecticides: A sample from site 340 (collected July 25, 2007), which caused a significant

reduction in *H. azteca* survival after PBO addition, contained 3 ng/l cyfluthrin and 16 ng/l esfenvalerate. Two samples from site 405, which caused significant amphipod mortality (collected September 4 and October 4, 2007), but no PBO effect, contained 3 ng/l esfenvalerate, and 5 ng/l permethrin, respectively. A sample collected on February 1, 2007, from Light 55 contained 6 ng/L of diazinon. In addition, several water samples that caused a significant negative PBO effect on *H. azteca* growth contained detectable amounts of pyrethroid pesticides: 5 ng/l cyfluthrin and 24 ng/l permethrin were detected at site 902 (August 22, 2006), 63 ng/l cyfluthrin at site 340 (February 13, 2007), and 2 and 3 ng/l lambda-cyhalothrin at sites 915 (February 28, 2007) and 508 (March 1, 2007), respectively.

Water samples were stored in the dark at 4^{o}C before chemical analysis, but most were not preserved with the solvent dichloromethane (DCM). Analytical results of water samples from site 405 (collected October 4, 2007), one spiked with DCM and the other without DCM, showed that pyrethroid insecticides can degrade during the 14-d storage. Analysis of the DCM-spiked sample resulted in the detection of 3 ng/l esfenvalerate, while the unspiked sample resulted in no detection.

SEDIMENT QUALITY OBJECTIVES PROGRAM - PHASE II, 9/17/2007-10/16/2007, SPRING 2008

In the fall of 2007 and spring 2008 sediments, were collected from 150 sites throughout the Delta and tested for toxicity with *H. azteca* using 10-day sediment tests (10-d survival/growth). A subset of 75 samples were also tested with *Chironomus dilutus* (10-d survival/growth), and these samples were analyzed for sediment quality measures and an extensive number of chemicals of concern.

Of 100 samples tested for *H. azteca* toxicity in 2007, samples from 3 stations caused significant reductions in survival compared to the controls and 15 samples caused significantly reduced growth. One sample caused significantly reduced survival to *C. dilutus* and 3 samples caused significantly lower growth. Tests results were evaluated by applying two statistical criteria: significance in T-test and above the MSD (minimum significant difference) threshold (Table 17). The 3 samples that had significant mortality had survival that ranged between 46-63%: EMP-0150 (Mildred Island), EMP-0006 (Latham Slough) and EMP-0049 (Indian Slough). The sample from EMP-0150 also caused reduced *C. dilutus* survival.

Chemicals: PAHs, DDE, methoxychlor, diuron and PBO were detected in over 85% of sediment samples analyzed in the fall of 2007. In the spring of 2008: PAHs, bifenthrin, diuron and PBO were present in over 85% of the samples. Other contaminants detected were chlorpyrifos, carbaryl and permethrin. Preliminary analyses indicated that pyrethroids were unlikely to be the only toxicants responsible for the detected sediment toxicity.

SACRAMENTO RIVER WATERSHED PROGRAM (SRWP); 2000-2007

The results of the 2000-2007 monitoring and of previous aquatic toxicity monitoring efforts showed that toxicity to test organisms occurred in surface waters throughout the Sacramento River watershed. TIEs indicated that *C. dubia* toxicity was attributable to OP pesticides in agricultural runoff and urban runoff in 2000-2004, but "labile compounds" were responsible for toxicity in a number of samples. Tests using the *P. promelas* and *S. capricornutum* were performed before 2000 and after 2003. TIEs (USEPA 1991, 1992, 1993) were performed on selected samples to identify the toxicants responsible for repeated adverse effects in toxicity tests. A more detailed summary on toxicity at Sacramento River at Freeport is provided below, as this site is most relevant to this review.

SACRAMENTO RIVER AT FREEPORT

In 2000-2001 monitoring, 22% and 44% of samples (n=9) caused lethal or sublethal toxicity to *C. dubia*, respectively. In 2001-2002 and 2002-2003 monitoring, 2 of 5 samples (40%) were chronically toxic to *C. dubia.* In 2003 and 2004, toxicity to both *C. dubia* and *P. promelas* was more frequently observed during the dry than during the wet season, with no clear indication of a specific source of toxicity.

In 2003-2004 monitoring, the most severe *C. dubia* toxicity was observed in the mainstem Sacramento River during the irrigation season (June 2004), and no significant mortality was observed during the wet season. The only apparent spatial pattern was that samples collected from mainstem Sacramento River sites exhibited a higher frequency of *P. promelas* toxicity than major tributaries or agricultural drains: eight of 20 (40%) samples were acutely toxic to *P. promelas*. Fish toxicity was more frequently detected in the upper river from Keswick to

Hamilton City than from Freeport, where only one of 4 samples or 25% caused significant mortality. No toxicity to *S. capricornutum* was observed in the 44 samples collected.

Toxicity Identification Evaluations were performed using *C. dubia* on toxic samples from the Sacramento River at Bend, Sacramento River at Freeport, and Arcade Creek, but toxicity was not persistent in the original samples at the time of re-testing.

2006-07: During this period the Sacramento River at Freeport was highly toxic to *C. dubia* in July, August and December 2006, and in April 2007 (Table 18). *P. promelas* toxicity was detected in October 2006 and June and July 2007. Results of TIEs indicated that toxicity was caused by (a) labile compound(s) in samples collected 7/26/06, 8/24/06 and 4/25/07. TIE results suggest that toxicity was likely due to several classes of chemicals in a sample collected 12/12/06.

NPDES PROGRAM

A largely incomplete dataset was obtained for NPDES monitoring. Data covering only part of the review period were available for 7 facilities that discharge effluent into the Delta and its tributaries. Two complete data sets were available for review; the Sacramento Regional WWTP (SRWWTP) and the Stockton WWTP (SWWTP). Data for the SRWWTP were available for effluent for 2005-2008 for three species and for acute and chronic endpoints. Data were available from the SWWTP from 2000-2008, but data for 2005 were missing.

Data available from the SRWWTP indicate that toxicity from the plant effluent occurred periodically during the 2005-2008 period. In order to interpret the results of the toxicity testing, the NPDES permit allows a dilution credit which specifies a permitted TU level of 8. Toxicity at less than 8 TU is not considered significant. Just under 78% of the 208 acute fathead minnow (*P. promelas*) tests indicated comparable survival of fish in the control and the effluent treatments. Approximately 15% of the tests were different by 5%, and approximately 5% of the tests were different by 10%. The cumulative frequency distribution indicates that in 95% of all tests, survival of controls and effluent treatments were different by 10% or less. In only 2.4% of the tests (6 tests) were the effluent and control treatments different by 15% or more. These tests occurred in March 2005, August 2005, February 2008 (2 tests) and March 2008 (2 tests). Quarterly algal chronic toxicity data were available from 2005-2008. One sample in April 2008

tested toxic to algae with 4 TUc. Sixty-seven acute and chronic toxicity tests were available for *C. dubia* from 2005-2007. A note in the dataset indicates that toxicity during the 2004-2007 period is an artifact of sampling and "Therefore, these reported toxicity results for *C. dubia* during this time are not necessarily representative of the effluent (sic) discharged to the environment." A Toxicity Reduction Evaluation (TRE) analysis undertaken to identify the cause of the toxicity and TIEs performed during that analysis indicated that a biological mechanism (bacterial) associated with the sampling methodology was responsible for the toxicity to *C. dubia*. Once the sampling was corrected in early 2008, a single TU of 16 occurred in January 2008, i.e. 22 of the 23 tests exceeding the TU limit of 8 occurred prior to January 2008. Twenty-four fathead minnow tests using both acute and chronic endpoints were performed between 2005 and 2008. One chronic test in October 2007 reached the 8 TUc permitted limit.

The available data indicate that Stockton WWTP effluent was toxic to *P. promelas* on 6 occasions in 2000-2001 with 2 to 8 toxic units (TU; survival) and 8 to >16 TU for the growth endpoint. No fish toxicity was detected in 2003-2004 and 2006-2008. Effluent was acutely toxic to *C. dubia* on 13 occasions in 2000/01, April 2003, June 2004, April 2006 and July 2006. Effluent was frequently toxic to green algae (*S. capricornutum*). The City of Brentwood data indicate no toxicity to fish or *C. dubia*, but no data were provided for 2000-2002. Toxicity to green algae was detected in March and December 2003. Discovery Bay effluent was toxic to green algae in January and May 2007. Tests on Tracy WWTP effluent were performed quarterly, and toxicity to *C. dubia* and fathead minnow was detected in most samples tested in 2000. Effluent was toxic to fish throughout 2001-2007. Toxicity to *C. dubia* and *S. capricornutum* was detected in the early part of most years during the review period.

RELEVANCE TO POD SPECIES

The relevance of the toxicity detected in the Delta and its tributaries for POD fish species can be assessed only indirectly, since little information is available on the sensitivity of these species to contaminants. Available toxicity data collected in 2000-2007 indicate that toxicity to test species in the water column as well as sediments is widespread in waterways of the Central, South and North Delta including the mainstem rivers. Of special concern is fish toxicity in the lower San Joaquin and Sacramento Rivers. The San Joaquin River and its tributaries were toxic to larval *P. promelas*. Monitoring in the lower Sacramento River was limited to a sampling site near Freeport with relatively few samples per year. However, fish toxicity was consistently detected in >20% of samples. *P. promelas* toxicity was less common in small Delta waterways and drains, and, in part traced to ammonia and possibly pyrethroid insecticides. In addition, it

has recently been shown that water quality in the lower Sacramento River is not favorable for larval delta smelt, which could partially be due to natural factors such as low turbidity and low electrical conductivity. The lack of concurrent toxicity to *C. dubia* and the relative tolerance of *P. promelas* to contaminants (relative to *C. dubia*) lead to the question of whether the cause is related to contaminants or some other stressor. Unfortunately, no TIE data exist across all programs examined to address the question.

Data analyzed for this report suggest that small creeks, canals and sloughs of the Delta tend to be more toxic to invertebrates and green algae than to fish. Water column toxicity has been traced to pesticides, dominantly OP and pyrethroid insecticides, cationic metals and herbicides. Additional contaminants frequently detected in sediments are DDE, a breakdown product of DDT, PAHs, especially the carcinogen benzo(a)pyrene, carbamate insecticides, the herbicide diuron and the pyrethroid-synergist, piperonyl-butoxide (PBO). OP pesticides, in particular chlorpyrifos, are less likely to directly affect fish survival since fish are considerably less sensitive to these chemicals than *C. dubia* and other arthropods. However, it is possible that invertebrate prey items of POD species are adversely affected by OP pesticides. In turn, phytoplankton populations may be affected by herbicides and cationic metals. Pyrethroid insecticides are a far bigger concern with regard to direct toxic effects on fish, as fish are highly sensitive to these chemicals relative to OP pesticides. The possibility of both direct and indirect effects on POD species therefore cannot be dismissed. More information on the effects of major contaminants on embryo development, hatching and rearing of these species is needed.

There is considerable uncertainty with regard to the sublethal effects and their consequences on fitness of fish in the wild. Copper, OP and pyrethroid pesticides have been shown to negatively affect the ability of fish to swim normally, respond to predators, and protect themselves from pathogens (Werner *et al.*, 2008; Werner and Moran, 2008, and references therein). In addition, pyrethroid insecticides and many wastewater associated chemicals have been shown to be endocrine disrupting chemicals (EDCs) (McCarthy *et al.* 2006). The sublethal and chronic effects of contaminant mixtures, for example ammonia or copper in combination with other contaminants, cannot be assessed at this point in time, but data collected in recent biomarker studies indicate that EDC concentrations are biologically significant (i.e. inducing vitellogenin/choriogenin production in male fish) in the lower Sacramento River, the San Joaquin River and in Suisun Marsh (Brander and Cherr, Loyo-Rosales *et al.*, Riordan *et al.* Calfed Science Conference 2008). Pharmaceuticals and personal care products are now also being seen as chemicals that can impact fish and invertebrates (Kostich and Lazorchak 2008).

Conclusions

- Although toxicity data were not reviewed from the period prior to the POD years, it appears that there was as much or more toxicity in water samples collected in the Delta in the pre-POD years compared to the POD years.

- There appears to be no difference in the percentage of toxic water samples to either *C. dubia* or *P. promelas* between the 2000-2002 step decline years and the later POD years (Table 10).

- The percentage of toxic samples in the January to June period varied between 0% and 7% (Table 10) across monitoring programs. Many of these toxic samples were collected from water bodies that are tributaries to the major rivers and it is not clear how transit time and dilution would affect the toxicity of these waters. The percentage of toxic samples collected from Delta waters is slightly lower and less frequent but indicates the potential for toxicity to prey items utilized by POD species.

- Significant toxicity (50% to 80% of tests performed) from sediment was common throughout the POD period. The significance of sediment toxicity is unknown as it is the interstitial water in the sediment that causes toxicity. Giesy *et al*. (1999) argued that concentrations of chlorpyrifos in sediment interstitial water could not be greater than the concentration in the water column arguing that resuspension of sediments and contaminants would not cause significant additional toxicity. The exception would involve metals in anaerobic conditions in aquatic sediments that would be exposed to oxygen.

HISTOPATHOLOGY

Histopathology markers are good indicators of environmental stress as they provide visible biological endpoints and measurable responses to subcellular mechanisms that can integrate exposure over time (Stentiford *et al.* 2003, Sherry 2008). Histological lesions were shown to be sensitive and reliable indicators of the health of wild fish populations in Europe and North America. These biomarkers may be important sentinel endpoints for assessing the impacts of contaminant exposure in fish in the Sacramento-San Joaquin River Delta. Here, we provide a brief synthesis of studies describing potential links between contaminant exposure and fish health in the San Francisco Bay – Delta system using histopathology as a principal indicator of exposure and effects of contaminants of concern.

HISTORICAL PERSPECTIVE

Very few data exist for the period prior to the POD. Fox and Archibald (1997) reference several studies that reported lesions in fish or measured biomarkers in various species, but the predominant analytical methodology was to measure concentrations of contaminants in various fish tissues. While providing evidence of exposure, fish tissue concentrations provide no measure of effects.

Fox and Archibald (1997) reported that over 500 striped bass from several major rivers on the east and west coats were examined as part of the Cooperative Striped Bass Study which was conducted from 1978 – 80. Sacramento River fish were determined to be in poor health compared to Oregon fish with subcutaneous lesions, parasites, and discolored fatty livers with occasional fibrous erosions. Eggs were obtained from female bass, fertilized, and allowed to develop. Eggs from four families had desynchronous cleavage and deformed embryos and larvae displayed scoliosis, and poor yolk sac utilization. Additional analyses found significant correlations between the concentration of organochlorine compounds and reduced fecundity and egg viability, abnormal egg development, and delayed maturation of eggs. All of the gross histopathologies found in the COSB study were attributed to organochlorine compounds including DDT, PCBs, and toxaphene. Histopathology studies of striped bass larvae exposed to water from the Colusa Basin Drain and the Sacramento River in 1991 found a number of lesions of both the central nervous system and the skeletal muscle. The majority were classified as

moderate to severe lesions. Between 1988 and 1991, 15 to 30% of striped bass in the Delta had liver lesions typical of exposure to rice pesticides.

Summer die-off of striped bass in the Sacramento-San Joaquin Delta in 1987 showed hepatotoxic conditions suggestive of urban chemical pollutants (Cashman *et al.* 1992). Contamination by aromatic hydrocarbons, alicyclic hexanes and DDTs were also shown to affect egg resorption, abnormal egg maturation and egg death in striped bass from the Bay (Setzler-Hamilton *et al.* 1988). In the Sacramento River, larval striped bass captured in 1988 – 1990 showed liver pathologies consistent with exposure to toxic compounds (Bennett *et al.* 1995) and acute toxicities from runoff water to the river (Bailey *et al.* 1995). Spawning habitats of the Delta smelt, *H. transpacificus* in the Delta showed high concentrations of pesticides in 1999–2000 but high spring outflows in 1998 reduced pesticide concentrations (Kuivila and Moon 2004). While the number of studies is not large, clearly there were gross lesions in fish identified as far back as the late 1970s and continuing until the 1990s.

POD SPECIES AND HISTOPATHOLOGY

No data are publically available for evaluation of histopathology evidence of the effects of contaminants on POD species. Instead, reports from state and federal agencies, and publications in peer reviewed journals were used to obtain information discussed below. Portions of this review were written by the author of some of those reports (S. Teh).

Evaluation of causal relationships between contaminant exposure and biological effects in fish and other aquatic animals is a notoriously difficult task due to the influence of complex and dynamic environmental factors on fish responses to natural stressors and toxicants through their entire life stages. Biomonitoring studies conducted in the Sacramento-San Joaquin Delta have focused on measurement of chemical concentrations in water, sediments and aquatic organisms as a component of habitat management and restoration strategy. This difficulty is due to the broad range of sensitivities to contaminants among the many aquatic organisms in the estuary (Thompson *et al.* 2007) as well as the paucity of studies on long-term, low-level exposures to contaminants (Forrester *et al.* 2003). Although toxicity testing of fish using sediments and water from the San Francisco estuary have been conducted extensively, this approach may be insensitive to subtle effects of long-term exposure to contaminants. Hence field-based studies of fish health are useful for assessing contaminant impacts (Thompson *et al.* 2007). Studies of biomarkers in fish from the Bay-Delta system may demonstrate contaminant

effects on cellular processes and tissue structure, although it is a challenge to correlate the presence of specific contaminants with effects (biomarkers) in fish (Stehr *et al.* 1997, Whitehead *et al*. 2004).

Very little information is available for the POD species from any period of time, and particularly during the period of the step-decline in 2000-2002. In the Sacramento River, eggs and larvae of striped bass showed higher concentrations of many pollutants including PCBs, PBDEs and chlorinated pesticides and histological analysis revealed developmental alterations (e.g. reduced growth, rapid yolk sac depletion, altered liver development) that may compromise field survival. Ostrach *et al.* (2008, 2009) performed both chemical and histopathology analyses on striped bass eggs collected from the Sacramento River in 1999 and 2001, and again in 2006 and 2007. In the 1999 and 2001 studies, chemical analyses indicated elevated levels of PCBs, PBDEs, current use (chlorpyrifos), and legacy pesticides and their breakdown products in eggs collected from the field compared to eggs from hatchery reared broodstock in all years. By day 5 posthatch, livers from control fish exhibited advanced cellular architecture, morphology, and abundant stores of glycogen. Livers from larvae from eggs collected in the river exhibited regressed/indistinct cellular architecture, hepatocytes were devoid of glycogen, and nuclear bunching was observed. By extension from results of other research, the exposure of the eggs to maternally transferred contaminants could result in poor growth and development, poor hatching success, alterations of the reproductive and nervous systems, learning and behavioral difficulties, abnormalities of the liver and other organ systems, and endocrine disruption. Ostrach *et al.* (2008) suggested that the combination of abnormal development, yolk deficiency, and altered, shrunken liver devoid of glycogen at day 5 posthatching would adversely affect subsequent growth and survival of fish in the river.

In the 2006-2007 study report, Ostrach *et al.* (2009) reported abnormalities in larvae and early juvenile striped bass similar to those observed in the 1999 and 2001 fish. The consistency between the 2006 and 2007 data suggested the continual maternal transfer of xenobiotics. Lesions were observed in the brains of larvae from 2006 and 2007 consistent with exposure to xenobiotics as reported in other studies. Ostrach found that the majority of juvenile striped bass examined in their study were under sublethal contaminant exposure as measured by P450-1A1 expression. EROD (7-ethoxyresorufin-O-deethylase) induction, the quantitative measure of P450-1A1 activity) was found in 65% of juvenile striped bass collected from August 2007 through January 2008. Vitellogenin expression was found in 22% and metallothionein induction was found in 34% of the fish. Overall, 33% of the fish examined were found to express multiple biomarkers of exposure to contaminants. Ostrach *et al.* (2009) concluded that

the “vast majority of juvenile striped bass are suffering from sub-lethal contaminant exposure of several types” “causing severe physiological stress, morbidity, and likely compromising the immune systems in the fish.” In addition, data on adult fish suggest that they are adversely affected by bioaccumulation of contaminants such as PBDEs. They concluded that contaminants should be considered as a significant stressor affecting the decline of striped bass and are likely causing population level effects by affecting the early life stages.

Teh (2007) recently reported on histopathologic analyses of 385 delta smelt collected in the Spring Kodiak Trawl Survey from January and February 2005. The 22 stations from which fish were obtained were placed subjectively into five geographic regions; Suisun Bay, Suisun Marsh, Central Delta, South Delta, and North Delta. Three fish were collected from the Napa River. Gonads, livers, and kidneys were examined to evaluate the health status of individual smelt. Tissue alterations were qualitatively scored on a scale of 0 (= not present) to 3 (= extensive severe pathological alterations). Prevalence of lesion scores were used to establish the percentage of fish with significant organ damage and scores were compared across stations.

Teh (2008) concluded that the overall health status of adult delta smelt was not a function of food limitation as 88.8% of the fish had prey items in their gut. Their health was also not a result of diseases as only 1.3% of the fish had internal parasites. Teh compared the results to a similar analysis of fish from the Fall Midwater Trawl by the California Department of Fish and Game (CDFG) who found very similar results. Kidney and gonad lesions in the 2005 SKT fish were mild and Teh concluded that they were unlikely to be the cause of the low smelt abundance index.

Scoring the health of the fish from the different geographic regions based on all lesions indicated that the fish from the two Central Delta stations were significantly healthier than fish from the other sites. The three fish from the Napa River were the least healthy but the estimate was accompanied by a large variance (due to the sample size of 3), which made the mean health measure statistically indistinguishable from all other treatments. The rank order of fish health from best to worst was: Central Delta, Suisun Bay, North Delta, Suisun Marsh, South Delta, and Napa River (see Figure 6 in Teh 2007).

Liver histopathologies were different for fish from different geographic regions. A high prevalence of glycogen depletion, fatty vacuole degeneration and single cell necrosis in fish from the Suisun Marsh and South Delta suggest that these fish were experiencing adverse impacts from exposure to contaminants. Fish from the North Delta experienced an elevated prevalence of glycogen depletion and single cell necrosis, but not fatty vacuole degeneration which suggests they were exposed to non-chemical stressors such as low dissolved oxygen or elevated CO_2. The absence of macrophage aggregates in livers of smelt suggests the histopathologies were developed only a few weeks prior to sampling. Teh (2007) concluded that exposure during the larval and juvenile stages to stressors such as starvation, heat, bacterial infections, and parasitic infections were unlikely to have been the cause of the significant liver lesions seen in their analysis. He further concluded that the histopathology results strongly suggest that the contaminant and physiochemical exposures and resulting effects occurred around the time of sampling in the regions where the fish were caught. Despite the fact that Teh concluded the histopathologies were the result of exposure of adults, he urged investigating smelt larvae as well.

Subsequent to the analysis of spring adult smelt, 47 adult delta smelt collected in the 2005 Fall Midwater Trawl between September and December were examined for liver and gonadal lesions. No food limitation was found based on stomachs containing prey items. There were no significant kidney lesions. Ova-testis, liver cell necrosis, and preneoplastic foci were the most significant lesions. Fifteen of the 28 smelt collected from Suisun Bay had mild to moderate liver cell necrosis and 4 of the 28 had preneoplastic foci. Teh (2008) concluded that the ova-testis indicate exposure to endocrine disrupting chemicals while the preneoplastic foci indicate exposure to xenobiotic carcinogens or promoters in Suisun Bay.

In 2006, 61 larval and juvenile fish collected by the CDFG during targeted POD fish sampling surveys were examined for histopathologies. Food limitation was not an issue except for larval smelt collected from the Napa River. Larval smelt collected from the Napa River had moderate to severe glycogen depletion, moderate kidney tubular dilation, and mild cell necrosis in the gill. The prevalence of these same lesions was lower in juveniles collected at the same location a month later. Prevalence of lesions was low in larval smelt collected in Suisun Bay but the prevalence of lesions was much higher in juveniles collected a month later. Lesions included moderate to severe kidney glycogen depletion and mild inflammation, moderate to severe kidney tubular dilation and inclusion, and severe epithelial and chloride cell hyperplasia and necrosis. One juvenile smelt had an invasive papilloma-opercular tumor (Teh 2008). Teh (2008)

concluded that some of the lesions suggest exposure to contaminants and that the lesions could have direct impacts on fish survival.

The California Department of Fish and Game and the US Fish and Wildlife Service completed three reports on the POD species. Table 2 of the 2005 report (Gartz 2005, http://www.science.calwater.ca.gov/pdf/workshops/POD/2005_final/Gartz_POD_Fish_condition_and_health_2005.pdf) provides information on the percentages of DS, Inland silverside, STRIPED BASS, and threadfin shad that had external parasites, eroded fins, gill parasites, internal parasites, or skin lesions. Fish were categorized as being collected in open water or shallow channels. Two percent of delta smelt collected in open channels had internal parasites (0% in fish from shallow water habitats) and 2% of threadfin shad from open channels had skin lesions (0% from shallow water). Ten percent of Inland silverside from open water had internal parasites, while in fish from shallow water 6% had eroded fins, 5% had internal parasites, and 1% had skin lesions. For striped bass from shallow water, 29% had internal parasites and in fish from open water, 1% had eroded gills, 1% had gill parasites, and 32% had internal parasites. However, the author concluded that the fish in the 2005 study were in good health as measured by length-weight comparisons, and that striped bass with parasites were in better condition than those without parasites, although the biological significance of this finding was questioned.

In 2006, the USFWS focused on the threadfin shad and longfin smelt (Foott et al 2006, http://www.science.calwater.ca.gov/pdf/workshops/POD/2006_final/Foott_POD-LFS_TFS-Health_2006.pdf). Of the 147 longfin smelt sampled, the authors concluded that no abnormalities were observable in the 13 tissues on which histology was performed. Internal parasites were seen in 16 fish and external parasites were found on 2 fish, but none of the fish were experiencing inflammation. Only 15 threadfin shad were used for histological examination. Chlamydia infection was observed in the gill lamellae of 11 fish, but the cysts observed appeared to be benign and no inflammation was seen. The study was repeated in 2007 with an increased sample size of larvae and juvenile fish (Foott and Stone 2007, http://www.science.calwater.ca.gov/pdf/workshops/POD/2008_final/Foott_POD-LFS_TFS-Health_2007.pdf). The authors concluded that there were no significant health problems in either species. No virus was isolated in over 800 samples and the low incidence of parasitic infections was not associated with inflammation. They did comment that hepatocytes vacuolation was seen in many longfin smelt but were not sure if it was normal or associated with toxic insults.

In a recent study with a non-pelagic species, the Sacramento splittail (*Pogonichthys macrolepidotus*) captured from the Sacramento-San Joaquin River Delta in 2001 and 2002, organochlorine contaminants (PCBs, DDTs, dieldrin, chlordanes and PBDEs) and trace metals (Ag, As, Cd, Co, CR, Cu, Fe, Mn, Mo, Ni, Pb, Se, Sn V, Zn) were observed in tissues of all fish examined (Greenfield *et al.* 2008). Histopathological analysis indicated toxic effects in the liver including glycogen depletion, lipidosis and cytoplasmic inclusion bodies and macrophage aggregates in tissues. Significant correlations were observed between histopathological indicators and fish health suggesting that histopathology was as a function of health and nutrition rather than exposure to contaminants. However, splittail are considered relatively insensitive to many contaminants relative to species such as salmonids or other species (Greenfield *et al.* 2008).

SUMMARY

There are no histopathology data from the POD species from the step-decline period. However, histopathology analyses of striped bass from prior to the step decline but during a period of population decline, suggests that there is evidence of exposure to contaminants that could adversely impact survival and reproduction. Recent studies also indicate the maternal transfer of PCBs, PBDEs, current use and legacy pesticides and their breakdown products (e.g. DDT, DDE, DDD) that negatively impact the development and survival of larval striped bass. Histopathology data from Delta smelt collected in 2005 and 2006 also suggest that there is exposure of larval, juvenile and adult smelt to contaminants in areas ranging from the Napa River to the South Delta. These lesions were sufficiently significant that they were assumed to impact survival and reproduction. Additional studies by CDFG and USFWS indicate that very little evidence of histopathologies are present and that a non-POD species, the inland silverside (which is undergoing a rapid population increase), experienced slightly greater percentages of various lesions compared to POD species. A non-POD species, the Sacramento splittail, was sampled during the 2000 – 2001 period which coincides with the step-decline period. Fish experienced several histopathologies indicative of poor health and nutrition rather than exposure to contaminants.

Conclusions

- There are insufficient data from the pre-POD period to determine if lesions were more or less common or severe prior to the POD years. Ostrach's research suggests that

striped bass have been experiencing reproductive failure due to organochlorine compounds since prior to the step decline years.

- There are insufficient data to determine if histopathologies were greater during the 2000-2002 POD period compared to the later POD years.
- Overall, there is little evidence of major histopathologies in POD species or non-POD species in the later POD years (2004-2007). Some lesions in some years in selected locations do suggest exposure to contaminants.
- Although lesions can take long periods of time to develop, some lesions were described as developing in the few weeks prior to capture of the fish in the fall.
- Full stomachs of individuals captured for analysis suggest that delta smelt are not starving. This suggests that the food supply has not been reduced by exposure to contaminants and can support populations of POD species.

CONCLUSIONS AND RECOMMENDATIONS

SYNTHESIS/CONCLUSIONS

The goal of the review was to bring together chemical, toxicological, and histopathology data and determine if there are sufficient data to state whether contaminants potentially could be wholly or partially responsible for the Pelagic Organism Decline. The review was not meant to make a definitive determination of the role of contaminants, but rather to determine if sufficient data are available to determine if contaminants could be implicated in the decline. However in some instances, the potential for contaminants to cause declines was possible to evaluate.

Data availability varies across the POD period (Table 10). While the categorizations are subjective, there is clearly a dearth of data for the period during which the step decline occurred. There also appears to be a dearth of data for locations within the Delta while waterbodies outside the Delta are well characterized. There were several monitoring programs in place in the years prior to 2000-2002, but most were not continued from the period prior to the POD through the POD years. The exception would be the NPDES compliance monitoring programs which are extremely limited in geographic scope. Conversely, SWAMP monitoring is extremely detailed with several constituents and many sites, but the locations move yearly preventing the development of an adequate time series of data for future use. As this synthesis illustrates, trying to extract data from multiple monitoring programs to cover the other's "weak links" clearly results in an inability to properly connect the presence of contaminants in the water column or sediment and demonstrated toxicity to POD species or their prey with changes in POD species' population dynamics. Until such time as a single monitoring program is put into place that frames sample collection for multiple ecological and toxicological parameters in a biologically meaningful way, future discussions of contaminant-related issues will end in disappointment.

Table 11. Data availability based on data that are publically available (with associated QA/QC data) for chemistry, toxicity and histopathology analysis for three time periods: pre POD, step decline, and later POD. Extremely limited – one dataset with few constituents, records, and locations ; Limited – one or more datasets with few constituents, records, and locations; Moderate – one or more datasets with many constituents, more than 10 sites, and data records at least monthly for several years; Good – more than one dataset with many constituents, many sites, and data records at least monthly for several years.

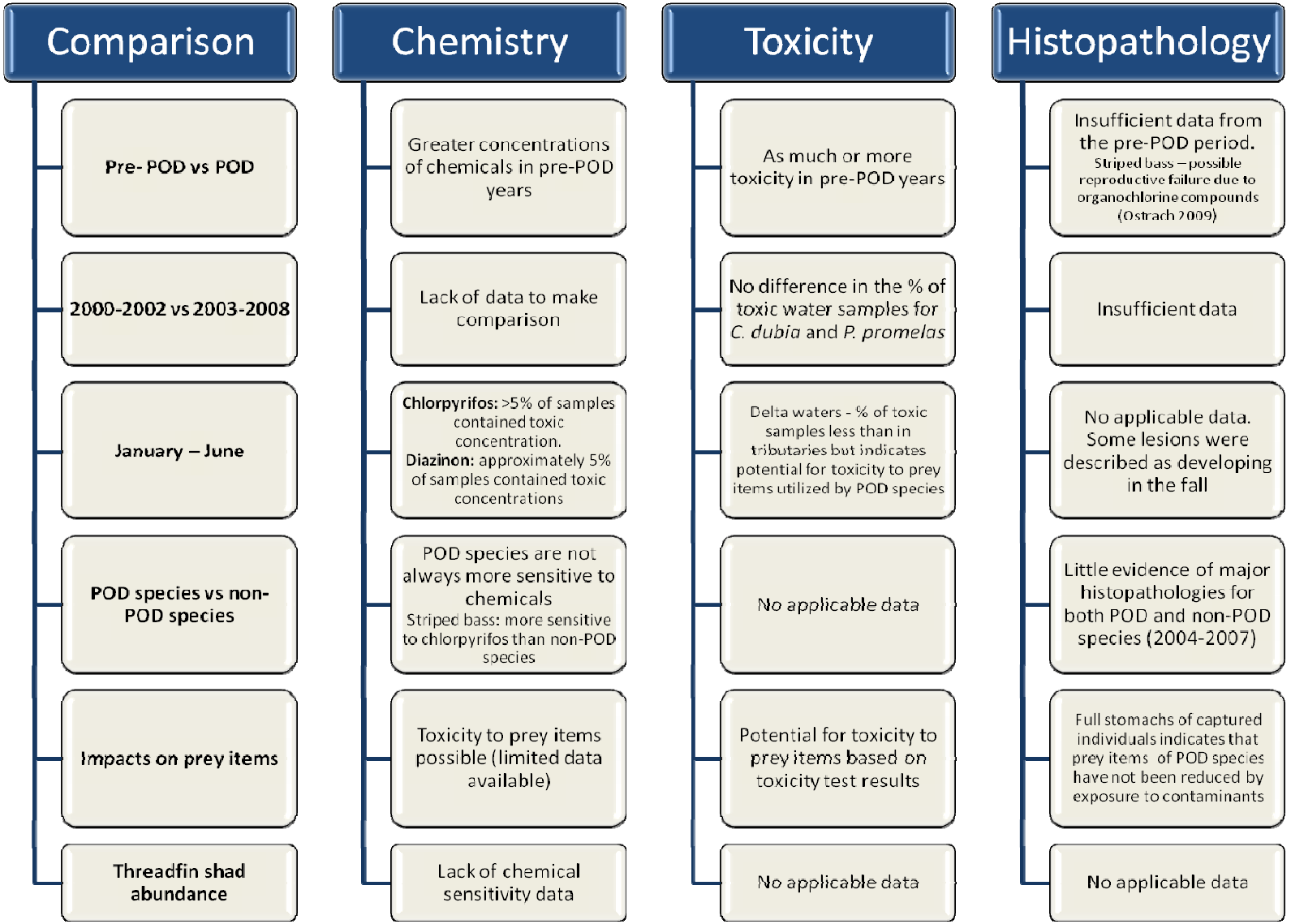
Data Type	Pre POD (before 2000)*		Step Decline (2000 – 2002)		Later POD (2003 – present)	
	Delta Channels	Outside of Delta	Delta Channels	Outside of Delta	Delta Channels	Outside of Delta
Chemistry†	Limited	Limited – Moderate	Extremely Limited	Extremely Limited	Limited	Moderate-Good
Toxicity	Limited	Moderate-Good	Extremely Limited	Extremely Limited	Moderate	Moderate-Good
Histopathology	Extremely Limited	Extremely Limited	None	None	Moderate	None

*Pre POD data was not included in the POD contaminant database; assessment is based on review by Fox and Archibald (1997).

†Contaminants including pesticides and other chemicals such as PAHs, PCBs, metals, etc.

The question about whether other aspects of POD species' life histories and demography, the success of non-POD pelagic species, and a review of the limited contaminants data can speak to the role of contaminants in the POD can still be posed. The series of hypotheses and predictions posed throughout this synthesis are summarized in Figure 12. A review of the life histories of the POD species suggests that exposure during larval and early juvenile periods would be effective in causing steep declines in abundance. With low survival of larvae and juvenile fish, additional small decreases in survival can have significant negative impacts on population growth rates. The lifespan and age at maturity of striped bass suggest that changes in early juvenile survival and adult survival can have significant impacts on population growth rate (see Velez-Espino *et al.* 2006). As all four POD species spawn during the late winter – spring period in the freshwater of the Delta, exposure to the same contaminant(s) during larval and early juvenile development could cause similar and significant population decreases across species. Although decreases in survival of adults cannot be ruled out as a cause of the decline, adults of the different species occupy different habitats making a single mechanism of exposure unlikely. Invoking contaminants as a cause of reduced adult survival at a level sufficient to cause severe population declines requires assumptions about the presence and effects of contaminants that are unlikely to be met.

Figure 11. Summary of hypothesis and predictions.



Recent analyses indicate that a major step decline in abundance occurred for three of the four POD species between 2000 and 2002. Two of the three POD species experiencing step declines continued to experience decreasing abundance after 2002. The remaining POD species not experiencing a step decline, instead experienced a steady decline in abundance through all of the POD years. One POD species, the threadfin shad, is experiencing increasing abundance after the step decline. Other species inhabiting the Delta, and presumably exposed to the same contaminants in the water column as the POD species, are experiencing increases in abundance leading to the question of whether contaminants could differentially affect POD and non-POD species. The response to this question lay in either differential exposure or differential dose-response relationships for three declining POD species compared to the one POD species experiencing a population increase and the non-POD species. If contaminants are responsible for the POD decline while at the same time not affecting the one POD species experiencing an increase in abundance and additional non-POD species within the Delta, at least one of the following must be true.

- Within the Delta during the period of spawning and larval and juvenile development, there are microscale differences in the concentrations of chemicals that correspond with the microscale differences in habitat utilized by the POD and non-POD species, i.e. POD species are exposed to water with concentrations of chemicals sufficient to cause decreased survival, while non-POD species are not exposed to that water. This requires strict habitat segregation among species within the Delta and that the flows moving water through the Delta result in separate exposure in the different habitats. Finally, it requires that the releases of chemicals into the Delta and from within the Delta be different and correspond to the differences in habitat.
- There is a differential sensitivity of POD and non-POD species to the chemicals to which all are exposed. POD species would necessarily be more susceptible to the effects of the chemicals than non-POD species such that differential population responses would be expected even with the same exposure to chemicals.
- There is a differential exposure and/or sensitivity to chemicals by different prey items used by POD and non-POD species. These differences result in differential mortality and/or recruitment to the zooplankton populations utilized by POD and non-POD species causing food limitation and decline in abundance of the POD species but not non-POD species.

WATER CHEMISTRY SYNTHESIS

There is a large amount of chemistry data available but the majority of the records were unusable for this review due to a lack of metadata and quality control information associated with the samples. Many samples were, and currently are analyzed for various constituents but the results are reported without accompanying detection or reporting limits. Additionally, very few monitoring programs report sufficient quality control data to evaluate the data properly. For example, blank, spiked and duplicate sample results are rarely reported making interpretation of results difficult. It is unknown if all these data will be of any use in the future to address other issues that may arise, but their usefulness is doubtful. Notable exceptions to this are monitoring data generated by the Surface Water Ambient Monitoring Program and the CVRWQCB's TMDL and Irrigated Lands Regulatory Programs.

After eliminating unusable data records, there are very few chemicals for which sufficient data are available to evaluate their role in the POD. Because the POD became recognized after the step decline period was past, monitoring for numerous constituents hypothesized to be responsible for the decline was initiated too late to understand if those constituents were present during the period when the POD species suffered significant decreases in abundance. Other constituents were monitored throughout the POD period but using detection and reporting limits that were too high to adequately assess their role as potential drivers of the decline.

Most of the chemicals for which monitoring data do exist were monitored at locations outside of the jurisdictional Delta calling into question the relevance of the monitoring data to the POD species. The search for data was extended to a 30 mile radius outside of the Delta because 30 miles was the distance water was assumed to travel in a day. Presumably, constituents measured upstream within 30 miles would reach the Delta within a day but due to dilution it is not known if those constituents enter the Delta at concentrations similar to those measured upstream. Also, in the summer irrigation season when flows are generally lower relative to winter high flow events, the one-day transit rule may not be correct especially for constituents located in smaller water bodies such as Orestimba Creek or Prairie Flower Drain in Stanislaus County. Consequently, there is very little understanding of what chemicals were present in the Delta during the POD period.

Increased monitoring for pyrethroids has not resulted in identification of a "smoking gun." The hundreds of samples analyzed for pyrethroids since 2004 have with a few exceptions, failed to detect the presence of pyrethroids, although such a result is not unexpected. For example, 675 measurements of bifenthrin in sample water were available in the database; one sample contained measureable amounts of bifenthrin and that sample was collected from Ulatis Creek @ Brown Rd, a location outside of the Delta (although draining to the Delta). The partitioning of pyrethroids would result in very small amounts of chemical in the dissolved phase. When pyrethroids are found in the dissolved phase, they undoubtedly will be present at concentrations below the detection limits employed in the analyses made to date. Monitoring of pyrethroids during the POD period was conducted employing detection limits too high to adequately characterize the concentration of pyrethroids in water. This is unfortunate as it is now known that pyrethroids are toxic to some aquatic organisms at concentrations as low as 2-4 ng/L range (Weston *et al.* in press). Also, because pyrethroids sorb to organic matter in the sample as well as sides of the containers in which the sample is stored, samples must be analyzed immediately or preserved with dichloromethane or the results may be biased low.

Overall, there is a lack of chemistry data from the Delta during the POD period to adequately determine if chemicals are wholly or partially responsible for the decline. There are few data available from the January – June period when the four POD species are present in the Delta as larval and juvenile fish. In addition, a review of historical data provided by Fox and Archibald (1997) indicates that similar concentrations of many chemicals, e.g. diazinon, were present in the Delta in the years prior to the POD.

TOXICITY SYNTHESIS

A review of toxicity data by Fox and Archibald (1997) was available for use in this analysis. The original data were not examined. The review indicates that toxicity was present at much higher levels in studies conducted between 1988 and 1995 compared to levels of toxicity observed in the POD period. Fish kills were common from 1965 to the early 1990s due to rice pesticides such as Ordram. When the rice industry increased holding times of water on rice fields, major fish kills stopped occurring. Consequently, it appears that acute toxicity is reduced in the POD period relative to the years preceding the POD. The limited number of TIEs that have been performed indicate a variety of causes but the majority of TIEs point to a metabolically activated pesticide such as diazinon or chlorpyrifos as the cause of the toxicity.

The exception to the reduced toxicity in the POD period is sediment toxicity which appears to be extremely elevated and has become the focus of many recent investigations. Sediment toxicity is common in many of the interior Delta Island drains, and many of the smaller tributaries to the San Joaquin River and Delta. It is possible that elevated flows could move the contaminated sediment from the tributaries to the Delta causing toxicity to the POD species. D. Weston has demonstrated that a very large percentage of the sediment toxicity he finds is a result of pyrethroids, with the percentage reaching almost 100% when the sediments are in urban creeks and storm drains. His most recent studies indicate that urban areas contribute a substantial amount of pyrethroid pesticides to the water column in the Delta from storm water runoff.

A lack of concurrent monitoring of water chemistry and toxicity in monitoring programs in the early POD period make the interpretation of toxicity data difficult. Fortunately, most of the monitoring programs from the recent past and those currently in progress conduct toxicity testing and water chemistry analyses concurrently, facilitating interpretation of toxicity data. When sufficient toxicity does occur, TIEs can be performed and the water chemistry data used to account for the toxicity in the sample. Such an approach has been useful in the CVRWQCB's Irrigated Lands Regulatory Program where it has been possible to identify specific chemicals responsible for toxicity of samples collected throughout the Central Valley including the Delta. Unfortunately, the samples in the Delta were collected from agricultural drains within islands and it is unknown if the concentration of chemicals in the drain water remains constant until the water is discharged to the Delta, or if mixing and dilution of the drain water with waters of the Delta are sufficient to reduce the risk of exposure to POD species. Given the large number of drain pumps in the Delta, it is possible that large quantities of agricultural chemicals are discharged to the Delta, but monitoring information from the POD period is insufficient to determine if this is true. Even today, there are relatively few samples collected from the Delta to determine if chemicals are present in the Delta in sufficient quantities to cause direct or indirect effects on POD species. Targeted monitoring for pyrethroids and toxicity over the last several years indicates that although some toxicity is present and is widespread geographically, it is unknown if it is at a level sufficient to cause the POD. Also, sediment toxicity is widespread in the small creeks found in urban areas around the Delta, but the relevance of sediment toxicity found at a location outside of the Delta to a pelagic food web and pelagic fish species in the Delta is unclear.

Because it appears that the level of acute toxicity during the POD years is much lower than the years preceding the POD, it is unlikely that acute toxicity to POD species or their prey items is responsible for the POD. However the overwhelming majority of the tests do not address

chronic toxicity that could be present as a result of the exposure to low concentrations of chemicals. An indication of chronic toxicity is possible by examining biomarkers. At a gross level, histopathologies are structural biomarkers of contaminant exposure, but additional biochemical indicators of contaminant exposure can be examined for evidence of exposure and effects. Unfortunately, there has been no systematic program for monitoring biochemical indicators of exposure and effects in the Delta. Fox and Archibald (1997) reference some biomarker data (e.g. hemoglobin, hematocrit, and brain acetylcholinesterase activity) from CDFG's rice pesticide monitoring program, but those data were not available for this study. Histopathology data are available on a limited basis.

HISTOPATHOLOGY SYNTHESIS

Data from the histopathology studies are equivocal in their ability to conclude whether contaminants are impacting POD species. The studies by Teh on Delta smelt indicate that with one exception, larval fish from the Napa River in the spring of 2006, there is no evidence of starvation among larval, juvenile, or adult Delta smelt. Stomachs contained zooplankton in most delta smelt collected and there were few lesions that are histopathologic indicators of starvation. Consequently, it is unknown if contaminants are impacting the food source of DS, but evidence from the limited period of the Teh studies suggests that there are limited or no impacts on prey items.

There is some evidence that delta smelt have been exposed to contaminants. While not all fish examined contained lesions typical of exposure to contaminants, fish collected at various times from Suisun Marsh, Napa River, and the South Delta had a combination of lesions typical of exposure to contaminants. The type of lesions seen and lesions absent suggested exposure occurred within a few weeks of collection as adults. Teh concluded that the lesions could affect survival. He also found some evidence of ova-testis indicating exposure to endocrine disrupting chemicals. Histopathology on splittail suggests lesions were the result of general health status and nutrition rather than exposure to contaminants, although splittail are considered insensitive to the effects of contaminants relative to other species. Ostrach's research indicates that striped bass are being exposed to numerous contaminants which are then passed from females to their eggs resulting in developmental difficulties for larval and juvenile fish. His research covers both the immediate pre-POD period and the late-POD period and suggests that these effects on striped bass have been occurring continuously since prior to the step decline.

It appears there are insufficient data to parameterize any statistical or physical model that might formally test the hypothesis that contaminants are a cause of the decline. Consequently Figure 12 uses the results and places them into a weight of evidence context. Of the 14 hypotheses presented in the figure, there is a positive response to 7 and a negative response to the remaining 7. The conclusion that can be drawn from these responses is that while contaminants are unlikely to be a major cause of the POD, they cannot be eliminated as a possible contributor to the decline. Unfortunately, further research can address only our understanding of the relative sensitivity of POD species to various contaminants in the system, it cannot recreate history.

RECOMMENDATIONS

1. **A long-term monitoring program should be developed that can identify possible involvement of contaminants in phenomena such as the POD.** Given the sophisticated statistical techniques used to analyze the POD specifically, and the variability in abundance of organisms in general, it is unlikely that monitoring of contaminants alone can portend problems such as the POD. However, a long-term water quality monitoring program will allow the rapid identification of new and emerging contaminants that might be toxic to biota, tracking increased discharge of current use chemicals, and perhaps potentially new exposure to species considered critical to the functioning of the Delta ecosystem. Toxicity testing should be conducted in association with water chemistry. Because of the time required to perform histopathology analyses, histopathology should be limited to fewer targeted samples. Although it is recommended that a long-term monitoring program be developed, existing baseline and regulatory-based programs like NPDES and the ILRP should be continued. Specific recommendations about testing and interpretation of data in a long term monitoring program include:

    - Because of their obvious limitations with regard to species sensitivity, representation of resident species, exposure scenarios, and sublethal effects, toxicity tests should not be used as the final quantitative indicator of absolute ecological impairment, but as one line of evidence or first tier investigation.
    - Every attempt should be made to use ecologically significant, sublethal toxicity endpoints, such as growth, reproductive success, and swimming ability.

- Toxicity testing should be accompanied by a toxicity test review process that is an important part of an overall quality assurance program (see review by D. Denton in Appendix IV for further details).
- Toxicity testing should utilize a bioequivalence testing approach that provides a consistent threshold for determining toxicity, controls the test power, and provides a streamlined data analysis approach.
- Biomarkers can provide important information on biologically active toxicants present at extremely low concentrations or as mixtures, and therefore difficult to detect by analytical chemistry. Well characterized biomarkers should be integrated into monitoring efforts. The report on biomarkers by Anderson *et al*. (2007) provides an overview of appropriate biomarkers that could be used in a long-term monitoring program. See comments by D. Denton in Appendix IV for additional information.
- Because the relative sensitivities of POD species and species used in toxicity testing are not known, using additional test species such as rainbow trout is encouraged. A rainbow trout assay has been developed and published (Miller et al. 2009) and used to screen water from the Sacramento River system.
- Where possible, *in situ* methods can be used to monitor ambient toxicity. In situ testing can integrate the toxicity signal over time. However, due to cost and effort, their use should be carefully validated prior to widespread implementation including methods for capturing toxic pulses for TIEs.
- Testing methods used have not been adequate to detect toxicity due to pyrethroid insecticides. Recent data indicates that *C. dubia* is far less sensitive to pyrethroids than the amphipod species *H. azteca,* while both species are highly sensitive to organophosphate insecticides. It is recommended that future water column toxicity testing include *H. azteca* as a test species; test protocols are available, organisms can be purchased commercially, and recently developed pyrethroid-focused TIE methods should be applied.
- Chemical analyses for pyrethroids should utilize DCM as a preservative or the analyses should be performed immediately on receipt of the samples. Container adsorption is known to reduce the concentration of pyrethroids in sample bottles (Wheelock et al. 2005) and lack of preservation could result in results that are biased low.
- Detection limits of analytical chemistry must be low enough to detect contaminants at concentrations toxic to sensitive aquatic species, for example, <3 ng/L for pyrethroid insecticides, and <10 ng/L for organophosphate insecticides.
- Current and past monitoring programs are spatially and geographically heterogeneous and often use different toxicity endpoints. A comprehensive, cohesive toxicity monitoring program using a specified nomenclature for site

identification is urgently needed. It is difficult at best to compare results of different programs.

- Sampling sites in small streams, sloughs and channels should be coordinated with sites in large channels to study contaminant fate and the geographic and temporal extent of impacted aquatic habitat in the Delta. For ambient monitoring, knowledge of land use, pesticide applications, and the hydrology is required to effectively establish monitoring stations and develop an adequate time frame for monitoring. Coordinating the monitoring of the storm hydrograph in storm drains by the permitted industrial and municipal entities is important because of the potential for the movement of significant quantities of contaminants to surface waters (see Miller *et al*. 2005). See comments by D. Denton in Appendix IV for further information.
- There is mounting evidence that sediment toxicity due to pyrethroid insecticides is widespread in small urban and agricultural surface waters of California, but other chemicals may contribute significantly to sediment toxicity in the Delta. It is therefore recommended to identify sources and pathways of how toxic chemicals enter Delta waterways in order to develop and promote preventative best management practices.
- Sediment TIEs should be integrated into monitoring programs to confirm and expand findings obtained by the Irrigated Lands Program and a recent toxicity survey (Sediment Quality Objectives – Phase II).
- It is recommended that toxicity testing at Sacramento River at Freeport and/or Hood be continued to monitor toxicity of Sacramento River water just before it enters the Delta.
- A long-term monitoring program should develop an approach to deal with repeated toxicity at a site. If a site repeatedly is toxic, there should be additional sampling to understand the duration, frequency, and magnitude of the toxicity. This strategy may involve dilution series and TIE analyses as well as resampling at the site. See comments by D. Denton in Appendix IV for further information.
- Data conversion to electronic files and a comprehensive review is needed for all NPDES toxicity data. Toxicity data should be put into perspective using information on volume of effluent discharged and percentage of flow in the receiving water body. These water bodies should be categorized with respect to their ecological habitat value, particularly with respect to species of concern. Included in the review should be a detailed discussion of the toxic effects of ammonia and other wastewater-associated contaminants, for example insecticides and EDCs.

2. **Develop a workable conceptual model of the Delta that combines the critical physical forcing functions and the biotic components of interest.** The long-term monitoring program should be developed based on a conceptual model of Delta ecosystem function

that allows sufficient interpretation of the system. The current conceptual model used by the POD team is too broad and insufficiently detailed to guide monitoring. It is recognized that the current model was not developed to guide a long term monitoring program. Models such as the DRERIP are too detailed. Chemical models should be developed to assist in understanding spatial and temporal trends in contaminant loading. Such models can be used to assess the effectiveness of the monitoring program and assist in the evaluation of proposed mitigation measures. See comments by D. Denton in Appendix IV for more information. Those comments identify several critical aspects of the model output.

3. **The long term monitoring program should have ongoing data interpretation and analysis as a co-equal goal along with sampling and analysis.**

    - The interpretation should be aimed at both policy makers and scientists.

    - Data submissions should be done frequently, preferably on a quarter by quarter basis with data no longer than one quarter behind in reporting. Program reports should be submitted within 3-6 months after the end of the monitoring year.

4. **Data from all water quality data generators in the Delta should be submitted to the State's Regional Data Center in SWAMP-comparable format.** SWAMP comparability should be interpreted strictly which may require a review of all data to determine that the data are SWAMP-comparable. This review could be performed by agency project managers, permit writers, project personnel, or third party personnel dedicated to review. The requirement for SWAMP comparability also necessitates a methodology by which generators can provide quality data in a cost effective and time efficient manner.

5. **Numerous research needs exist related to the effects of contaminants in the Bay-Delta system.** The relevance of the available toxicity data for POD fish species is difficult to assess as little information is available for these species. It is clear that invertebrate toxicity in the water column as well as sediments is widespread in waterways of the Central, South and North Delta including the mainstem rivers. Toxicity has, in part, been traced to insecticides, dominantly OP and pyrethroid insecticides, ammonia, cationic metals and herbicides, and there is some evidence that contaminants are transported into larger channels.

    - Establish a Regional Center for TIE Support to develop methods to identify unknown ambient toxicants. Such a center was identified as a research priority by SETAC in 2005 to address method development issues in TIE analyses. The center could be further enhanced by the addition of a center for the development of analytical methods to identify toxicants that are poorly characterized chemically.

- It is likely that invertebrate species, which constitute an important food source for fish species of concern, are affected by contaminants in small Delta waterways, and to a lesser degree in large channels of the Delta. The potential implications of sublethal amphipod toxicity for fitness of Delta organisms are unknown, it is possible that some species are more sensitive to contaminants than *H. azteca,* e.g. copepods. More information on relative sensitivity to contaminants is needed. As more information on environmental concentrations of specific contaminants emerges, this data will be valuable for focusing future research and monitoring efforts
- The significance of small Delta water bodies for the survival and rearing of POD fish species determines the extent of the impact of ambient toxicity. More information on the effects of contaminants of concern on embryo development, hatching and rearing is needed.
- Available toxicity and chemistry data from before the year 2000 should be reviewed and summarized in greater detail than provided in this report. A report already exists (Fox and Archibald, 1997) for the period 1965-1995.
- A better understanding is needed with regard to sublethal effects (e.g. swimming ability, behavior) and their consequences on fitness of fish in the wild.
- A better understanding is needed regarding the toxic effects of contaminant mixtures from different sources (e.g. storm water runoff, treated wastewater, irrigation return water). The USGS and other monitoring entities have confirmed that multiple potentially toxic chemicals are frequently present in ambient samples. A greater understanding of the toxicity of contaminant mixtures can be derived from the application of successful TIE analyses, and can be facilitated by the Regional Center described above.
- The effects of endocrine disrupting chemicals on fish species of concern need to be better understood.
- The impact of sediment toxicity on invertebrate communities and the food web needs to be understood to assess its risk to POD species.

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POD Synthesis Report Appendices

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TABULATED WATER CHEMISTRY RESULTS

Table 1. POD Contaminant Database – Data Sources (chemistry data).

Data Source	Data Source Name	Description
UCD RDC	University of California Davis, Regional Data Center	The UCD RDC is one of four Regional Data Centers in California that manages data to be transferred to the online California Data Exchange Network (CEDEN). Data housed at the UCD RDC is considered "SWAMP Comparable" and includes associated quality assurance data and result qualifiers.
BDAT	Bay Delta and Tributaries	BDAT is an online database that was developed by the Department of Water Resources. Data was queried from BDAT and reformatted.
NPDES	National Pollutant Discharge Elimination System	Data obtained from NPDES permits.
RB5	Central Valley Regional Water Quality Control Board - Region 5	Surface Water Ambient Monitoring Project (SWAMP) data from Region 5.

Table 2. POD Contaminant Database – Project Descriptions. Project IDs, project descriptions and data sources for chemistry results (water and sediment).

Data Source	ProjectID	Project Description
UCD RDC	02TM5001	TMDL pesticide study initiated in 2002, contracted by the Regional Board
UCD RDC	04AG5001	AgWaiver project to evaluate agricultural inputs to the waters of the states within Region 5 initiated in 2004, contracted by the Regional Board
UCD RDC	04ES5001	East San Joaquin Water Quality Coalition under the Irrigated Lands Regulatory Program, initiated in 2004
UCD RDC	04SJ5001	San Joaquin Water Quality Coalition under the Irrigated Lands Regulatory Program, initiated in 2004
UCD RDC	06GP5P50	Prop50 Grant, A comparison of the effects of orchard floor management practices on pesticide runoff to surface waters funded by SWCRB, Contact Anja Wehrmann abwehrmann@ucdavis.edu
UCD RDC	07TM5001	TMDL pesticide study; original project initiated in 2002 and re-contracted in 2007, contracted by the Regional Board
BDAT	CALFEDMT	Mercury Transport
BDAT	DHS CDWD	DHS, California Drinking Water Data
BDAT	EMPLTCW CDWR	Environmental Monitoring Program - Long-term monitoring WQ data / Continuous Water Quality Monitoring, California Department of Water Resources
BDAT	EMPSSWQC CDWR	Environmental Monitoring Program - Special Study WQ data / Continuous Water Quality Monitoring, California Department of Water Resources
BDAT	EMPSSWQCC DWR	Environmental Monitoring Program - Special study WQ data / Comparison of chlorophyll extraction method, California Department of Water Resources
BDAT	R2JSM DFG	Region 2, Juvenile Salmon Monitoring, California Department of Fish and Game
BDAT	SJRDO CDWR	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO City Of Stockton	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO CRWQCV Region 5	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO LBNL	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO Turlock	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO UOP	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO USBR	San Joaquin River Dissolved Oxygen Study
BDAT	SJRDO USGS	San Joaquin River Dissolved Oxygen Study
BDAT	SJSM USFWS	Stockton Office, Juvenile Salmon Monitoring, USFWS
BDAT	SMFM UCD	Suisun Marsh Fisheries Monitoring
NPDES	BWWTP_CB	Brentwood Waste Water Treatment Plant

Data Source	ProjectID	Project Description
NPDES	DRWMR LPW	NPDES Wastewater Treatment Facility, NPDES Discharger Receiving Water Monitoring Report, City of Lodi Public Works Department
NPDES	DRWMR MPW_WQCF	City of Manteca Public Works Department, NPDES Discharger Receiving Water Monitoring Report, City of Manteca Wastewater Quality Control Facility
NPDES	DRWMR MPW_WQCFP	City of Manteca Public Works Department, NPDES Discharger Receiving Water Monitoring Report, City of Manteca Wastewater Quality Control Facility
NPDES	DRWR STKPW_STKRCSD	City of Stockton Regional Wastewater Treatment Plant, NPDES Discharger Receiving Water Monitoring Report, Stockton Regional County Sanitation District
NPDES	MWQCF_CM	City of Modesto Water Quality Control Facility
NPDES	SRWTP NPDES	Sacramento Regional Wastewater Treatment Plant NPDES Monitoring
NPDES	SRWTP_P4 NPDES	Sacramento Regional Wastewater Treatment Plant NPDES Monitoring, 4xyearly
NPDES	TMUDSWMD	Municipal Utilities Department (MUD) Storm Water Management Division , City of Stockton and County of San Joaquin Storm Water Management Program (SWMP)
NPDES	TWWTP_CT	Turlock Waste Water Treatment Plant
NPDES	WDR_RVPW	City of Rio Vista, Waste Discharge Reports, Rio Vista Public Works
RB5	03AG5001	Ag Waiver Phase I monitoring
RB5	AD_RB5S	RWQCB Region5 Surface Water Ambient Monitoring Project
RB5	SWAMP_RB2	RWQCB Region5 Surface Water Ambient Monitoring Project
RB5	SWAMP_RB5L	RWQCB Region5 Surface Water Ambient Monitoring Project
RB5	SWAMP_RB5S	RWQCB Region5 Surface Water Ambient Monitoring Project
RB5	SWAMP_SB	RWQCB Region5 Surface Water Ambient Monitoring Project

Table 3. POD Contaminant Database – Sediment and Water Chemistry Data used in analysis. Summary of sample water chemistry results by analyte name (all projects). Results are tabulated by start and end date of samples, result counts, minimum of results with non detects quantified as one half the MDL, minimum of result and maximum of result.

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
WATER CHEMISTRY							
1,1-dichloroethene	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.49
1,2-Benzanthracene	µg/L	1/7/2003	1/7/2003	1	0.12	0.12	0.12
1,2-diphenylhydrazine	µg/L	1/23/2002	8/7/2007	53	0.3	-1.2	0.9
1,3-dichloropropene(total)	µg/L	1/10/2002	8/17/2007	61	0.22	-0.5	0.3
13C-1,2,3,4,6,7,8-HpCDD	pg/L	2/17/2004	2/17/2004	2	1110	1110	1560
13C-1,2,3,4,6,7,8-HpCDF	pg/L	11/12/2003	2/17/2004	4	1030	1030	1620
13C-1,2,3,4,7,8,9-HpCDF	pg/L	11/12/2003	2/17/2004	4	1220	1220	1680
13C-1,2,3,4,7,8-HxCDD	pg/L	11/12/2003	2/17/2004	4	1120	1120	1740
13C-1,2,3,4,7,8-HxCDF	pg/L	11/12/2003	2/17/2004	4	1100	1100	1570
13C-1,2,3,6,7,8-HxCDD	pg/L	11/12/2003	2/17/2004	4	1010	1010	1680
13C-1,2,3,6,7,8-HxCDF	pg/L	11/12/2003	2/17/2004	4	1060	1060	1680
13C-1,2,3,7,8,9-HxCDF	pg/L	11/12/2003	2/17/2004	4	1200	1200	1620
13C-1,2,3,7,8-PeCDD	pg/L	11/12/2003	2/17/2004	4	1050	1050	1740
13C-1,2,3,7,8-PeCDF	pg/L	11/12/2003	2/17/2004	4	1120	1120	1620
13C-2,3,4,6,7,8-HxCDF	pg/L	11/12/2003	2/17/2004	4	1170	1170	1670
13C-2,3,4,7,8-PeCDF	pg/L	11/12/2003	2/17/2004	4	1130	1130	1710
13C-2,3,7,8-TCDD	pg/L	11/12/2003	2/17/2004	4	1200	1200	1680
13C-2,3,7,8-TCDF	pg/L	11/12/2003	2/17/2004	4	1220	1220	1690
13C-OCDD	pg/L	11/12/2003	2/17/2004	4	1610	1610	3530
13C-OCDF	pg/L	11/12/2003	2/17/2004	4	1840	1840	3350
2,4-D	µg/L	1/23/2002	12/18/2002	14	5	-10	5.3
2-chloroethylvinyl ether	µg/L	1/10/2002	8/17/2007	91	0.1	-2	0.32
3,3'-dichlorobenzidine	µg/L	1/23/2002	8/17/2007	56	0.3	-5	0.6
3,4-Benzofluoranthene	µg/L	1/7/2003	1/7/2003	1	0.11	0.11	0.11
37Cl-2,3,7,8-TCDD	pg/L	2/17/2004	2/17/2004	2	583	583	627
4,4'-DDD	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
4,4'-DDE	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
4,4'-DDT	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
Acenaphthene	µg/L	1/23/2002	8/17/2007	61	0.00859	-2.4	0.17
Acenaphthylene	µg/L	1/23/2002	8/17/2007	56	0.02	-10	0.03
Acetone	µg/L	6/10/2002	11/6/2002	6	5	-10	-10
Acrolein	µg/L	1/10/2002	8/17/2007	90	0.56	-20	3.3
acrylonitrile	µg/L	1/10/2002	8/17/2007	90	0.33	-2	1.6

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Alachlor	µg/L	1/23/2002	3/17/2003	15	0.3	-1	0.3
Aldrin	µg/L	6/10/2002	8/17/2007	49	0.005	-0.1	0.005
Aluminum	µg/L	7/1/1986	1/17/2008	392	0.05	-100	5200
Aluminum	mg/L	10/21/1998	2/9/2005	54	0.03	0.03	4.3
Ammonia	mg/L	1/13/1999	1/17/2008	2081	0	-1	16
Ammonia & Organic Nitrogen	mg/L	7/12/2000	11/16/2001	94	0.041	0.041	5.3
Ammonia as N	µg/L	6/10/2002	10/21/2002	8	250	-1000	-500
Ammonia as N	mg/L	11/7/1999	2/26/2008	1582	0.0005	-0.07	31
Ammonia as NH3	mg/L	3/26/2003	8/26/2003	129	0.05	-0.1	8
Anthracene	µg/L	1/23/2002	8/17/2007	58	0.0117	-10	0.16
Antimony	µg/L	12/26/2001	8/17/2007	180	0.01	-10	134
Antimony	mg/L	10/21/1998	10/25/2005	52	0.005	0.005	0.2
Arsenic	µg/L	8/14/1984	1/17/2008	709	0.01	-5	13
Arsenic	mg/L	10/21/1998	2/23/2006	58	0.0021	0.0021	3.4
Atrazine	µg/L	1/23/2002	3/17/2003	17	0.02	-1	0.8
Azinphos methyl	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
Barban	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
Barium	µg/L	3/19/1986	8/17/2007	125	0.09	0.09	450
Barium	mg/L	10/21/1998	4/24/2001	50	0.046	0.046	0.73
Bentazon	µg/L	1/23/2002	12/18/2002	14	0.84	-2	0.84
Benz(a)anthracene	µg/L	9/18/2001	8/17/2007	61	0.00949	-10	0.12
Benzene	µg/L	1/10/2002	8/17/2007	90	0.06	-0.5	0.3
benzidine	µg/L	1/23/2002	8/7/2007	54	0.3	-6	3
Benzo(a)pyrene	µg/L	10/2/2001	8/17/2007	60	0.0146	-2.4	0.09
Benzo(b)fluoranthene	µg/L	10/2/2001	3/22/2006	18	0.0104	0.0104	0.11
Benzo(e)pyrene	µg/L	10/2/2001	6/13/2005	5	0.0101	0.0101	0.134
Benzo(g,h,i)perylene	µg/L	1/23/2002	8/17/2007	63	0.0151	-5	0.255
Benzo(k)fluoranthene	µg/L	10/2/2001	8/17/2007	63	0.00877	-3	0.16
Beryllium	µg/L	12/26/2001	8/17/2007	150	0.05	-10	0.3
Beryllium	mg/L	10/21/1998	10/25/2005	54	0.0005	0.0005	0.06
BHC-alpha	µg/L	6/10/2002	8/17/2007	47	0.05	-0.1	-0.1
BHC-beta	µg/L	6/10/2002	8/17/2007	50	0.015	-0.1	0.043
BHC-delta	µg/L	6/10/2002	8/17/2007	54	0.003	-0.2	0.015
BHC-gamma (Lindane)	µg/L	6/10/2002	8/17/2007	49	0.015	-0.2	0.13
Bifenthrin	µg/L	7/8/2004	2/26/2008	563	0.00025	-0.006	0.43
Biphenyl	µg/L	4/21/2003	6/13/2005	5	0.00891	0.00891	0.0232
Bis(2-chloroethoxy)methane	µg/L	1/23/2002	8/17/2007	56	0.3	-5	0.9

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Bis(2-chloroethyl)ether	µg/L	1/23/2002	8/17/2007	56	0.3	-2.4	0.7
Bis(2-chloroisopropyl) ether	µg/L	1/23/2002	8/17/2007	56	0.6	-10	1
Bis(2-ethylhexyl)phthalate	µg/L	1/23/2002	10/3/2007	75	0.3	-5	25
Bolstar	µg/L	2/17/2002	3/6/2002	6	0.1	0.1	1.4
Boron	µg/L	7/24/1984	8/17/2007	132	0.12	-100	11000
Boron	mg/L	10/2/1995	8/30/2007	11872	0.01	0.01	20
Bromide	mg/L	7/1/1986	7/1/1986	1	0.07	0.07	0.07
Bromobenzene	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Bromodichloromethane	µg/L	1/10/2002	11/6/2007	150	0.06	-0.5	25.7
Bromoform	µg/L	1/10/2002	10/3/2007	133	0.04	-2	1
Bromomethane	µg/L	1/10/2002	8/17/2007	90	0.05	-2	0.5
Bromophenyl phenyl ether, 4-	µg/L	1/23/2002	8/17/2007	56	0.4	-10	2
Butanone, 2-	µg/L	6/10/2002	8/17/2007	8	0.25	-1	-0.5
Butyl benzyl phthalate	µg/L	1/23/2002	8/17/2007	56	0.1	-10	0.8
Butylbenzene, n-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Butylbenzene, sec-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Butylbenzene, tert-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Cadmium	µg/L	4/16/1984	8/17/2007	349	0.002	-2	60
Cadmium	mg/L	10/21/1998	10/25/2005	54	0.0003	0.0003	0.03
Carbaryl	µg/L	6/10/2002	2/26/2008	583	0.0035	-10	0.61
Carbofuran	µg/L	1/23/2002	2/26/2008	508	0.005	-10	2.31
Carbon tetrachloride	µg/L	1/10/2002	8/17/2007	92	0.06	-0.5	0.6
Carbophenothion	µg/L	9/26/2001	9/26/2001	1	0.054	0.054	0.054
Chlordane	µg/L	1/23/2002	8/17/2007	55	0.005	-1	0.1
Chlordane, Alpha-	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
Chlordane, gamma-	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
Chlordane, trans-	µg/L	4/22/2003	4/22/2003	1	0.001	0.001	0.001
Chlordene, gamma-	µg/L	6/18/2002	6/18/2002	1	0.0015	0.0015	0.0015
Chloro-3-methylphenol, 4-	µg/L	1/23/2002	8/17/2007	56	0.3	-5	0.93
Chlorobenzene	µg/L	1/10/2002	8/17/2007	79	0.06	-2	0.3
Chlorodibromomethane	µg/L	1/7/2003	1/7/2003	1	0.3	0.3	0.3
Chloroethane	µg/L	1/10/2002	8/17/2007	90	0.07	-2	0.34
Chloroform	µg/L	1/10/2002	11/6/2007	256	0.027	-2	43
Chloromethane	µg/L	1/10/2002	8/17/2007	93	0.04	-2	0.7
Chloronaphthalene, 2-	µg/L	1/23/2002	8/17/2007	56	0.3	-10	0.6
Chlorophenol, 2-	µg/L	1/23/2002	8/17/2007	56	0.4	-2.4	1.2
Chlorophenyl phenyl ether, 4-	µg/L	1/23/2002	8/17/2007	56	0.4	-5	2.4

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Chlorotoluene, 2-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Chlorotoluene, 4-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Chlorpropham	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
Chlorpyrifos	µg/L	2/10/2000	2/26/2008	1032	0.001295	-1	1.7
Chromium	µg/L	4/1/1991	8/17/2007	1282	0.039	-20	110
Chromium	mg/L	10/21/1998	10/25/2005	54	0.002	0.002	4.6
Chromium (VI) LowLevel	µg/L	1/29/2002	12/4/2002	11	0.2	0.2	0.8
Chromium VI	µg/L	1/10/2002	3/17/2004	34	0.16	-5	2
chromium, hexavalent	µg/L	4/18/2007	4/18/2007	1	11	11	11
Chrysene	µg/L	9/19/2001	8/17/2007	62	0.00743	-5	0.14
Chrysenes, C1 -	µg/L	4/21/2003	6/13/2005	4	0.00679	0.00679	0.156
Chrysenes, C2 -	µg/L	4/21/2003	6/13/2005	4	0.00612	0.00612	0.203
Chrysenes, C3 -	µg/L	4/21/2003	6/13/2005	3	0.00917	0.00917	0.38
cis-1,2-Dichloroethene	µg/L	1/10/2002	8/17/2007	91	0.05	-0.5	0.44
Copper	µg/L	3/7/1985	2/26/2008	2408	0.0025	-10	4403
Copper	mg/L	10/21/1998	10/25/2005	28	0.003	0.003	4.5
Cyanide	µg/L	1/10/2002	8/17/2007	73	0.0025	-5	5
Cyanide	mg/L	4/9/2002	8/7/2007	21	0.0006	-5	0.0006
Dacthal	µg/L	9/26/2001	6/18/2002	3	0.0015	0.0015	0.005
Dalapon	µg/L	1/23/2002	12/18/2002	14	1.6	-10	10
DDD (unsp.)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	-0.1
DDD(o,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.005
DDD(p,p')	µg/L	7/8/2004	2/26/2008	516	0.0005	-0.003	0.01
DDE (unsp.)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.1	-0.1
DDE(o,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.005
DDE(p,p')	µg/L	4/8/2002	2/26/2008	521	0.0005	-0.004	0.48
DDMU(p,p')	µg/L	9/18/2001	6/18/2002	3	0.0015	0.0015	0.002
DDT (unsp.)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	0.5
DDT(o,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.011
DDT(p,p')	µg/L	4/8/2002	2/26/2008	520	0.001	-0.007	0.4
Demeton - O and - S	µg/L	2/9/2005	12/14/2006	29	0.047	0.047	0.05
Demeton-s	µg/L	1/8/2001	12/5/2001	7	0.2	0.2	2.4
Di(2-ethylhexyl)adipate	µg/L	1/23/2002	8/7/2007	47	0.51	-6	0.51
Diazinon	µg/L	11/7/1999	2/26/2008	1108	0.0015	-0.5	2.5
Dibenz(a,h)anthracene	µg/L	4/11/2002	4/21/2003	3	0.035	0.035	0.0448
Dibenzo(a,h)anthracene	µg/L	1/23/2002	8/17/2007	56	0.03	-2.4	0.04
Dibenzothiophene	µg/L	4/21/2003	4/21/2003	1	0.0933	0.0933	0.0933

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Dibenzothiophenes, C1 -	µg/L	1/21/2003	4/12/2005	13	0.00641	0.00641	0.316
Dibenzothiophenes, C2 -	µg/L	1/21/2003	6/13/2005	17	0.00565	0.00565	0.623
Dibenzothiophenes, C3 -	µg/L	1/21/2003	4/12/2005	10	0.0075	0.0075	0.411
Dibromo-3-Chloropropane, 1,2-(DBCP)	µg/L	1/23/2002	10/21/2002	12	0.005	-0.01	0.007
Dibromochloromethane	µg/L	1/10/2002	11/6/2007	138	0.02	-0.5	7
Dibromomethane	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Dicamba	µg/L	6/10/2002	10/21/2002	8	0.75	-1.5	-1.5
Dichlorobenzene, 1,2-	µg/L	1/10/2002	8/17/2007	89	0.05	-2	0.5
Dichlorobenzene, 1,3-	µg/L	1/10/2002	8/17/2007	89	0.07	-2	0.3
Dichlorobenzene, 1,4-	µg/L	1/10/2002	8/17/2007	92	0.06	-2	0.53
Dichlorobromomethane	µg/L	1/7/2003	1/7/2003	1	0.2	0.2	0.2
Dichlorodifluoromethane	µg/L	6/10/2002	12/14/2006	36	0.06	-1	0.3
Dichloroethane, 1,1-	µg/L	1/10/2002	8/17/2007	89	0.05	-2	0.34
Dichloroethane, 1,2-	µg/L	1/10/2002	8/17/2007	90	0.06	-1	0.2
Dichloroethylene, 1,1-	µg/L	6/10/2002	8/17/2007	49	0.25	-2	0.42
Dichloroethylene, trans 1,2-	µg/L	1/7/2003	1/7/2003	1	0.43	0.43	0.43
Dichloromethane	µg/L	6/10/2002	8/17/2007	63	0.07	-2	1.8
Dichlorophenol, 2,4-	µg/L	1/23/2002	8/17/2007	56	0.3	-3	0.9
Dichloropropane, 1,2-	µg/L	1/10/2002	8/17/2007	91	0.05	-1	0.5
Dichloropropane, 1,3-	µg/L	6/10/2002	11/6/2002	6	0.25	-0.5	-0.5
Dichloropropane, 2,2-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Dichloropropene, 1,2-	µg/L	6/10/2002	11/6/2002	6	0.25	-0.5	-0.5
Dichloropropene, cis 1,3-	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.25
Dichloropropene, trans 1,3-	µg/L	1/7/2003	12/14/2006	31	0.05	0.05	0.3
Dichlorotrifluoroethane	µg/L	1/7/2003	12/14/2006	31	0.06	0.06	0.4
Dichlorvos	µg/L	11/29/2001	11/29/2001	1	0.13	0.13	0.13
Dieldrin	µg/L	6/10/2002	2/26/2008	572	0.0005	-0.1	0.11
Diethyl phthalate	µg/L	1/23/2002	8/17/2007	57	0.4	-2.4	4.9
Dimethoate	µg/L	6/10/2002	3/17/2003	11	5	-10	-10
Dimethyl phthalate	µg/L	1/23/2002	8/17/2007	56	0.4	-2.4	0.7
Dimethylnaphthalene, 2,6-	µg/L	4/12/2005	6/13/2005	2	0.0071	0.0071	0.0271
Dimethylphenol, 2,4-	µg/L	1/23/2002	8/17/2007	56	0.3	-2.4	1.1
Di-n-butyl phthalate	µg/L	1/23/2002	8/17/2007	57	0.2	-10	1
Dinitro-2-methylphenol, 4,6-	µg/L	1/23/2002	8/17/2007	56	0.4	-12.1	2
Dinitrophenol, 2,4-	µg/L	1/23/2002	8/17/2007	56	0.3	-12.1	3.9
Dinitrotoluene, 2,4-	µg/L	1/23/2002	8/17/2007	56	0.3	-5	0.9
Dinitrotoluene, 2,6-	µg/L	1/23/2002	8/17/2007	56	0.3	-5	0.6

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Di-n-octyl phthalate	µg/L	1/23/2002	8/17/2007	56	0.4	-10	0.9
Dinoseb	µg/L	1/23/2002	12/18/2002	14	0.49	-2	0.49
Dioxathion	µg/L	9/26/2001	9/26/2001	1	0.04	0.04	0.04
Diquat	µg/L	1/23/2002	1/7/2003	11	0.2	-0.4	0.8
Dissolved Organic Carbon	mg/L	7/11/2000	6/14/2005	271	0.7	0.7	23.9
Disulfoton	µg/L	11/7/1999	12/14/2006	56	0.012	0.012	1.2
Diuron	µg/L	6/10/2002	2/26/2008	505	0.001	-4	81
Endosulfan I	µg/L	9/18/2001	8/17/2007	51	0.007	-0.5	0.01
Endosulfan II	µg/L	6/10/2002	8/17/2007	49	0.01	-0.5	0.01
Endosulfan sulfate	µg/L	6/10/2002	8/17/2007	48	0.15	-1	-0.3
Endothal	µg/L	1/23/2002	1/7/2003	11	19	-45	19
Endrin	µg/L	6/10/2002	8/17/2007	50	0.005	-0.2	0.01
Endrin Aldehyde	µg/L	6/10/2002	8/17/2007	50	0.01	-0.5	0.015
Endrin Ketone	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
EPTC	µg/L	11/7/1999	11/7/1999	3	0.21	0.21	0.39
Ethion	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
Ethylbenzene	µg/L	1/10/2002	8/17/2007	89	0.06	-2	0.4
Ethylene Dibromide	µg/L	6/10/2002	10/21/2002	8	0.01	-0.02	-0.02
Fensulfothion	µg/L	11/29/2001	11/29/2001	6	0.36	0.36	3.4
Fenthion	µg/L	1/8/2001	1/8/2001	1	0.33	0.33	0.33
Fenuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
Fipronil	µg/L	4/19/2007	7/12/2007	12	0.025	-0.05	0.402
Fluometuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
Fluoranthene	µg/L	1/23/2002	8/17/2007	63	0.00544	-10	0.0794
Fluoranthene/Pyrenes, C1 -	µg/L	4/21/2003	6/13/2005	8	0.00516	0.00516	0.106
Fluorene	µg/L	9/19/2001	8/17/2007	61	0.013	-10	0.035
Fluorenes, C1 -	µg/L	4/21/2003	4/12/2005	6	0.007	0.007	0.0568
Fluorenes, C3 -	µg/L	4/21/2003	4/12/2005	10	0.00714	0.00714	0.102
Fluorescence	NR	2/6/2002	12/22/2004	63	0	0	49.42
Fonofos	µg/L	6/17/2002	6/18/2002	4	0.03	0.03	0.03
gamma-BHC (Lindane)	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
Glyphosate	µg/L	1/23/2002	10/21/2002	11	2.5	-5	4.6
HCH, gamma	µg/L	10/2/2001	10/2/2001	1	0.003	0.003	0.003
Heptachlor	µg/L	6/10/2002	8/17/2007	49	0.015	-0.2	0.015
Heptachlor epoxide	µg/L	6/10/2002	8/17/2007	49	0.01	-0.1	0.01
Hexachlorobenzene	µg/L	1/23/2002	8/17/2007	57	0.00075	-2.4	3.2
Hexachlorobutadiene	µg/L	1/23/2002	8/17/2007	57	0.2	-1	0.8

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Hexachlorocyclopentadiene	µg/L	1/23/2002	8/17/2007	55	0.1	-5	3.2
Hexachloroethane	µg/L	1/23/2002	8/17/2007	50	0.2	-2.4	0.9
HpCDD, 1,2,3,4,6,7,8-	pg/L	6/10/2002	8/7/2007	48	0.24	-23	1840
HpCDF, 1,2,3,4,6,7,8-	pg/L	6/10/2002	8/7/2007	44	0.165	-22	2.11
HpCDF, 1,2,3,4,7,8,9-	pg/L	6/10/2002	8/7/2007	44	0.18	-14	2.86
HxCDD, 1,2,3,4,7,8-	pg/L	6/10/2002	8/7/2007	44	0.335	-18	2.9
HxCDD, 1,2,3,6,7,8-	pg/L	6/10/2002	8/7/2007	44	0.325	-15	2.95
HxCDD, 1,2,3,7,8,9-	pg/L	6/10/2002	8/7/2007	44	0.315	-14	3.8
HxCDF, 1,2,3,4,7,8-	pg/L	6/10/2002	8/7/2007	44	0.195	-18	1.05
HxCDF, 1,2,3,6,7,8-	pg/L	6/10/2002	8/7/2007	44	0.17	-15	1.02
HxCDF, 1,2,3,7,8,9-	pg/L	6/10/2002	8/7/2007	44	0.21	-19	1.71
HxCDF, 2,3,4,6,7,8-	pg/L	6/10/2002	8/7/2007	44	0.19	-18	1.27
Hydroxide as CaCO3	mg/L	11/6/2002	11/6/2002	1	10	-20	-20
Indeno(1,2,3-c,d)pyrene	µg/L	1/23/2002	8/17/2007	60	0.0154	-6	0.131
Iron	µg/L	12/1/1982	10/3/2007	196	0.05	0.05	7800
Iron	mg/L	1/10/2002	12/14/2006	65	0.03	0.03	4.4
Isophorone	µg/L	1/23/2002	8/17/2007	56	0.3	-2.4	0.8
Isopropylbenzene	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Isopropyltoluene, p-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Lead	µg/L	6/2/1986	8/17/2007	421	0.002	-10	50
Lead	mg/L	10/21/1998	10/25/2005	54	0.001	0.001	0.99
Linuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
Lithium	µg/L	7/1/1986	7/11/1986	2	10	10	10
Malathion	µg/L	11/7/1999	2/26/2008	669	0.01	-0.05	46
MBAS	mg/L	1/23/2002	1/21/2003	44	0.02	-0.05	0.22
Mercury	µg/L	6/23/1986	11/6/2007	285	0.00093	-1	0.9
Mercury	mg/L	10/21/1998	10/25/2005	52	0.00002	0.00002	0.0082
Mercury	ng/L	10/14/1993	4/18/2007	296	0.23	0.23	2210
Mercury, Methyl	ng/L	3/28/2000	12/14/2006	76	0.014	0.014	1.09
Mercury, Trace Level	µg/L	1/29/2002	12/4/2002	24	0.0008	0.0008	0.013
Mercury, Trace Level	ng/L	6/10/2002	8/7/2007	45	0.1	-0.5	16.4
Merphos	µg/L	1/8/2001	1/8/2001	5	1	1	2.7
Methiocarb	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
Methoxychlor	µg/L	6/10/2002	8/17/2007	49	0.015	-10	10
Methyl-2-pentanone, 4-	µg/L	6/10/2002	11/6/2002	6	5	-10	-10
Methyldibenzothiophene, 4-	µg/L	4/12/2005	4/12/2005	1	0.00619	0.00619	0.00619
Methylene Chloride	µg/L	1/10/2002	12/14/2006	42	0.07	0.07	0.4

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Methylfluorene, 1-	µg/L	4/12/2005	4/12/2005	1	0.0228	0.0228	0.0228
Methylnaphthalene, 1-	µg/L	9/18/2001	6/13/2005	9	0.0101	0.0101	0.0454
Methylnaphthalene, 2-	µg/L	4/21/2003	6/13/2005	8	0.0065	0.0065	0.0563
Methylphenanthrene, 1-	µg/L	4/21/2003	4/12/2005	2	0.0068	0.0068	0.0159
Metolachlor	µg/L	6/10/2002	3/17/2003	11	5	-10	-10
Metribuzin	µg/L	6/10/2002	3/17/2003	11	0.5	-1	-1
Mevinphos	µg/L	9/26/2001	9/26/2001	1	0.056	0.056	0.056
Molinate	µg/L	1/23/2002	3/17/2003	17	0.03	-2	0.28
Molybdenum	µg/L	12/26/2001	10/3/2007	168	0.4	-10	10
Molybdenum	mg/L	10/25/1995	8/16/2007	762	0.00072	0.00072	0.048
Monuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
MTBE	µg/L	1/10/2002	8/17/2007	102	0.06	-3	3.4
Naphthalene	µg/L	1/23/2002	8/17/2007	73	0.00537	-10	0.44
Naphthalenes, C1 -	µg/L	1/21/2003	6/13/2005	11	0.00705	0.00705	0.0993
Naphthalenes, C2 -	µg/L	1/21/2003	6/13/2005	15	0.00596	0.00596	0.1646
Naphthalenes, C3 -	µg/L	1/21/2003	4/12/2005	21	0.00594	0.00594	0.163
Naphthalenes, C4 -	µg/L	1/21/2003	4/12/2005	9	0.0098	0.0098	0.305
Neburon	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
Nickel	µg/L	4/1/1991	8/17/2007	830	0.002	-20	180
Nickel	mg/L	10/21/1998	4/24/2001	52	0.005	0.005	0.008
Nitrate + Nitrite as N	mg/L	7/11/2000	12/4/2002	340	0.1	0.1	18.7
Nitrate + Nitrite as NO3	mg/L	7/27/2000	10/11/2001	301	0.01	0.01	5.24
Nitrate as N	µg/L	4/1/1991	10/8/1991	2	0.1	0.1	0.1
Nitrate as N	mg/L	11/7/1999	12/19/2007	1031	0.009	-1	110
Nitrite as N	mg/L	7/11/2000	12/19/2007	357	0.001	-1	0.34
Nitrobenzene	µg/L	1/23/2002	8/17/2007	50	0.3	-10	0.7
Nitrogen	mg/L	11/7/1999	10/15/2003	167	0.06	-85.6	8.83
Nitrogen, Total Kjeldahl	mg/L	2/25/1999	6/21/2007	1096	0.06	-87.73	32
Nitrogen,Total	mg/L	11/27/2001	12/19/2002	216	0.11	0.11	30.7
Nitrophenol, 2-	µg/L	1/23/2002	8/17/2007	56	0.3	-10	1.1
Nitrophenol, 4-	µg/L	1/23/2002	8/17/2007	55	0.2	-12.1	4
Nitrosodi-n-propylamine, N-	µg/L	1/23/2002	8/17/2007	56	0.3	-5	0.8
N-Nitrodimethylamine	µg/L	6/10/2002	8/7/2007	23	2.5	-5	-5
N-nitrosodimethylamine	µg/L	1/23/2002	8/7/2007	30	0.4	-6	0.6
N-nitrosodiphenylamine	µg/L	1/23/2002	8/17/2007	56	0.4	-2.4	0.7
OCDD	pg/L	6/10/2002	8/7/2007	50	1.05	-51	140
OCDF	pg/L	6/10/2002	8/7/2007	46	0.455	-23	12.7

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Organic Nitrogen	mg/L	7/11/2000	10/18/2000	31	0.19	0.19	0.7
OrthoPhosphate as P	mg/L	7/11/2000	6/21/2007	897	0.007	0.007	11
Oxadiazon	µg/L	9/18/2001	6/14/2005	40	0.0015	0.0015	0.364
Oxamyl	µg/L	1/23/2002	10/21/2002	11	2.6	-10	2.6
Parathion, Ethyl	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
Parathion, Methyl	µg/L	1/8/2001	12/14/2006	33	0.028	0.028	0.16
PCB 005	µg/L	9/18/2001	9/18/2001	1	0.003	0.003	0.003
PCB 018	µg/L	9/19/2001	9/19/2001	1	0.002	0.002	0.002
PCB 101	µg/L	9/19/2001	9/19/2001	1	0.003	0.003	0.003
PCB AROCLOR 1016	µg/L	1/23/2002	8/17/2007	55	0.05	-1	0.15
PCB AROCLOR 1221	µg/L	1/23/2002	8/17/2007	56	0.03	-1	0.25
PCB AROCLOR 1232	µg/L	1/23/2002	8/17/2007	56	0.04	-1	0.3
PCB AROCLOR 1242	µg/L	1/23/2002	8/17/2007	56	0.042	-1	0.2
PCB AROCLOR 1248	µg/L	1/23/2002	8/17/2007	56	0.025	-1	0.052
PCB AROCLOR 1254	µg/L	1/23/2002	8/17/2007	56	0.063	-1	0.3
PCB AROCLOR 1260	µg/L	1/23/2002	8/17/2007	56	0.05	-1	0.15
PeCDD, 1,2,3,7,8-	pg/L	6/10/2002	8/7/2007	44	0.465	-19	2.15
PeCDF, 1,2,3,7,8-	pg/L	6/10/2002	8/7/2007	44	0.295	-12	2.59
PeCDF, 2,3,4,7,8-	pg/L	6/10/2002	8/7/2007	44	0.29	-12	2.38
Pentachlorophenol	µg/L	1/23/2002	8/17/2007	61	0.02	-12.1	3.9
Permethrin, total	µg/L	7/8/2004	2/26/2008	547	0.0015	-0.01	0.036
Permethrin-1	µg/L	1/26/2005	2/28/2006	121	0.0015	-0.01	0.216
Permethrin-2	µg/L	1/26/2005	2/28/2006	121	0.0015	-0.01	0.39
Perylene	µg/L	6/17/2002	4/21/2003	6	0.0334	0.0334	0.12
Phenanthrene	µg/L	1/23/2002	8/17/2007	67	0.0051	-8	0.0429
Phenanthrene/Anthracene, C1 -	µg/L	1/21/2003	6/13/2005	17	0.0063	0.0063	0.139
Phenanthrene/Anthracene, C2 -	µg/L	1/21/2003	6/13/2005	19	0.00601	0.00601	0.178
Phenanthrene/Anthracene, C3 -	µg/L	1/21/2003	6/13/2005	12	0.0061	0.0061	0.186
Phenanthrene/Anthracene, C4 -	µg/L	4/21/2003	6/13/2005	3	0.00643	0.00643	0.0699
Phenol	µg/L	1/23/2002	8/17/2007	56	0.2	-3	0.8
Phorate	µg/L	1/8/2001	1/8/2001	5	0.11	0.11	0.14
Phosphate as P	mg/L	1/29/2002	6/19/2002	10	0.2	0.2	3.9
Phosphorus	µg/L	6/10/2002	11/6/2002	9	5	-10	5750
Phosphorus as P	mg/L	11/7/1999	6/21/2007	1642	0.01	0.01	63
Phosphorus as P	Seconds	9/20/2001	10/4/2001	6	1	1	1
Picloram	µg/L	1/23/2002	12/18/2002	14	0.27	-1	0.27
Prometon	µg/L	11/7/1999	11/7/1999	2	0.09	0.09	0.44

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Prometryn	µg/L	2/20/2002	3/17/2003	12	0.4	-2	0.4
Propachlor	µg/L	6/10/2002	3/17/2003	11	0.25	-0.5	-0.5
Propazine	µg/L	6/18/2002	6/18/2002	1	0.035	0.035	0.035
Propham	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
Propoxur	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
Propylbenzene, n-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Prowl	µg/L	11/7/1999	2/10/2000	6	0.06	0.06	0.52
Pyrene	µg/L	1/23/2002	8/17/2007	65	0.00665	-10	0.126
Secbumeton	µg/L	6/18/2002	6/18/2002	1	0.035	0.035	0.035
Selenium	µg/L	10/3/1986	2/26/2008	12395	0.04	-5	134
Selenium	mg/L	10/21/1998	8/17/2007	81	0.005	-5	3.5
Siduron	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
Silver	µg/L	2/22/1989	8/17/2007	185	0.009	-20	22
Silver	mg/L	10/21/1998	4/24/2001	52	0.0004	0.0004	0.005
Simazine	µg/L	1/23/2002	2/26/2008	608	0.009	-4	7
Strontium	mg/L	10/21/1998	3/28/2001	38	0.21	0.21	0.93
Styrene	µg/L	1/10/2002	8/17/2007	90	0.06	-0.5	0.4
Sulfate	µg/L	6/10/2002	11/6/2002	9	3300	3300	278000
Sulfate	mg/L	9/4/1981	2/16/2006	1472	0.3	0.3	2000
Sulfide as S	µg/L	6/10/2002	11/6/2002	9	500	-1000	-1000
Sulfite (SO3)	µg/L	6/10/2002	11/6/2002	9	1000	-5000	-2000
Swep	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
TCDD, 2,3,7,8-	pg/L	6/10/2002	8/7/2007	44	0.255	-12	1.01
TCDF, 2,3,7,8-	pg/L	6/10/2002	8/7/2007	44	0.325	-8.2	1.53
Tetrachloroethane, 1,1,1,2-	µg/L	6/10/2002	8/17/2007	8	0.25	-1	-0.5
Tetrachloroethane, 1,1,2,2-	µg/L	1/10/2002	8/17/2007	90	0.06	-0.5	0.34
tetrachloroethene	µg/L	1/10/2002	12/14/2006	30	0.06	0.06	0.44
Tetrachloroethene (PCE)	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
Thallium	µg/L	12/26/2001	8/17/2007	170	0.01	-200	1.2
Thallium	mg/L	10/21/1998	10/25/2005	54	0.002	0.002	0.092
Thiobencarb	µg/L	9/26/2001	2/26/2008	492	0.01	-0.1	150
Toluene	µg/L	1/10/2002	8/17/2007	98	0.06	-2	0.9
Total HpCDD	pg/L	4/15/2003	2/17/2004	5	2.78	2.78	14.6
Total PeCDF	pg/L	4/15/2003	4/15/2003	1	1.78	1.78	1.78
Total TCDD	pg/L	4/15/2003	2/17/2004	3	2	2	7.88
Total TCDF	pg/L	4/15/2003	4/15/2003	1	1.58	1.58	1.58
Toxaphene	µg/L	1/23/2002	8/17/2007	55	0.2	-1	0.75

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Toxic Equivalent Quotient as 2,3,7,8-TCDD	pg/L	1/7/2003	3/17/2004	2	0.0241	0.0241	0.0407
TP, 2,4,5-	µg/L	1/23/2002	12/18/2002	14	0.42	-1	0.42
trans-1,2-dichloroethene	µg/L	1/10/2002	8/17/2007	91	0.05	-1	0.43
Tributyltin	µg/L	1/10/2002	1/7/2003	33	0.0014	-0.02	0.05
Tributyltin	ng/L	7/16/2002	7/16/2002	3	2	2	2
Trichlorobenzene, 1,2,3-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Trichlorobenzene, 1,2,4-	µg/L	1/10/2002	8/17/2007	90	0.05	-5	0.4
Trichloroethane, 1,1,1-	µg/L	1/10/2002	8/17/2007	90	0.06	-2	0.49
Trichloroethane, 1,1,2-	µg/L	1/10/2002	8/17/2007	89	0.07	-2	0.3
trichloroethene	µg/L	1/10/2002	12/14/2006	36	0.06	0.06	0.3
Trichloroethylene	µg/L	6/10/2002	8/17/2007	49	0.5	-2	-1
Trichlorofluoromethane	µg/L	1/10/2002	8/17/2007	90	0.05	-5	0.48
Trichlorophenol, 2,4,6-	µg/L	1/23/2002	10/3/2007	82	0.2	-10	10
Trichloropropane, 1,2,3-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Trichlorotrifluoroethane	µg/L	6/10/2002	8/17/2007	79	0.07	-10	0.38
Trifluralin	µg/L	3/2/2001	3/2/2001	1	0.7	0.7	0.7
Trimethylbenzene, 1,2,4-	µg/L	6/10/2002	5/15/2006	7	0.5	-5	-1
Trimethylbenzene, 1,3,5-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
Trimethylnaphthalene, 2,3,5-	µg/L	9/18/2001	4/12/2005	3	0.0149	0.0149	0.035
Vinyl Chloride	µg/L	1/10/2002	8/17/2007	89	0.05	-0.5	0.47
Xylene, m/p-	µg/L	6/10/2002	8/17/2007	49	0.25	-1	-0.5
Xylene, o-	µg/L	6/10/2002	8/17/2007	38	0.25	-1	-0.5
Xylenes, total	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.4
Zinc	µg/L	9/4/1981	8/17/2007	1503	0.01	-1	914
Zinc	mg/L	10/21/1998	4/9/2002	54	0.01	0.01	6
SEDIMENT CHEMISTRY							
Acenaphthene	ng/g dw	9/19/2001	4/12/2005	9	2.07	2.07	8.95
Acenaphthylene	ng/g dw	9/19/2001	4/12/2005	4	2	2	6.54
Aldrin	ng/g dw	4/12/2005	4/12/2005	1	0.326	0.326	0.326
Aluminum	mg/Kg dw	9/18/2001	4/12/2005	23	10150	10150	52811
Anthracene	ng/g dw	9/19/2001	4/12/2005	13	1.36	1.36	32.3
Arsenic	mg/Kg dw	9/18/2001	4/12/2005	23	1.04	1.04	12
Benz(a)anthracene	ng/g dw	9/18/2001	4/12/2005	20	1.11	1.11	149
Benzo(a)pyrene	ng/g dw	9/18/2001	4/12/2005	20	1.24	1.24	278
Benzo(b)fluoranthene	ng/g dw	9/18/2001	4/12/2005	23	1.21	1.21	351
Benzo(e)pyrene	ng/g dw	9/18/2001	4/12/2005	20	1.73	1.73	212
Benzo(g,h,i)perylene	ng/g dw	9/18/2001	4/12/2005	22	2.27	2.27	293

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Benzo(k)fluoranthene	ng/g dw	9/19/2001	4/12/2005	17	1.67	1.67	131
Bifenthrin	µg/Kg	8/10/2004	12/7/2005	37	0.165	-0.33	286.39
Bifenthrin	ng/g dw	4/8/2003	9/19/2005	31	0.401	0.401	436.6
Biphenyl	ng/g dw	9/19/2001	4/12/2005	13	1.4	1.4	11.5
Cadmium	mg/Kg dw	9/18/2001	4/12/2005	23	0.07	0.07	1.24
Chlordane, Alpha-	µg/Kg	8/10/2004	12/7/2005	37	0.3	-0.6	1.24
Chlordane, cis-	ng/g dw	9/19/2001	6/15/2005	17	1.01	1.01	11.6
Chlordane, gamma-	µg/Kg	8/10/2004	12/7/2005	37	0.15	-0.3	-0.3
Chlordane, trans-	ng/g dw	9/19/2001	6/15/2005	22	0.62	0.62	16.5
Chlordene, alpha-	ng/g dw	6/17/2002	6/17/2002	1	2.74	2.74	2.74
Chlordene, gamma-	ng/g dw	6/17/2002	4/22/2003	2	1.02	1.02	2.16
Chlorpyrifos	µg/Kg	8/10/2004	12/7/2005	37	0.22	-0.44	5.69
Chlorpyrifos	ng/g dw	6/17/2002	6/15/2005	16	1.67	1.67	19.31
Chromium	mg/Kg dw	9/18/2001	4/12/2005	23	26.8	26.8	475
Chrysene	ng/g dw	9/18/2001	4/12/2005	22	2.1	2.1	204
Chrysenes, C1 -	ng/g dw	9/18/2001	4/12/2005	22	1.43	1.43	182
Chrysenes, C2 -	ng/g dw	9/18/2001	4/12/2005	22	1.32	1.32	318
Chrysenes, C3 -	ng/g dw	9/18/2001	4/12/2005	20	3.32	3.32	233
Copper	mg/Kg	8/30/2004	8/10/2005	3	8.17	8.17	70.9
Copper	mg/Kg dw	9/18/2001	4/12/2005	23	10.6	10.6	73.4
Cyfluthrin, total	ng/g dw	9/24/2004	11/7/2004	15	0.9	0.9	179.9
Cyhalothrin, lambda, total	ng/g dw	4/8/2003	9/19/2005	22	0.432	0.432	18.2
Cypermethrin, total	ng/g dw	9/24/2004	6/15/2005	16	1.3	1.3	295.8
Dacthal	ng/g dw	9/19/2001	6/18/2002	2	1.12	1.12	5.35
DDD(o,p')	ng/g dw	6/17/2002	4/12/2005	8	1.21	1.21	8.39
DDD(p,p')	µg/Kg	8/10/2004	12/7/2005	37	0.2	-0.4	9.8
DDD(p,p')	ng/g dw	9/19/2001	6/15/2005	18	1.05	1.05	31.7
DDE(o,p')	ng/g dw	6/15/2005	6/15/2005	1	6.21	6.21	6.21
DDE(p,p')	µg/Kg	8/10/2004	12/7/2005	37	0.185	-0.37	74.57
DDE(p,p')	ng/g dw	9/19/2001	6/15/2005	28	1.15	1.15	134
DDMU(p,p')	ng/g dw	4/12/2005	6/15/2005	3	1.88	1.88	5.03
DDT(o,p')	ng/g dw	6/17/2002	6/15/2005	5	1.7	1.7	17.7
DDT(p,p')	µg/Kg	8/10/2004	12/7/2005	37	0.195	-0.39	28.53
DDT(p,p')	ng/g dw	6/17/2002	4/12/2005	30	1.3	1.3	30.68
Deltamethrin	ng/g dw	9/24/2004	11/7/2004	12	1.8	1.8	48.04
Diazinon	ng/g dw	4/8/2003	4/9/2003	4	1.23	1.23	2.26
Dibenz(a,h)anthracene	ng/g dw	9/18/2001	4/12/2005	21	1.83	1.83	86.8

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Dibenzothiophene	ng/g dw	9/18/2001	4/12/2005	14	1.26	1.26	38.3
Dibenzothiophenes, C1 -	ng/g dw	9/18/2001	4/12/2005	18	1.7	1.7	142
Dibenzothiophenes, C2 -	ng/g dw	9/18/2001	4/12/2005	20	1.57	1.57	418
Dibenzothiophenes, C3 -	ng/g dw	9/18/2001	4/12/2005	19	1.87	1.87	694
Dieldrin	µg/Kg	8/10/2004	12/7/2005	37	0.305	-0.61	3.88
Dieldrin	ng/g dw	9/26/2001	4/12/2005	22	0.795	0.795	12.6
Dimethylnaphthalene, 2,6-	ng/g dw	9/18/2001	4/12/2005	19	1.77	1.77	9.51
Dimethylphenanthrene, 3,6-	ng/g dw	4/12/2005	4/12/2005	7	1.55	1.55	9.42
Endosulfan II	ng/g dw	6/15/2005	6/15/2005	1	1.26	1.26	1.26
Endrin	ng/g dw	10/24/2004	6/15/2005	3	1.06	1.06	1.29
Endrin Aldehyde	ng/g dw	9/24/2004	9/24/2004	1	2.54	2.54	2.54
Esfenvalerate/Fenvalerate, total	ng/g dw	4/8/2003	10/24/2004	9	0.985	0.985	11.4
Fluoranthene	ng/g dw	9/18/2001	4/12/2005	22	1.43	1.43	468
Fluoranthene/Pyrenes, C1 -	ng/g dw	9/18/2001	4/12/2005	22	1.58	1.58	303
Fluorene	ng/g dw	9/19/2001	4/12/2005	11	1.66	1.66	10.5
Fluorenes, C1 -	ng/g dw	9/19/2001	4/12/2005	16	2.17	2.17	9.4
Fluorenes, C2 -	ng/g dw	9/18/2001	4/12/2005	12	1.6	1.6	26
Fluorenes, C3 -	ng/g dw	9/26/2001	4/12/2005	20	2.25	2.25	76.9
HCH, alpha	ng/g dw	9/25/2004	9/25/2004	1	2.38	2.38	2.38
Heptachlor	ng/g dw	4/22/2003	4/22/2003	1	0.848	0.848	0.848
Heptachlor epoxide	ng/g dw	9/18/2001	4/12/2005	11	0.708	0.708	3.2
Hexachlorobenzene	ng/g dw	9/19/2001	4/12/2005	8	0.152	0.152	132
Indeno(1,2,3-c,d)pyrene	ng/g dw	9/18/2001	4/12/2005	22	1.91	1.91	377
Lead	mg/Kg dw	9/18/2001	4/12/2005	23	4.24	4.24	130
Mercury	mg/Kg dw	9/18/2001	4/12/2005	17	0.006	0.006	1.171
Methoxychlor	ng/g dw	9/24/2004	10/24/2004	3	1.65	1.65	7.63
Methyldibenzothiophene, 4-	ng/g dw	4/12/2005	4/12/2005	3	1.63	1.63	6.46
Methylfluoranthene, 2-	ng/g dw	4/11/2005	4/12/2005	8	1.57	1.57	8.52
Methylfluorene, 1-	ng/g dw	4/12/2005	4/12/2005	4	1.66	1.66	2.69
Methylnaphthalene, 1-	ng/g dw	9/19/2001	4/12/2005	18	1.5	1.5	8.76
Methylnaphthalene, 2-	ng/g dw	9/19/2001	4/12/2005	18	2.47	2.47	16.7
Methylphenanthrene, 1-	ng/g dw	9/19/2001	4/12/2005	17	1.89	1.89	49.4
Naphthalene	ng/g dw	9/18/2001	4/12/2005	21	0.94	0.94	17.7
Naphthalenes, C1 -	ng/g dw	9/19/2001	4/12/2005	20	2.2	2.2	26.7
Naphthalenes, C2 -	ng/g dw	9/18/2001	4/12/2005	21	3.74	3.74	29.8
Naphthalenes, C3 -	ng/g dw	9/19/2001	4/12/2005	20	3.76	3.76	26.2
Naphthalenes, C4 -	ng/g dw	9/19/2001	4/12/2005	18	1.58	1.58	17

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Nickel	mg/Kg dw	9/18/2001	4/12/2005	23	13.7	13.7	269
Nonachlor, cis-	ng/g dw	6/17/2002	4/12/2005	7	1.18	1.18	5.36
Nonachlor, trans-	ng/g dw	9/19/2001	6/15/2005	19	0.53	0.53	21.7
Oxadiazon	ng/g dw	9/18/2001	4/12/2005	15	1.65	1.65	267
Oxychlordane	ng/g dw	6/17/2002	4/12/2005	3	0.503	0.503	1.54
Parathion, Ethyl	ng/g dw	9/19/2001	9/19/2001	1	6.17	6.17	6.17
PCB 008	ng/g dw	6/17/2002	6/17/2002	1	0.508	0.508	0.508
PCB 018	ng/g dw	6/17/2002	4/12/2005	8	0.15	0.15	0.876
PCB 027	ng/g dw	9/18/2001	9/18/2001	1	0.212	0.212	0.212
PCB 028	ng/g dw	9/19/2001	4/12/2005	20	0.091	0.091	1.97
PCB 031	ng/g dw	9/18/2001	4/12/2005	16	0.163	0.163	1.22
PCB 033	ng/g dw	9/18/2001	4/12/2005	12	0.086	0.086	1.05
PCB 044	ng/g dw	9/19/2001	4/12/2005	17	0.191	0.191	1.76
PCB 049	ng/g dw	6/17/2002	4/12/2005	13	0.091	0.091	0.929
PCB 052	ng/g dw	9/19/2001	4/12/2005	22	0.211	0.211	2.75
PCB 056	ng/g dw	6/17/2002	4/12/2005	9	0.059	0.059	0.428
PCB 060	ng/g dw	6/17/2002	4/22/2003	6	0.066	0.066	0.368
PCB 066	ng/g dw	9/19/2001	4/12/2005	17	0.133	0.133	4.36
PCB 070	ng/g dw	9/19/2001	4/12/2005	16	0.152	0.152	1.65
PCB 074	ng/g dw	6/17/2002	4/12/2005	6	0.168	0.168	0.472
PCB 087	ng/g dw	9/18/2001	4/12/2005	19	0.114	0.114	2.56
PCB 095	ng/g dw	9/19/2001	4/12/2005	21	0.177	0.177	4.62
PCB 097	ng/g dw	6/17/2002	4/12/2005	13	0.062	0.062	1.85
PCB 099	ng/g dw	9/19/2001	4/12/2005	15	0.081	0.081	2.17
PCB 101	ng/g dw	9/18/2001	4/12/2005	23	0.148	0.148	5.58
PCB 105	ng/g dw	9/26/2001	4/12/2005	19	0.139	0.139	1.43
PCB 110	ng/g dw	9/18/2001	4/12/2005	23	0.147	0.147	5.63
PCB 114	ng/g dw	6/17/2002	4/12/2005	5	0.095	0.095	0.673
PCB 118	ng/g dw	9/18/2001	4/12/2005	23	0.224	0.224	5.43
PCB 128	ng/g dw	6/17/2002	4/12/2005	10	0.09	0.09	1.12
PCB 137	ng/g dw	6/17/2002	4/12/2005	4	0.155	0.155	0.627
PCB 138	ng/g dw	9/18/2001	4/12/2005	23	0.163	0.163	11.9
PCB 141	ng/g dw	6/17/2002	4/12/2005	9	0.075	0.075	2.12
PCB 149	ng/g dw	9/19/2001	4/12/2005	17	0.338	0.338	10.5
PCB 151	ng/g dw	9/26/2001	4/12/2005	14	0.131	0.131	3.96
PCB 153	ng/g dw	9/19/2001	4/12/2005	18	0.181	0.181	12.1
PCB 156	ng/g dw	6/17/2002	4/12/2005	9	0.061	0.061	1.1

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
PCB 157	ng/g dw	6/17/2002	4/12/2005	3	0.094	0.094	0.241
PCB 158	ng/g dw	6/17/2002	4/12/2005	9	0.152	0.152	0.956
PCB 170	ng/g dw	9/26/2001	4/12/2005	12	0.056	0.056	3.71
PCB 174	ng/g dw	9/26/2001	4/12/2005	10	0.301	0.301	4.46
PCB 177	ng/g dw	6/17/2002	4/12/2005	9	0.118	0.118	2.96
PCB 180	ng/g dw	9/19/2001	4/12/2005	18	0.187	0.187	9.2
PCB 183	ng/g dw	6/17/2002	4/12/2005	9	0.096	0.096	2.52
PCB 187	ng/g dw	9/19/2001	4/12/2005	15	0.065	0.065	5.69
PCB 194	ng/g dw	9/26/2001	4/12/2005	10	0.173	0.173	2.36
PCB 195	ng/g dw	6/17/2002	4/12/2005	6	0.135	0.135	0.94
PCB 200	ng/g dw	6/17/2002	4/12/2005	5	0.128	0.128	0.454
PCB 201	ng/g dw	9/26/2001	4/12/2005	11	0.225	0.225	2.53
PCB 203	ng/g dw	9/26/2001	4/12/2005	11	0.233	0.233	1.82
PCB 206	ng/g dw	9/26/2001	4/12/2005	9	0.097	0.097	1.23
PCB 209	ng/g dw	9/26/2001	4/12/2005	3	0.287	0.287	0.353
PCB AROCLOR 1248	ng/g dw	6/17/2002	6/17/2002	2	37	37	38
PCB AROCLOR 1254	ng/g dw	6/17/2002	4/12/2005	13	5	5	73
PCB AROCLOR 1260	ng/g dw	6/17/2002	4/12/2005	12	5	5	86
Permethrin, total	µg/Kg	8/10/2004	12/7/2005	37	0.295	-0.59	23.6
Permethrin, total	ng/g dw	4/8/2003	4/11/2005	5	2.27	2.27	50.6
Permethrin-1	µg/Kg	8/10/2004	12/7/2005	37	0.14	-0.28	14.31
Permethrin-1	ng/g dw	9/24/2004	11/7/2004	20	0.28	0.28	231.5
Permethrin-2	µg/Kg	8/10/2004	12/7/2005	37	0.155	-0.31	10.26
Permethrin-2	ng/g dw	9/24/2004	11/7/2004	20	0.31	0.31	106.5
Perylene	ng/g dw	9/18/2001	4/12/2005	20	2.94	2.94	56
Phenanthrene	ng/g dw	9/18/2001	4/12/2005	22	1.31	1.31	206
Phenanthrene/Anthracene, C1 -	ng/g dw	9/18/2001	4/12/2005	23	1.68	1.68	142
Phenanthrene/Anthracene, C2 -	ng/g dw	9/18/2001	4/12/2005	23	1.7	1.7	379
Phenanthrene/Anthracene, C3 -	ng/g dw	9/18/2001	4/12/2005	22	1.27	1.27	521
Phenanthrene/Anthracene, C4 -	ng/g dw	9/18/2001	4/12/2005	20	1.6	1.6	388
Pyrene	ng/g dw	9/18/2001	4/12/2005	23	1.27	1.27	395
Selenium	mg/Kg	8/30/2004	8/10/2005	3	0.05	-0.1	0.24
Selenium	mg/Kg dw	4/11/2005	4/12/2005	10	0.06	0.06	0.59
Silver	mg/Kg dw	9/18/2001	4/12/2005	23	0.0963	0.0963	0.499
Tedion	ng/g dw	9/19/2001	4/12/2005	6	1.52	1.52	44.4
Toxaphene	ng/g dw	6/15/2005	6/15/2005	1	678	678	678
Trimethylnaphthalene, 2,3,5-	ng/g dw	6/17/2002	4/12/2005	6	1.8	1.8	4.34

Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
Zinc	mg/Kg dw	9/18/2001	4/12/2005	23	5.78	5.78	320
SEDIMENT (INTERSTITIAL WATER) CHEMISTRY							
Chlorpyrifos	µg/L	5/29/2002	6/15/2005	2	0.0561	0.0561	0.122
Diazinon	µg/L	5/29/2002	9/19/2002	2	0.0365	0.0365	0.037
Water Chemistry Date Ranges and Counts		**9/4/1981**	**2/26/2008**	**67823**			
Sediment Chemistry Date Ranges and Counts		**9/18/2001**	**12/7/2005**	**2471**			
Sediment (interstitial water) Chemistry Date Ranges and Counts		**5/29/2002**	**6/15/2005**	**4**			
OVERALL DATE RANGES AND COUNTS		**9/4/1981**	**2/26/2008**	**70298**			

Table 4. POD Contaminant Database – Water Chemistry Data. Summary of sample water chemistry results by data source, projectID and analyte name. Results are tabulated by start and end date of samples, result counts, minimum of results with non detects quantified as one half the MDL, minimum of result and maximum of result.

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
UCD RDC	02TM5001	Carbaryl	µg/L	1/14/2006	4/25/2006	118	0.0035	-0.01	0.026
UCD RDC	02TM5001	Carbofuran	µg/L	1/14/2006	4/25/2006	34	0.005	-0.01	-0.01
UCD RDC	02TM5001	Chlorpyrifos	µg/L	1/14/2006	8/31/2006	185	0.0015	-0.004	0.062
UCD RDC	02TM5001	Diazinon	µg/L	1/14/2006	8/31/2006	185	0.0015	-0.007	0.246
UCD RDC	02TM5001	Diuron	µg/L	1/14/2006	4/25/2006	34	0.001	-0.002	35.9
UCD RDC	02TM5001	Malathion	µg/L	1/14/2006	8/31/2006	101	0.01	-0.03	0.052
UCD RDC	02TM5001	Simazine	µg/L	1/14/2006	3/27/2006	84	0.009	0.009	1.2
UCD RDC	04AG5001	Ammonia as N	mg/L	7/8/2004	11/28/2007	246	0.02	-0.04	12.3
UCD RDC	04AG5001	Bifenthrin	µg/L	7/8/2004	11/28/2007	257	0.00025	-0.005	0.018
UCD RDC	04AG5001	Carbaryl	µg/L	7/8/2004	11/28/2007	185	0.005	-0.01	0.256
UCD RDC	04AG5001	Carbofuran	µg/L	7/8/2004	11/28/2007	185	0.005	-0.01	0.104
UCD RDC	04AG5001	Chloroform	µg/L	7/8/2004	8/12/2004	37	0.027	-0.054	0.09
UCD RDC	04AG5001	Chlorpyrifos	µg/L	7/8/2004	11/28/2007	259	0.0015	-0.003	0.28
UCD RDC	04AG5001	Copper	µg/L	7/8/2004	10/25/2007	218	0.69	0.69	4403
UCD RDC	04AG5001	DDD(o,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.005
UCD RDC	04AG5001	DDD(p,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.01
UCD RDC	04AG5001	DDE(o,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.005
UCD RDC	04AG5001	DDE(p,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.06
UCD RDC	04AG5001	DDT(o,p')	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.011
UCD RDC	04AG5001	DDT(p,p')	µg/L	7/8/2004	11/28/2007	257	0.001	-0.002	0.027
UCD RDC	04AG5001	Diazinon	µg/L	7/8/2004	11/28/2007	259	0.0015	-0.003	1.1
UCD RDC	04AG5001	Dieldrin	µg/L	7/8/2004	11/28/2007	257	0.0005	-0.001	0.01
UCD RDC	04AG5001	Diuron	µg/L	7/8/2004	11/28/2007	185	0.001	-0.002	0.95
UCD RDC	04AG5001	Malathion	µg/L	7/8/2004	11/28/2007	259	0.015	-0.03	46

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
UCD RDC	04AG5001	Permethrin, total	µg/L	7/8/2004	11/28/2007	136	0.0015	-0.01	0.006
UCD RDC	04AG5001	Permethrin-1	µg/L	1/26/2005	2/28/2006	121	0.0015	-0.01	0.216
UCD RDC	04AG5001	Permethrin-2	µg/L	1/26/2005	2/28/2006	121	0.0015	-0.01	0.39
UCD RDC	04AG5001	Selenium	µg/L	7/8/2004	10/25/2007	218	0.05	-0.4	6.88
UCD RDC	04AG5001	Simazine	µg/L	7/8/2004	11/28/2007	223	0.01	-0.02	5.4
UCD RDC	04AG5001	Thiobencarb	µg/L	7/8/2004	11/28/2007	223	0.01	-0.1	150
UCD RDC	04ES5001	Ammonia as N	mg/L	5/18/2006	2/26/2008	59	0.02	-0.07	18
UCD RDC	04ES5001	Bifenthrin	µg/L	9/21/2005	2/26/2008	64	0.003	-0.006	0.037
UCD RDC	04ES5001	Carbaryl	µg/L	5/18/2006	2/26/2008	59	0.025	-0.05	0.25
UCD RDC	04ES5001	Carbofuran	µg/L	5/18/2006	2/26/2008	59	0.025	-0.05	-0.05
UCD RDC	04ES5001	Chlorpyrifos	µg/L	7/31/2004	2/26/2008	93	0.001295	-0.0254	0.094
UCD RDC	04ES5001	Copper	µg/L	5/18/2006	2/26/2008	59	1.9	1.9	84
UCD RDC	04ES5001	DDD(p,p')	µg/L	5/18/2006	2/26/2008	59	0.0015	-0.003	0.003
UCD RDC	04ES5001	DDE(p,p')	µg/L	5/18/2006	2/26/2008	59	0.002	-0.004	-0.004
UCD RDC	04ES5001	DDT(p,p')	µg/L	5/18/2006	2/26/2008	59	0.0035	-0.007	-0.007
UCD RDC	04ES5001	Diazinon	µg/L	7/31/2004	2/26/2008	90	0.001765	-0.0282	0.037
UCD RDC	04ES5001	Dieldrin	µg/L	5/18/2006	2/26/2008	59	0.0025	-0.005	-0.005
UCD RDC	04ES5001	Diuron	µg/L	5/18/2006	2/26/2008	59	0.1	-0.2	37
UCD RDC	04ES5001	Malathion	µg/L	7/31/2004	2/26/2008	61	0.025	-0.05	-0.05
UCD RDC	04ES5001	Permethrin, total	µg/L	7/31/2004	2/26/2008	85	0.0045	-0.009	-0.009
UCD RDC	04ES5001	Selenium	µg/L	5/18/2006	2/26/2008	39	0.35	-0.9	4
UCD RDC	04ES5001	Simazine	µg/L	5/18/2006	2/26/2008	59	0.04	-0.08	2.5
UCD RDC	04ES5001	Thiobencarb	µg/L	5/18/2006	2/26/2008	60	0.03	-0.06	0.1
UCD RDC	04SJ5001	Ammonia as N	mg/L	5/16/2006	1/23/2008	119	0.02	-0.07	10
UCD RDC	04SJ5001	Bifenthrin	µg/L	9/20/2005	1/23/2008	242	0.003	-0.006	0.43
UCD RDC	04SJ5001	Carbaryl	µg/L	5/16/2006	1/23/2008	202	0.025	-0.05	0.61

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
UCD RDC	04SJ5001	Carbofuran	µg/L	5/16/2006	1/23/2008	202	0.025	-0.05	0.09
UCD RDC	04SJ5001	Chlorpyrifos	µg/L	8/24/2004	1/23/2008	341	0.001295	-0.0254	1.7
UCD RDC	04SJ5001	Copper	µg/L	5/16/2006	1/23/2008	127	0.9	0.9	100
UCD RDC	04SJ5001	DDD(p,p')	µg/L	5/16/2006	1/23/2008	200	0.0015	-0.003	-0.003
UCD RDC	04SJ5001	DDE(p,p')	µg/L	5/16/2006	1/23/2008	200	0.002	-0.004	0.48
UCD RDC	04SJ5001	DDT(p,p')	µg/L	5/16/2006	1/23/2008	200	0.0035	-0.007	0.4
UCD RDC	04SJ5001	Diazinon	µg/L	8/24/2004	1/23/2008	330	0.001765	-0.0282	0.45
UCD RDC	04SJ5001	Dieldrin	µg/L	5/16/2006	1/23/2008	202	0.0025	-0.005	0.11
UCD RDC	04SJ5001	Diuron	µg/L	5/16/2006	1/23/2008	202	0.1	-0.2	29
UCD RDC	04SJ5001	Malathion	µg/L	5/16/2006	1/23/2008	201	0.025	-0.05	0.05
UCD RDC	04SJ5001	Permethrin, total	µg/L	8/24/2004	1/23/2008	326	0.0045	-0.009	0.036
UCD RDC	04SJ5001	Selenium	µg/L	5/16/2006	1/23/2008	90	0.11	-1.8	3
UCD RDC	04SJ5001	Simazine	µg/L	5/16/2006	1/23/2008	202	0.04	-0.08	7
UCD RDC	04SJ5001	Thiobencarb	µg/L	5/16/2006	1/23/2008	202	0.03	-0.06	0.57
UCD RDC	06GP5P50	Diazinon	µg/L	2/27/2006	1/24/2008	11	0.0015	-0.003	0.96
UCD RDC	07TM5001	Carbaryl	µg/L	4/19/2007	7/12/2007	12	0.005	-0.01	-0.01
UCD RDC	07TM5001	Carbofuran	µg/L	4/19/2007	7/12/2007	12	0.005	-0.01	0.063
UCD RDC	07TM5001	Chlorpyrifos	µg/L	4/5/2007	7/26/2007	16	0.0015	-0.003	0.016
UCD RDC	07TM5001	Diazinon	µg/L	4/5/2007	7/26/2007	16	0.0015	-0.003	2.5
UCD RDC	07TM5001	Diuron	µg/L	4/19/2007	7/12/2007	12	0.001	-0.002	0.092
UCD RDC	07TM5001	Fipronil	µg/L	4/19/2007	7/12/2007	12	0.025	-0.05	0.402
UCD RDC	07TM5001	Malathion	µg/L	4/5/2007	7/26/2007	16	0.015	-0.03	-0.03
UCD RDC	07TM5001	Simazine	µg/L	4/19/2007	7/12/2007	12	0.01	-0.02	-0.02
BDAT	CALFEDMT	Mercury	ng/L	10/14/1993	10/1/2001	204	0.25	0.25	2210
BDAT	CALFEDMT	Mercury, Methyl	ng/L	3/28/2000	10/1/2001	18	0.014	0.014	0.32
BDAT	CALFEDMT	Suspended Sediment Concentration	mg/L	1/18/1994	10/1/2001	160	5.6	5.6	2556.8

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
BDAT	DHS CDWD	Alkalinity as CaCO3	mg/L	1/3/1984	12/20/1991	141	8	8	330
BDAT	DHS CDWD	Aluminum	µg/L	7/1/1986	12/20/1991	32	0.05	0.05	1500
BDAT	DHS CDWD	Arsenic	µg/L	8/14/1984	10/8/1991	25	0.8	0.8	10.6
BDAT	DHS CDWD	Barium	µg/L	3/19/1986	12/20/1991	18	0.09	0.09	161
BDAT	DHS CDWD	Boron	µg/L	7/24/1984	12/3/1991	23	0.12	0.12	300
BDAT	DHS CDWD	Bromide	mg/L	7/1/1986	7/1/1986	1	0.07	0.07	0.07
BDAT	DHS CDWD	Cadmium	µg/L	4/16/1984	4/1/1991	14	0.01	0.01	25
BDAT	DHS CDWD	Calcium	mg/L	9/4/1981	12/20/1991	167	0.65	0.65	110
BDAT	DHS CDWD	Chloride	mg/L	9/4/1981	12/20/1991	150	0.5	0.5	2310
BDAT	DHS CDWD	Chromium	µg/L	4/1/1991	4/1/1991	4	0.5	0.5	6.3
BDAT	DHS CDWD	Copper	µg/L	3/7/1985	10/8/1991	16	6.7	6.7	300
BDAT	DHS CDWD	Fluoride	mg/L	1/3/1984	12/20/1991	83	0.09	0.09	45
BDAT	DHS CDWD	Hardness as CaCO3	mg/L	9/4/1981	12/20/1991	157	8.5	8.5	780
BDAT	DHS CDWD	Iron	µg/L	12/1/1982	12/20/1991	80	0.05	0.05	3200
BDAT	DHS CDWD	Lead	µg/L	6/2/1986	4/1/1991	6	2	2	50
BDAT	DHS CDWD	Lithium	µg/L	7/1/1986	7/11/1986	2	10	10	10
BDAT	DHS CDWD	Magnesium	mg/L	9/4/1981	12/20/1991	159	0.48	0.48	173
BDAT	DHS CDWD	Manganese	µg/L	1/3/1984	10/8/1991	36	0.06	0.06	360
BDAT	DHS CDWD	Mercury	µg/L	6/23/1986	8/27/1990	3	0.2	0.2	0.9
BDAT	DHS CDWD	Nickel	µg/L	4/1/1991	10/8/1991	5	0.002	0.002	0.007
BDAT	DHS CDWD	Nitrate as N	µg/L	4/1/1991	10/8/1991	2	0.1	0.1	0.1
BDAT	DHS CDWD	pH	none	9/4/1981	12/20/1991	155	5.8	5.8	725
BDAT	DHS CDWD	Potassium	mg/L	1/3/1984	12/20/1991	73	0.3	0.3	46
BDAT	DHS CDWD	Selenium	µg/L	10/3/1986	10/31/1986	2	2	2	2
BDAT	DHS CDWD	Silica	mg/L	2/8/1984	12/3/1991	25	5.6	5.6	26
BDAT	DHS CDWD	Silver	µg/L	2/22/1989	5/16/1989	3	1.3	1.3	22

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
BDAT	DHS CDWD	Sodium	mg/L	9/4/1981	12/20/1991	157	1	1	1260
BDAT	DHS CDWD	SpecificConductivity	µmhos/cm	9/4/1981	12/20/1991	153	21	21	7540
BDAT	DHS CDWD	Sulfate	mg/L	9/4/1981	12/20/1991	134	0.3	0.3	310
BDAT	DHS CDWD	Turbidity	NTU	8/10/1988	12/3/1991	2	3.72	3.72	22
BDAT	DHS CDWD	Zinc	µg/L	9/4/1981	10/21/1991	22	0.01	0.01	914
BDAT	EMPLTCW CDWR	Chlorophyll a	µg/L	6/8/1998	12/28/2006	800	0.16	0.16	243
BDAT	EMPLTCW CDWR	Fluorescence	NR	3/25/2004	12/22/2004	62	0	0	38.1
BDAT	EMPLTCW CDWR	pH	none	3/25/2004	12/22/2004	62	7.13	7.13	9.1
BDAT	EMPLTCW CDWR	Pheophytin a	µg/L	6/8/1998	12/28/2006	800	0.12	0.12	56.2
BDAT	EMPLTCW CDWR	SpecificConductivity	µmhos/cm	3/25/2004	12/22/2004	62	127	127	27270
BDAT	EMPLTCW CDWR	Temperature	°C	3/25/2004	12/22/2004	62	9.1	9.1	25.8
BDAT	EMPLTCW CDWR	Turbidity	NTU	3/25/2004	12/22/2004	62	0.92	0.92	62.5
BDAT	EMPSSWQC CDWR	Chlorophyll a	µg/L	1/21/2005	4/21/2005	12	1.47	1.47	17
BDAT	EMPSSWQC CDWR	Pheophytin a	µg/L	1/21/2005	4/21/2005	12	0.8	0.8	7.24
BDAT	EMPSSWQCC DWR	Chlorophyll a	µg/L	6/11/2001	7/18/2002	38	0.88	0.88	119
BDAT	EMPSSWQCC DWR	Depth	ft	2/6/2002	2/6/2002	1	19.2	19.2	19.2
BDAT	EMPSSWQCC DWR	Fluorescence	NR	2/6/2002	2/6/2002	1	49.42	49.42	49.42
BDAT	EMPSSWQCC DWR	Oxygen, Dissolved	mg/L	8/9/2001	3/4/2002	3	9.1	9.1	10.6
BDAT	EMPSSWQCC DWR	Pheophytin a	µg/L	6/11/2001	7/18/2002	37	0.29	0.29	13.9
BDAT	EMPSSWQCC DWR	Secchi Depth	cm	2/6/2002	2/6/2002	1	20	20	20
BDAT	EMPSSWQCC DWR	SpecificConductivity	µmhos/cm	8/9/2001	3/4/2002	3	806	806	4659
BDAT	EMPSSWQCC DWR	Temperature	°C	8/9/2001	3/4/2002	3	8.88	8.88	26.7
BDAT	EMPSSWQCC DWR	Turbidity	NTU	8/9/2001	3/4/2002	2	12	12	19
BDAT	R2JSM DFG	Secchi Depth	cm	6/28/1995	4/20/2001	298	7	7	1200
BDAT	R2JSM DFG	Temperature	°C	1/1/1991	2/13/2008	6032	0.5	0.5	800
BDAT	R2JSM DFG	Turbidity	NTU	1/1/1991	2/13/2008	4446	0.06	0.06	351

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
BDAT	R2JSM DFG	Velocity	ft/s	1/1/1991	2/13/2008	3365	0	0	1216.88
BDAT	R2JSM DFG	WaterDepth	ft-Datum Unk	1/1/1991	2/13/2008	1776	10	10	180
BDAT	SJRDO CDWR	Ammonia	mg/L	7/27/2000	10/11/2001	300	0.01	0.01	2.4
BDAT	SJRDO CDWR	BOD	mg/L	7/27/2000	10/11/2001	272	0.5	0.5	6.4
BDAT	SJRDO CDWR	BOD10day	mg/L	7/27/2000	10/11/2001	279	0.4	0.4	11.9
BDAT	SJRDO CDWR	BOD5day	mg/L	6/26/2001	10/3/2001	48	0.2	0.2	1.1
BDAT	SJRDO CDWR	Chloride	mg/L	7/27/2000	10/11/2001	293	42	42	210
BDAT	SJRDO CDWR	Chlorophyll a	µg/L	7/27/2000	10/11/2001	329	0.8	0.8	90.3
BDAT	SJRDO CDWR	Nitrate + Nitrite as NO3	mg/L	7/27/2000	10/11/2001	301	0.01	0.01	5.24
BDAT	SJRDO CDWR	Nitrogen, Total Kjeldahl	mg/L	7/27/2000	10/11/2001	296	0.1	0.1	2.9
BDAT	SJRDO CDWR	OrthoPhosphate as P	mg/L	7/27/2000	10/11/2001	299	0.01	0.01	0.9
BDAT	SJRDO CDWR	Oxygen, Dissolved	%	8/14/2000	11/15/2000	47	59.3	59.3	146.6
BDAT	SJRDO CDWR	Pheophytin a	µg/L	7/27/2000	10/11/2001	304	0.864	0.864	121
BDAT	SJRDO CDWR	Phosphorus as P	mg/L	7/27/2000	10/11/2001	301	0.01	0.01	63
BDAT	SJRDO CDWR	Secchi Depth	ft	8/14/2000	10/25/2000	33	0.93	0.93	2.9
BDAT	SJRDO CDWR	Solids	mg/L	7/27/2000	10/11/2001	302	1	1	999
BDAT	SJRDO CDWR	Temperature	°C	8/14/2000	11/15/2000	44	10.84	10.84	25
BDAT	SJRDO CDWR	Total Organic Carbon	mg/L	7/27/2000	10/11/2001	296	2.4	2.4	10.9
BDAT	SJRDO City Of Stockton	1% Light Depth	in.	6/20/2000	10/2/2001	376	9	9	56
BDAT	SJRDO City Of Stockton	Ammonia as N	mg/L	6/20/2000	8/28/2001	198	0.1	0.1	24.6
BDAT	SJRDO City Of Stockton	BOD	mg/L	6/12/2001	8/28/2001	248	0.23	0.23	27
BDAT	SJRDO City Of Stockton	BOD10day	mg/L	6/20/2000	8/28/2001	263	0.61	0.61	63
BDAT	SJRDO City Of Stockton	Chloride	mg/L	6/20/2000	10/31/2000	284	15	15	148
BDAT	SJRDO City Of Stockton	Chlorophyll a	µg/L	6/20/2000	8/28/2001	557	1	1	91
BDAT	SJRDO City Of Stockton	Nitrate + Nitrite as N	mg/L	6/12/2001	8/28/2001	195	0.33	0.33	11
BDAT	SJRDO City Of Stockton	Nitrogen, Total Kjeldahl	mg/L	6/12/2001	8/28/2001	194	0.2	0.2	17

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BDAT	SJRDO City Of Stockton	Oxygen, Dissolved	mg/L	6/20/2000	10/2/2001	362	3.1	3.1	12.4
BDAT	SJRDO City Of Stockton	pH	none	6/12/2001	10/2/2001	266	4.6	4.6	8.93
BDAT	SJRDO City Of Stockton	Pheophytin a	µg/L	6/20/2000	8/28/2001	556	0.85	0.85	75
BDAT	SJRDO City Of Stockton	Phosphorus as P	mg/L	6/12/2001	8/28/2001	257	0.08	0.08	4.6
BDAT	SJRDO City Of Stockton	Solids	mg/L	6/20/2000	9/25/2001	632	1.4	1.4	141
BDAT	SJRDO City Of Stockton	SpecificConductivity	µmhos/cm	6/20/2000	9/25/2001	549	186	186	1315
BDAT	SJRDO City Of Stockton	Temperature	°C	6/20/2000	10/2/2001	377	13.8	13.8	28.4
BDAT	SJRDO City Of Stockton	Total Organic Carbon	mg/L	6/20/2000	8/28/2001	494	2	2	22
BDAT	SJRDO City Of Stockton	Turbidity	NTU	6/20/2000	10/2/2001	637	4.8	4.8	83
BDAT	SJRDO CRWQCV Region 5	BOD10day	mg/L	7/11/2000	10/18/2001	460	0.2	0.2	39.2
BDAT	SJRDO CRWQCV Region 5	BOD15day	mg/L	7/25/2000	9/20/2000	48	0.75	0.75	26.3333
BDAT	SJRDO CRWQCV Region 5	BOD20day	mg/L	7/25/2000	9/20/2000	48	1.0667	1.0667	33.4833
BDAT	SJRDO CRWQCV Region 5	BOD25day	mg/L	7/25/2000	9/20/2000	48	1.2	1.2	41.5333
BDAT	SJRDO CRWQCV Region 5	BOD30day	mg/L	7/25/2000	9/20/2000	48	1.05	1.05	49.7833
BDAT	SJRDO CRWQCV Region 5	BOD5day	mg/L	7/11/2000	10/18/2000	128	0.1	0.1	16.8
BDAT	SJRDO LBNL	Ammonia	mg/L	6/13/2001	10/4/2001	32	0.014	0.014	1.8928
BDAT	SJRDO LBNL	BOD	mg/L	6/27/2001	10/4/2001	44	1.59	1.59	23.16
BDAT	SJRDO LBNL	Chlorophyll a	µg/L	6/27/2001	10/4/2001	40	2	2	176
BDAT	SJRDO LBNL	Dissolved Organic Carbon	mg/L	6/13/2001	10/4/2001	46	2	2	18
BDAT	SJRDO LBNL	Flow	cfs	6/13/2001	10/4/2001	45	1	1	410
BDAT	SJRDO LBNL	OrthoPhosphate as P	mg/L	6/13/2001	10/4/2001	26	0.0151	0.0151	6.7884
BDAT	SJRDO LBNL	pH	none	7/11/2001	10/4/2001	36	7	7	9
BDAT	SJRDO LBNL	Pheophytin a	µg/L	6/27/2001	10/4/2001	40	0.1295	0.1295	28.8004
BDAT	SJRDO LBNL	Phosphorus as P	Seconds	9/20/2001	10/4/2001	6	1	1	1
BDAT	SJRDO LBNL	Solids	mg/L	6/27/2001	10/4/2001	66	4	4	390
BDAT	SJRDO LBNL	Total Organic Carbon	mg/L	6/13/2001	10/4/2001	46	1.61	1.61	13.13

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
BDAT	SJRDO Turlock	Ammonia	mg/L	1/13/1999	1/24/2001	175	0.1	0.1	15
BDAT	SJRDO Turlock	BOD	mg/L	1/13/1999	1/24/2001	154	0.2	0.2	999.99
BDAT	SJRDO Turlock	Discharge	MGD	1/13/1999	8/30/2000	57	3.8	3.8	18.3
BDAT	SJRDO Turlock	Flow	cfs	4/7/1999	8/30/2000	51	645	645	7183
BDAT	SJRDO Turlock	Oxygen, Dissolved	mg/L	1/13/1999	1/24/2001	216	2.6	2.6	11.9
BDAT	SJRDO Turlock	pH	none	1/13/1999	1/24/2001	216	6.1	6.1	8.6
BDAT	SJRDO Turlock	Solids	mg/L	8/11/1999	1/24/2001	311	0	0	111
BDAT	SJRDO Turlock	SpecificConductivity	µmhos/cm	1/13/1999	1/24/2001	216	300	300	1660
BDAT	SJRDO Turlock	Temperature	°F	1/13/1999	1/24/2001	216	7.3	7.3	85
BDAT	SJRDO Turlock	Turbidity	NTU	1/13/1999	1/24/2001	216	1.7	1.7	348
BDAT	SJRDO UOP	Chlorophyll a	g/(m 2 hr)	8/31/2000	11/9/2000	9	0.0001	0.0001	0.0061
BDAT	SJRDO UOP	Chlorophyll a	m/hr	8/31/2000	11/9/2000	4	0.0477	0.0477	0.2211
BDAT	SJRDO UOP	Chlorophyll a	mg/hr	8/31/2000	9/28/2000	3	0.0052	0.0052	0.0108
BDAT	SJRDO UOP	Chlorophyll a	mg/L	6/14/2001	10/25/2001	52	1.7211	1.7211	77.43
BDAT	SJRDO UOP	Chlorophyll a + Pheophytin a	g/(m 2 hr)	8/31/2000	11/9/2000	6	0.0003	0.0003	0.0171
BDAT	SJRDO UOP	Chlorophyll a + Pheophytin a	m/hr	8/31/2000	11/9/2000	6	0.05	0.05	0.3991
BDAT	SJRDO UOP	Chlorophyll a + Pheophytin a	mg/hr	8/31/2000	11/9/2000	8	0.0008	0.0008	0.0436
BDAT	SJRDO UOP	Oxygen, Dissolved	mg/L	6/14/2001	10/25/2001	157	2.3	2.3	10.3
BDAT	SJRDO UOP	Pheophytin a	mg/L	6/14/2001	10/25/2001	52	5.8545	5.8545	40.1034
BDAT	SJRDO UOP	Secchi Depth	cm	6/14/2001	10/25/2001	132	30	30	76
BDAT	SJRDO UOP	Solids	g/(m 2 hr)	7/28/2000	11/9/2000	12	0.498	0.498	51.3124
BDAT	SJRDO UOP	Solids	m/hr	8/16/2000	11/9/2000	5	0.5593	0.5593	2.8913
BDAT	SJRDO UOP	Solids	mg/hr	7/28/2000	11/9/2000	4	0.9567	0.9567	61.568
BDAT	SJRDO UOP	Solids	mg/L	6/14/2001	10/25/2001	52	1.8667	1.8667	51.2
BDAT	SJRDO UOP	SpecificConductivity	µmhos/cm	6/14/2001	10/25/2001	64	653	653	600000
BDAT	SJRDO UOP	Temperature	°C	6/14/2001	10/25/2001	157	15.63	15.63	27.7

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
BDAT	SJRDO UOP	Turbidity	NTU	6/14/2001	10/25/2001	148	13	13	60
BDAT	SJRDO USBR	Flow	cfs	5/15/2001	11/27/2001	575	0	0	214.1
BDAT	SJRDO USBR	SpecificConductivity	µmhos/cm	5/15/2001	11/27/2001	577	513.25	513.25	1421.4
BDAT	SJRDO USBR	Temperature	°C	5/15/2001	11/27/2001	577	11.8	11.8	28.9
BDAT	SJRDO USGS	Alkalinity	mg/L	7/11/2000	11/16/2001	115	31	31	300
BDAT	SJRDO USGS	Ammonia & Organic Nitrogen	mg/L	7/12/2000	11/16/2001	94	0.041	0.041	5.3
BDAT	SJRDO USGS	Ammonia as N	mg/L	7/11/2000	11/16/2001	55	0.043	0.043	2.43
BDAT	SJRDO USGS	Calcium	mg/L	7/11/2000	11/16/2001	118	7.26	7.26	227
BDAT	SJRDO USGS	Chloride	mg/L	7/11/2000	11/16/2001	118	2	2	539
BDAT	SJRDO USGS	Chlorophyll a	µg/L	7/11/2000	11/16/2001	124	0.2	0.2	110
BDAT	SJRDO USGS	Discharge	cfs	7/11/2000	11/16/2001	117	0.9	0.9	2670
BDAT	SJRDO USGS	Dissolved Organic Carbon	mg/L	7/11/2000	10/18/2001	105	2.3	2.3	14
BDAT	SJRDO USGS	Fluoride	mg/L	7/11/2000	11/16/2001	82	0.1	0.1	0.4
BDAT	SJRDO USGS	Hardness as CaCO3	mg/L	7/11/2000	10/18/2000	31	100	100	350
BDAT	SJRDO USGS	Iron	µg/L	7/26/2000	11/16/2001	46	7.7	7.7	150
BDAT	SJRDO USGS	Magnesium	µg/L	6/13/2001	11/15/2001	33	5.8	5.8	115
BDAT	SJRDO USGS	Magnesium	mg/L	7/11/2000	11/16/2001	82	3.09	3.09	87.4
BDAT	SJRDO USGS	Manganese	µg/L	7/11/2000	10/18/2000	31	3	3	70
BDAT	SJRDO USGS	Nitrate + Nitrite as N	mg/L	7/11/2000	11/16/2001	103	0.257	0.257	18.7
BDAT	SJRDO USGS	Nitrate as N	mg/L	7/11/2000	11/16/2001	103	0.009	0.009	4.54
BDAT	SJRDO USGS	Nitrite as N	mg/L	7/11/2000	10/18/2000	31	0.011	0.011	0.204
BDAT	SJRDO USGS	Nitrogen	mg/L	7/11/2000	10/18/2000	31	1.5	1.5	6.6
BDAT	SJRDO USGS	Organic Nitrogen	mg/L	7/11/2000	10/18/2000	31	0.19	0.19	0.7
BDAT	SJRDO USGS	OrthoPhosphate as P	mg/L	7/11/2000	11/16/2001	100	0.042	0.042	2.27
BDAT	SJRDO USGS	Oxygen, Dissolved	mg/L	7/11/2000	11/16/2001	120	4	4	16.4
BDAT	SJRDO USGS	pH	none	7/11/2000	11/16/2001	207	7.1	7.1	9

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BDAT	SJRDO USGS	Pheophytin a	µg/L	6/12/2001	11/16/2001	94	0.7	0.7	57
BDAT	SJRDO USGS	Phosphorus as P	mg/L	7/11/2000	11/16/2001	103	0.053	0.053	2.58
BDAT	SJRDO USGS	Potassium	mg/L	7/11/2000	11/16/2001	118	1	1	18.7
BDAT	SJRDO USGS	Secchi Depth	ft	7/12/2000	10/17/2000	26	0.4	0.4	1.6
BDAT	SJRDO USGS	Silica	mg/L	7/11/2000	11/16/2001	118	7.9	7.9	46.1
BDAT	SJRDO USGS	Sodium	%	7/12/2000	10/18/2000	11	2	2	5
BDAT	SJRDO USGS	Sodium	Abs. Ratio	7/12/2000	10/4/2000	10	49	49	58
BDAT	SJRDO USGS	Sodium	mg/L	7/11/2000	11/16/2001	97	3.2	3.2	628
BDAT	SJRDO USGS	Solids	mg/L	7/11/2000	11/16/2001	107	12	12	3210
BDAT	SJRDO USGS	SpecificConductivity	µmhos	6/12/2001	11/16/2001	82	93	93	4260
BDAT	SJRDO USGS	SpecificConductivity	µmhos/cm	7/11/2000	11/16/2001	121	73	73	3730
BDAT	SJRDO USGS	Sulfate	mg/L	7/11/2000	11/16/2001	118	2.4	2.4	1330
BDAT	SJRDO USGS	SUVA-Organic Carbon Calculation	Abs./(DOC mg/L)	7/11/2000	10/18/2000	27	0.0204	0.0204	0.0306
BDAT	SJRDO USGS	Temperature	°C	7/11/2000	11/16/2001	122	10	10	29
BDAT	SJRDO USGS	Total Organic Carbon	mg/L	7/11/2000	10/18/2001	94	0.3	0.3	7.9
BDAT	SJRDO USGS	UV Absorbance @254nm	absorbance/cm	7/11/2000	10/18/2001	97	0.052	0.052	0.424
BDAT	SJRDO USGS	UV Absorbance @280 nm	absorbance/cm	7/11/2000	10/18/2001	97	0.04	0.04	0.886
BDAT	SJSM USFWS	Secchi Depth	cm	8/2/2006	7/31/2007	4967	17	17	151
BDAT	SJSM USFWS	SpecificConductivity	µmhos/cm	8/1/2006	7/31/2007	1997	0.576	0.576	51700
BDAT	SJSM USFWS	Temperature	°C	8/1/2006	7/31/2007	7172	1	1	29.2
BDAT	SMFM UCD	Electrical Conductance	µmhos/cm	10/23/1979	12/14/2005	6072	10	10	171520
BDAT	SMFM UCD	Oxygen, Dissolved	mg/L	12/9/2005	12/9/2005	1	9	9	9
BDAT	SMFM UCD	Salinity	ppt	12/9/2005	12/9/2005	1	5.8	5.8	5.8
BDAT	SMFM UCD	Secchi Depth	cm	12/9/2005	12/9/2005	1	50	50	50
BDAT	SMFM UCD	SpecificConductivity	µmhos/cm	12/9/2005	12/9/2005	1	10303	10303	10303

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BDAT	SMFM UCD	Temperature	°C	5/16/1979	12/14/2005	8492	0.5	0.5	29.5
NPDES	BWWTP_CB	BOD	mg/L	1/7/2003	5/8/2007	243	2.5	-6	1050
NPDES	BWWTP_CB	Coliform	MPN/100 mL	1/20/2003	5/8/2007	238	7.44	7.44	160000
NPDES	BWWTP_CB	ElectricalConductivity	µmhos/cm	1/3/2003	5/30/2007	684	239	239	2380
NPDES	BWWTP_CB	Oxygen, Dissolved	mg/L	1/3/2003	5/30/2007	684	1.76	1.76	13.88
NPDES	BWWTP_CB	pH	none	1/3/2003	5/30/2007	684	6.3	6.3	8.65
NPDES	BWWTP_CB	Temperature	°F	1/3/2003	5/30/2007	684	44.2	44.2	82
NPDES	BWWTP_CB	Turbidity	NTU	1/3/2003	5/30/2007	687	0.5	-1	1540
NPDES	DRWMR LPW	Ammonia	mg/L	1/4/2000	10/10/2007	233	0.0001	0.0001	13
NPDES	DRWMR LPW	Coliform	MPN/100 mL	2/15/2000	10/10/2007	355	2	2	1600
NPDES	DRWMR LPW	Oxygen, Dissolved	mg/L	1/4/2000	10/31/2007	4707	0.3	0.3	22.2
NPDES	DRWMR LPW	pH	none	1/4/2000	10/25/2007	1511	6	6	9.4
NPDES	DRWMR LPW	SpecificConductivity	µmhos/cm	2/11/2000	10/25/2007	1461	110	110	900
NPDES	DRWMR LPW	Temperature	°C	1/4/2000	10/25/2007	1514	5	5	31
NPDES	DRWMR LPW	Turbidity	NTU	1/4/2000	10/3/2007	398	0.3	0.3	19.7
NPDES	DRWMR MPW_WQCF	13C-1,2,3,4,6,7,8-HpCDD	pg/L	2/17/2004	2/17/2004	2	1110	1110	1560
NPDES	DRWMR MPW_WQCF	13C-1,2,3,4,6,7,8-HpCDF	pg/L	11/12/2003	2/17/2004	4	1030	1030	1620
NPDES	DRWMR MPW_WQCF	13C-1,2,3,4,7,8,9-HpCDF	pg/L	11/12/2003	2/17/2004	4	1220	1220	1680
NPDES	DRWMR MPW_WQCF	13C-1,2,3,4,7,8-HxCDD	pg/L	11/12/2003	2/17/2004	4	1120	1120	1740
NPDES	DRWMR MPW_WQCF	13C-1,2,3,4,7,8-HxCDF	pg/L	11/12/2003	2/17/2004	4	1100	1100	1570
NPDES	DRWMR MPW_WQCF	13C-1,2,3,6,7,8-HxCDD	pg/L	11/12/2003	2/17/2004	4	1010	1010	1680
NPDES	DRWMR MPW_WQCF	13C-1,2,3,6,7,8-HxCDF	pg/L	11/12/2003	2/17/2004	4	1060	1060	1680
NPDES	DRWMR MPW_WQCF	13C-1,2,3,7,8,9-HxCDF	pg/L	11/12/2003	2/17/2004	4	1200	1200	1620
NPDES	DRWMR MPW_WQCF	13C-1,2,3,7,8-PeCDD	pg/L	11/12/2003	2/17/2004	4	1050	1050	1740
NPDES	DRWMR MPW_WQCF	13C-1,2,3,7,8-PeCDF	pg/L	11/12/2003	2/17/2004	4	1120	1120	1620
NPDES	DRWMR MPW_WQCF	13C-2,3,4,6,7,8-HxCDF	pg/L	11/12/2003	2/17/2004	4	1170	1170	1670

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NPDES	DRWMR MPW_WQCF	13C-2,3,4,7,8-PeCDF	pg/L	11/12/2003	2/17/2004	4	1130	1130	1710
NPDES	DRWMR MPW_WQCF	13C-2,3,7,8-TCDD	pg/L	11/12/2003	2/17/2004	4	1200	1200	1680
NPDES	DRWMR MPW_WQCF	13C-2,3,7,8-TCDF	pg/L	11/12/2003	2/17/2004	4	1220	1220	1690
NPDES	DRWMR MPW_WQCF	13C-OCDD	pg/L	11/12/2003	2/17/2004	4	1610	1610	3530
NPDES	DRWMR MPW_WQCF	13C-OCDF	pg/L	11/12/2003	2/17/2004	4	1840	1840	3350
NPDES	DRWMR MPW_WQCF	37Cl-2,3,7,8-TCDD	pg/L	2/17/2004	2/17/2004	2	583	583	627
NPDES	DRWMR MPW_WQCF	Aluminum	µg/L	4/27/2004	10/3/2007	30	183	183	5200
NPDES	DRWMR MPW_WQCF	Ammonia	mg/L	4/27/2004	12/19/2007	360	0.0077	0.0077	0.682
NPDES	DRWMR MPW_WQCF	Antimony	µg/L	4/18/2007	4/18/2007	2	0.1	0.1	0.1
NPDES	DRWMR MPW_WQCF	Arsenic	µg/L	4/27/2004	10/3/2007	32	0.1	0.1	10
NPDES	DRWMR MPW_WQCF	asbestos	MFL	4/18/2007	4/18/2007	3	0	0	0
NPDES	DRWMR MPW_WQCF	Beryllium	µg/L	4/18/2007	4/18/2007	1	0.1	0.1	0.1
NPDES	DRWMR MPW_WQCF	Bis(2-ethylhexyl)phthalate	µg/L	4/27/2004	10/3/2007	26	0.7	0.7	10
NPDES	DRWMR MPW_WQCF	Bromodichloromethane	µg/L	4/27/2004	10/3/2007	30	0.06	0.06	1
NPDES	DRWMR MPW_WQCF	Bromoform	µg/L	4/27/2004	10/3/2007	30	0.07	0.07	1
NPDES	DRWMR MPW_WQCF	Butyl benzyl phthalate	µg/L	4/18/2007	4/18/2007	2	0.1	0.1	0.1
NPDES	DRWMR MPW_WQCF	Chlorine	mg/L	11/7/2006	12/19/2007	44	0.05	0.05	0.32
NPDES	DRWMR MPW_WQCF	Chloroform	µg/L	4/27/2004	10/3/2007	31	0.04	0.04	1
NPDES	DRWMR MPW_WQCF	Chloromethane	µg/L	4/18/2007	4/18/2007	1	0.1	0.1	0.1
NPDES	DRWMR MPW_WQCF	Chromium	µg/L	4/18/2007	4/18/2007	2	3	3	3.2
NPDES	DRWMR MPW_WQCF	CL2 RESIDUAL	mg/L	3/14/2000	2/17/2004	148	0	0	7.9
NPDES	DRWMR MPW_WQCF	Coliform	MPN/100 mL	4/27/2004	12/19/2007	180	11	11	1600
NPDES	DRWMR MPW_WQCF	Copper	µg/L	4/27/2004	10/3/2007	32	1.3	1.3	50
NPDES	DRWMR MPW_WQCF	Dibromochloromethane	µg/L	4/27/2004	10/3/2007	30	0.02	0.02	1
NPDES	DRWMR MPW_WQCF	ElectricalConductivity	µmhos/cm	4/27/2004	12/19/2007	180	113	113	994
NPDES	DRWMR MPW_WQCF	Flow	cfs, monthly avg	11/5/2005	12/5/2007	26	677.9525	677.9525	23260.32

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	DRWMR MPW_WQCF	hardness	mg/L	4/18/2007	4/18/2007	2	0	0	0
NPDES	DRWMR MPW_WQCF	HpCDD, 1,2,3,4,6,7,8-	pg/L	4/15/2003	2/17/2004	4	6.04	6.04	1840
NPDES	DRWMR MPW_WQCF	Iron	µg/L	4/27/2004	10/3/2007	30	355	355	7800
NPDES	DRWMR MPW_WQCF	Lead	µg/L	4/18/2007	4/18/2007	2	0.5	0.5	0.5
NPDES	DRWMR MPW_WQCF	Manganese	µg/L	4/27/2004	10/3/2007	30	30.4	30.4	420
NPDES	DRWMR MPW_WQCF	Mercury	µg/L	4/27/2004	10/3/2007	30	0.00093	0.00093	0.0194
NPDES	DRWMR MPW_WQCF	Mercury	ng/L	4/18/2007	4/18/2007	2	5	5	5.74
NPDES	DRWMR MPW_WQCF	Molybdenum	µg/L	4/27/2004	10/3/2007	30	0.9	0.9	5
NPDES	DRWMR MPW_WQCF	Nickel	µg/L	4/18/2007	4/18/2007	2	3	3	3
NPDES	DRWMR MPW_WQCF	Nitrate as N	mg/L	4/27/2004	12/19/2007	180	0.0645	0.0645	3.76
NPDES	DRWMR MPW_WQCF	Nitrite as N	mg/L	4/27/2004	12/19/2007	180	0.001	0.001	0.109
NPDES	DRWMR MPW_WQCF	OCDD	pg/L	4/15/2003	2/17/2004	6	19	19	73.3
NPDES	DRWMR MPW_WQCF	OCDF	pg/L	4/15/2003	2/17/2004	2	12.2	12.2	12.7
NPDES	DRWMR MPW_WQCF	Oxygen, Dissolved	mg/L	1/4/2000	12/19/2007	732	5.47	5.47	14.8
NPDES	DRWMR MPW_WQCF	pH	none	1/4/2000	12/19/2007	548	0	0	9.82
NPDES	DRWMR MPW_WQCF	Selenium	µg/L	4/18/2007	4/18/2007	2	1.8	1.8	1.8
NPDES	DRWMR MPW_WQCF	SpecificConductivity	µmhos/cm	4/18/2007	4/18/2007	2	0	0	0
NPDES	DRWMR MPW_WQCF	Temperature	°C	4/27/2004	12/19/2007	359	6.4	6.4	27.8
NPDES	DRWMR MPW_WQCF	Temperature	°F	1/4/2000	3/16/2004	185	22	22	82.8
NPDES	DRWMR MPW_WQCF	Toluene	µg/L	4/18/2007	4/18/2007	1	0.2	0.2	0.2
NPDES	DRWMR MPW_WQCF	Total HpCDD	pg/L	4/15/2003	2/17/2004	5	2.78	2.78	14.6
NPDES	DRWMR MPW_WQCF	Total PeCDF	pg/L	4/15/2003	4/15/2003	1	1.78	1.78	1.78
NPDES	DRWMR MPW_WQCF	Total TCDD	pg/L	4/15/2003	2/17/2004	3	2	2	7.88
NPDES	DRWMR MPW_WQCF	Total TCDF	pg/L	4/15/2003	4/15/2003	1	1.58	1.58	1.58
NPDES	DRWMR MPW_WQCF	Trichlorophenol, 2,4,6-	µg/L	4/27/2004	10/3/2007	26	0.2	0.2	10
NPDES	DRWMR MPW_WQCF	Turbidity	NTU	1/4/2000	12/19/2007	364	7.05	7.05	155.6
NPDES	DRWMR MPW_WQCF	Zinc	µg/L	4/18/2007	4/18/2007	2	4.4	4.4	4.5

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	DRWMR MPW_WQCFP	Aldrin	µg/L	4/26/2006	4/26/2006	1	0.005	0.005	0.005
NPDES	DRWMR MPW_WQCFP	Antimony	µg/L	4/27/2004	4/18/2007	8	0.1	0.1	0.5
NPDES	DRWMR MPW_WQCFP	Arsenic	µg/L	4/27/2004	4/18/2007	8	0.6	0.6	12.7
NPDES	DRWMR MPW_WQCFP	asbestos	MFL	4/26/2006	4/26/2006	3	0	0	0
NPDES	DRWMR MPW_WQCFP	benzidine	µg/L	5/4/2005	5/4/2005	1	3	3	3
NPDES	DRWMR MPW_WQCFP	BHC-beta	µg/L	4/27/2004	4/27/2004	1	0.043	0.043	0.043
NPDES	DRWMR MPW_WQCFP	BHC-delta	µg/L	4/27/2004	4/26/2006	5	0.003	0.003	0.008
NPDES	DRWMR MPW_WQCFP	Bis(2-ethylhexyl)phthalate	µg/L	4/27/2004	4/26/2006	2	2	2	25
NPDES	DRWMR MPW_WQCFP	Bromodichloromethane	µg/L	4/27/2004	4/18/2007	6	0.2	0.2	25.7
NPDES	DRWMR MPW_WQCFP	Bromoform	µg/L	4/26/2006	4/26/2006	2	0.2	0.2	0.4
NPDES	DRWMR MPW_WQCFP	Butyl benzyl phthalate	µg/L	4/18/2007	4/18/2007	1	0.3	0.3	0.3
NPDES	DRWMR MPW_WQCFP	Cadmium	µg/L	4/26/2006	4/26/2006	1	0.09	0.09	0.09
NPDES	DRWMR MPW_WQCFP	Carbon tetrachloride	µg/L	4/26/2006	4/26/2006	1	0.1	0.1	0.1
NPDES	DRWMR MPW_WQCFP	Chloroform	µg/L	4/27/2004	4/18/2007	5	0.07	0.07	43
NPDES	DRWMR MPW_WQCFP	Chloromethane	µg/L	4/26/2006	4/18/2007	2	0.06	0.06	0.2
NPDES	DRWMR MPW_WQCFP	Chromium	µg/L	4/27/2004	4/18/2007	10	0.7	0.7	2.8
NPDES	DRWMR MPW_WQCFP	chromium, hexavalent	µg/L	4/18/2007	4/18/2007	1	11	11	11
NPDES	DRWMR MPW_WQCFP	Copper	µg/L	4/27/2004	4/18/2007	10	1.8	1.8	10
NPDES	DRWMR MPW_WQCFP	Cyanide	µg/L	4/27/2004	4/26/2006	6	2	2	5
NPDES	DRWMR MPW_WQCFP	Dibromochloromethane	µg/L	4/27/2004	4/18/2007	6	0.3	0.3	5.5
NPDES	DRWMR MPW_WQCFP	Dichlorobenzene, 1,4-	µg/L	4/26/2006	4/18/2007	2	0.1	0.1	0.1
NPDES	DRWMR MPW_WQCFP	Di-n-butyl phthalate	µg/L	4/18/2007	4/18/2007	1	0.2	0.2	0.2
NPDES	DRWMR MPW_WQCFP	Endrin	µg/L	4/27/2004	4/27/2004	1	0.005	0.005	0.005
NPDES	DRWMR MPW_WQCFP	Endrin Aldehyde	µg/L	4/27/2004	4/27/2004	1	0.01	0.01	0.01
NPDES	DRWMR MPW_WQCFP	hardness	mg/L	4/27/2004	4/18/2007	10	0	0	179
NPDES	DRWMR MPW_WQCFP	Lead	µg/L	4/27/2004	4/18/2007	10	0.2	0.2	0.7

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	DRWMR MPW_WQCFP	Mercury	ng/L	4/27/2004	4/18/2007	10	4.42	4.42	12.8
NPDES	DRWMR MPW_WQCFP	Naphthalene	µg/L	5/4/2005	5/4/2005	1	0.4	0.4	0.4
NPDES	DRWMR MPW_WQCFP	Nickel	µg/L	4/27/2004	4/18/2007	10	1.5	1.5	3.1
NPDES	DRWMR MPW_WQCFP	pH	none	4/27/2004	4/18/2007	10	0	0	7.72
NPDES	DRWMR MPW_WQCFP	Selenium	µg/L	4/27/2004	4/18/2007	6	0.6	0.6	1.6
NPDES	DRWMR MPW_WQCFP	Silver	µg/L	4/27/2004	4/18/2007	4	0.13	0.13	0.86
NPDES	DRWMR MPW_WQCFP	SpecificConductivity	µmhos/cm	4/27/2004	4/18/2007	10	0	0	1150
NPDES	DRWMR MPW_WQCFP	Toluene	µg/L	4/26/2006	4/18/2007	3	0.07	0.07	0.2
NPDES	DRWMR MPW_WQCFP	Zinc	µg/L	4/27/2004	4/18/2007	10	2.8	2.8	60.2
NPDES	DRWR STKPW_STKRCSD	Alkalinity as CaCO3	mg/L	5/22/2002	12/4/2002	9	80	80	180
NPDES	DRWR STKPW_STKRCSD	Aluminum	µg/L	1/29/2002	12/4/2002	24	60	60	1900
NPDES	DRWR STKPW_STKRCSD	Ammonia as N	mg/L	1/29/2002	12/4/2002	24	0.1	0.1	31
NPDES	DRWR STKPW_STKRCSD	Antimony	µg/L	1/29/2002	12/4/2002	15	0.04	0.04	0.7
NPDES	DRWR STKPW_STKRCSD	Arsenic	µg/L	1/29/2002	12/4/2002	24	1.7	1.7	4.3
NPDES	DRWR STKPW_STKRCSD	Barium	µg/L	1/29/2002	12/4/2002	24	5.3	5.3	73
NPDES	DRWR STKPW_STKRCSD	Bicarbonate as CaCO3	mg/L	5/22/2002	12/4/2002	9	70	70	180
NPDES	DRWR STKPW_STKRCSD	Bis(2-ethylhexyl)phthalate	µg/L	2/20/2002	11/2/2005	7	2	2	8.9
NPDES	DRWR STKPW_STKRCSD	Bromodichloromethane	µg/L	1/29/2002	11/6/2007	20	0.06	0.06	4.7
NPDES	DRWR STKPW_STKRCSD	Bromoform	µg/L	4/17/2002	8/20/2003	2	0.2	0.2	0.3
NPDES	DRWR STKPW_STKRCSD	Cadmium	µg/L	3/20/2002	12/4/2002	2	0.03	0.03	0.04
NPDES	DRWR STKPW_STKRCSD	Calcium	mg/L	5/22/2002	12/4/2002	5	31	31	39
NPDES	DRWR STKPW_STKRCSD	Carbofuran	µg/L	2/20/2002	11/13/2002	2	2.3	2.3	2.31
NPDES	DRWR STKPW_STKRCSD	Carbonate as CaCO3	mg/L	6/19/2002	6/19/2002	1	40	40	40
NPDES	DRWR STKPW_STKRCSD	Chloride	mg/L	1/29/2002	12/4/2002	24	38	38	210
NPDES	DRWR STKPW_STKRCSD	Chloroform	µg/L	1/29/2002	11/6/2007	84	0.06	0.06	6.5
NPDES	DRWR STKPW_STKRCSD	Chloromethane	µg/L	10/16/2002	10/16/2002	1	0.7	0.7	0.7

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	DRWR STKPW_STKRCSD	Chromium	µg/L	1/29/2002	12/4/2002	17	0.3	0.3	3.8
NPDES	DRWR STKPW_STKRCSD	Chromium (VI) LowLevel	µg/L	1/29/2002	12/4/2002	11	0.2	0.2	0.8
NPDES	DRWR STKPW_STKRCSD	Copper	µg/L	1/29/2002	12/4/2002	24	1.5	1.5	6.3
NPDES	DRWR STKPW_STKRCSD	Cyanide	µg/L	1/29/2002	11/13/2002	11	1.1	1.1	5
NPDES	DRWR STKPW_STKRCSD	Dibromochloromethane	µg/L	4/17/2002	11/6/2007	11	0.2	0.2	1.3
NPDES	DRWR STKPW_STKRCSD	Dichlorobenzene, 1,4-	µg/L	1/29/2002	1/29/2002	1	0.5	0.5	0.5
NPDES	DRWR STKPW_STKRCSD	Dichloromethane	µg/L	8/3/2005	8/15/2007	14	0.07	0.07	0.12
NPDES	DRWR STKPW_STKRCSD	Diethyl phthalate	µg/L	5/22/2002	5/22/2002	1	0.8	0.8	0.8
NPDES	DRWR STKPW_STKRCSD	Dissolved Solids	mg/L	1/29/2002	12/4/2002	24	260	260	730
NPDES	DRWR STKPW_STKRCSD	Electrical Conductance	µmhos/cm	1/29/2002	12/4/2002	24	170	170	1400
NPDES	DRWR STKPW_STKRCSD	Flow	cfs	1/29/2002	12/4/2002	12	1150	1150	3142
NPDES	DRWR STKPW_STKRCSD	Flow Effluent (Net Daily Flow)	cfs	1/29/2002	12/4/2002	23	38.342	38.342	1890
NPDES	DRWR STKPW_STKRCSD	Fluoride	mg/L	1/29/2002	12/4/2002	24	0.1	0.1	0.6
NPDES	DRWR STKPW_STKRCSD	hardness	mg/L	1/29/2002	12/4/2002	24	90	90	240
NPDES	DRWR STKPW_STKRCSD	Iron	mg/L	1/29/2002	12/4/2002	17	0.08	0.08	3.2
NPDES	DRWR STKPW_STKRCSD	Lead	µg/L	1/29/2002	12/4/2002	24	0.04	0.04	1.1
NPDES	DRWR STKPW_STKRCSD	Magnesium	mg/L	5/22/2002	12/4/2002	5	11	11	19
NPDES	DRWR STKPW_STKRCSD	Manganese	µg/L	1/29/2002	12/4/2002	24	11	11	240
NPDES	DRWR STKPW_STKRCSD	MBAS	mg/L	1/29/2002	12/4/2002	11	0.09	0.09	0.2
NPDES	DRWR STKPW_STKRCSD	Mercury	µg/L	6/19/2002	11/6/2007	148	0.0011	0.0011	0.011
NPDES	DRWR STKPW_STKRCSD	Mercury, Trace Level	µg/L	1/29/2002	12/4/2002	24	0.0008	0.0008	0.013
NPDES	DRWR STKPW_STKRCSD	Molybdenum	µg/L	5/22/2002	12/4/2002	13	2	2	10
NPDES	DRWR STKPW_STKRCSD	MTBE	µg/L	1/29/2002	12/4/2002	12	0.3	0.3	3.4
NPDES	DRWR STKPW_STKRCSD	Nickel	µg/L	1/29/2002	12/4/2002	24	2.8	2.8	6.4
NPDES	DRWR STKPW_STKRCSD	Nitrate + Nitrite as N	mg/L	10/16/2002	12/4/2002	4	0.1	0.1	2.1
NPDES	DRWR STKPW_STKRCSD	Nitrate as N	mg/L	1/29/2002	12/4/2002	23	0.1	0.1	13
NPDES	DRWR STKPW_STKRCSD	Nitrite as N	mg/L	1/29/2002	8/7/2002	6	0.03	0.03	0.34

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	DRWR STKPW_STKRCSD	pH	none	1/29/2002	12/4/2002	24	6.56	6.56	9.1
NPDES	DRWR STKPW_STKRCSD	Phosphate as P	mg/L	1/29/2002	6/19/2002	10	0.2	0.2	3.9
NPDES	DRWR STKPW_STKRCSD	Phosphorus as P	mg/L	5/22/2002	12/4/2002	14	0.2	0.2	1.6
NPDES	DRWR STKPW_STKRCSD	Potassium	mg/L	5/22/2002	12/4/2002	5	5	5	34
NPDES	DRWR STKPW_STKRCSD	Prometryn	µg/L	2/20/2002	2/20/2002	1	0.4	0.4	0.4
NPDES	DRWR STKPW_STKRCSD	River Flow Ratio (Stockton/Vernalis)	Ratio	2/20/2002	12/4/2002	9	0.126	0.126	0.868
NPDES	DRWR STKPW_STKRCSD	Selenium	µg/L	1/29/2002	12/4/2002	23	0.4	0.4	2
NPDES	DRWR STKPW_STKRCSD	Silver	µg/L	1/29/2002	12/4/2002	10	0.02	0.02	0.4
NPDES	DRWR STKPW_STKRCSD	Sodium	mg/L	5/22/2002	12/4/2002	5	82	82	160
NPDES	DRWR STKPW_STKRCSD	Sulfate	mg/L	1/29/2002	12/4/2002	24	10	10	180
NPDES	DRWR STKPW_STKRCSD	Temperature	°C	1/29/2002	12/4/2002	24	6.5	6.5	26.4
NPDES	DRWR STKPW_STKRCSD	Temperature	°F	1/29/2002	12/4/2002	24	43.7	43.7	79.52
NPDES	DRWR STKPW_STKRCSD	Thallium	µg/L	1/29/2002	12/4/2002	15	0.04	0.04	0.3
NPDES	DRWR STKPW_STKRCSD	Toluene	µg/L	4/17/2002	7/10/2002	4	0.4	0.4	0.9
NPDES	DRWR STKPW_STKRCSD	trichloroethene	µg/L	2/9/2005	11/2/2005	6	0.1	0.1	0.2
NPDES	DRWR STKPW_STKRCSD	Zinc	µg/L	1/29/2002	12/4/2002	24	2	2	13
NPDES	MWQCF_CM	1,1-dichloroethene	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.49
NPDES	MWQCF_CM	1,2-Benzanthracene	µg/L	1/7/2003	1/7/2003	1	0.12	0.12	0.12
NPDES	MWQCF_CM	1,2-diphenylhydrazine	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.9
NPDES	MWQCF_CM	1,3-dichloropropene(total)	µg/L	1/10/2002	1/7/2003	12	0.22	0.22	0.3
NPDES	MWQCF_CM	2,4-D	µg/L	1/23/2002	12/18/2002	6	5.3	5.3	5.3
NPDES	MWQCF_CM	2-chloroethylvinyl ether	µg/L	1/10/2002	12/14/2006	42	0.1	0.1	0.32
NPDES	MWQCF_CM	3,3'-dichlorobenzidine	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.6
NPDES	MWQCF_CM	3,4-Benzofluoranthene	µg/L	1/7/2003	1/7/2003	1	0.11	0.11	0.11
NPDES	MWQCF_CM	4,4'-DDD	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
NPDES	MWQCF_CM	4,4'-DDE	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015

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NPDES	MWQCF_CM	4,4'-DDT	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Acenaphthene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.17
NPDES	MWQCF_CM	Acenaphthylene	µg/L	1/23/2002	3/22/2006	8	0.02	0.02	0.03
NPDES	MWQCF_CM	Acrolein	µg/L	1/10/2002	12/14/2006	42	0.56	0.56	3.3
NPDES	MWQCF_CM	acrylonitrile	µg/L	1/10/2002	12/14/2006	42	0.33	0.33	1.6
NPDES	MWQCF_CM	Alachlor	µg/L	1/23/2002	4/9/2002	4	0.3	0.3	0.3
NPDES	MWQCF_CM	Aluminum	µg/L	1/23/2002	12/14/2006	102	710	710	4200
NPDES	MWQCF_CM	Aluminum	mg/L	10/21/1998	2/9/2005	54	0.03	0.03	4.3
NPDES	MWQCF_CM	Anthracene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.16
NPDES	MWQCF_CM	Antimony	µg/L	12/26/2001	12/14/2006	106	0.01	0.01	0.7
NPDES	MWQCF_CM	Antimony	mg/L	10/21/1998	10/25/2005	52	0.005	0.005	0.2
NPDES	MWQCF_CM	Arsenic	µg/L	12/26/2001	12/14/2006	98	1.3	1.3	6
NPDES	MWQCF_CM	Arsenic	mg/L	10/21/1998	2/23/2006	58	0.0021	0.0021	3.4
NPDES	MWQCF_CM	asbestos	MFL	3/22/2006	3/22/2006	2	0.5	0.5	0.5
NPDES	MWQCF_CM	asbestos	MFL>10um	1/10/2002	3/17/2004	36	0.2	0.2	5.12
NPDES	MWQCF_CM	Atrazine	µg/L	1/23/2002	1/7/2003	6	0.02	0.02	0.8
NPDES	MWQCF_CM	Azinphos methyl	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
NPDES	MWQCF_CM	Barium	µg/L	1/23/2002	12/15/2004	42	26	26	100
NPDES	MWQCF_CM	Barium	mg/L	10/21/1998	4/24/2001	50	0.046	0.046	0.73
NPDES	MWQCF_CM	Bentazon	µg/L	1/23/2002	12/18/2002	6	0.84	0.84	0.84
NPDES	MWQCF_CM	Benz(a)anthracene	µg/L	1/23/2002	3/22/2006	8	0.02	0.02	0.12
NPDES	MWQCF_CM	Benzene	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.3
NPDES	MWQCF_CM	benzidine	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	1
NPDES	MWQCF_CM	Benzo(a)pyrene	µg/L	1/23/2002	3/22/2006	8	0.02	0.02	0.09
NPDES	MWQCF_CM	Benzo(b)fluoranthene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.11
NPDES	MWQCF_CM	Benzo(g,h,i)perylene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.06
NPDES	MWQCF_CM	Benzo(k)fluoranthene	µg/L	1/23/2002	3/22/2006	8	0.04	0.04	0.16

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	Beryllium	µg/L	12/26/2001	12/14/2006	100	0.05	0.05	0.3
NPDES	MWQCF_CM	Beryllium	mg/L	10/21/1998	10/25/2005	54	0.0005	0.0005	0.06
NPDES	MWQCF_CM	BHC-beta	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	BHC-delta	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	BHC-gamma (Lindane)	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Bis(2-chloroethoxy)methane	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.9
NPDES	MWQCF_CM	Bis(2-chloroethyl)ether	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.7
NPDES	MWQCF_CM	Bis(2-chloroisopropyl) ether	µg/L	1/23/2002	3/22/2006	8	0.6	0.6	1
NPDES	MWQCF_CM	Bis(2-ethylhexyl)phthalate	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.8
NPDES	MWQCF_CM	Boron	µg/L	12/26/2001	12/14/2006	68	81	81	1400
NPDES	MWQCF_CM	Boron	mg/L	2/20/2002	10/31/2006	28	0.11	0.11	1
NPDES	MWQCF_CM	Bromodichloromethane	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.46
NPDES	MWQCF_CM	Bromoform	µg/L	1/10/2002	12/14/2006	42	0.07	0.07	0.2
NPDES	MWQCF_CM	Bromomethane	µg/L	1/10/2002	12/14/2006	41	0.05	0.05	0.5
NPDES	MWQCF_CM	Bromophenyl phenyl ether, 4-	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	2
NPDES	MWQCF_CM	Butyl benzyl phthalate	µg/L	1/23/2002	1/7/2003	5	0.4	0.4	0.8
NPDES	MWQCF_CM	Cadmium	µg/L	12/26/2001	12/14/2006	102	0.02	0.02	0.08
NPDES	MWQCF_CM	Cadmium	mg/L	10/21/1998	10/25/2005	54	0.0003	0.0003	0.03
NPDES	MWQCF_CM	Carbofuran	µg/L	1/23/2002	4/9/2002	4	1.3	1.3	1.3
NPDES	MWQCF_CM	Carbon tetrachloride	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.5
NPDES	MWQCF_CM	Chlordane	µg/L	1/23/2002	3/22/2006	7	0.005	0.005	0.1
NPDES	MWQCF_CM	Chlordane, Alpha-	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Chlordane, gamma-	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Chloride	mg/L	1/11/2002	12/18/2002	20	110	110	240
NPDES	MWQCF_CM	Chloro-3-methylphenol, 4-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.93
NPDES	MWQCF_CM	Chlorobenzene	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.3
NPDES	MWQCF_CM	Chlorodibromomethane	µg/L	1/7/2003	1/7/2003	1	0.3	0.3	0.3

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	Chloroethane	µg/L	1/10/2002	12/14/2006	41	0.07	0.07	0.34
NPDES	MWQCF_CM	Chloroform	µg/L	1/10/2002	12/14/2006	42	0.05	0.05	0.6
NPDES	MWQCF_CM	Chloromethane	µg/L	1/10/2002	12/14/2006	41	0.04	0.04	0.46
NPDES	MWQCF_CM	Chloronaphthalene, 2-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.6
NPDES	MWQCF_CM	Chlorophenol, 2-	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	1.2
NPDES	MWQCF_CM	Chlorophenyl phenyl ether, 4-	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	2
NPDES	MWQCF_CM	Chlorpyrifos	µg/L	1/23/2002	12/14/2006	34	0.006	0.006	0.2
NPDES	MWQCF_CM	Chromium	µg/L	12/26/2001	12/14/2006	100	0.4	0.4	9.1
NPDES	MWQCF_CM	Chromium	mg/L	10/21/1998	10/25/2005	54	0.002	0.002	4.6
NPDES	MWQCF_CM	Chromium VI	µg/L	1/10/2002	3/17/2004	26	0.16	0.16	2
NPDES	MWQCF_CM	Chrysene	µg/L	1/23/2002	3/22/2006	8	0.04	0.04	0.14
NPDES	MWQCF_CM	cis-1,2-Dichloroethene	µg/L	1/10/2002	12/14/2006	42	0.05	0.05	0.44
NPDES	MWQCF_CM	Copper	µg/L	10/21/1998	12/14/2006	174	0.003	0.003	11
NPDES	MWQCF_CM	Copper	mg/L	10/21/1998	10/25/2005	28	0.003	0.003	4.5
NPDES	MWQCF_CM	Cyanide	µg/L	1/10/2002	3/22/2006	32	0.6	0.6	3
NPDES	MWQCF_CM	Cyanide	mg/L	4/9/2002	4/9/2002	2	0.0006	0.0006	0.0006
NPDES	MWQCF_CM	Dalapon	µg/L	1/23/2002	12/18/2002	6	1.6	1.6	1.6
NPDES	MWQCF_CM	Demeton - O and - S	µg/L	2/9/2005	12/14/2006	29	0.047	0.047	0.05
NPDES	MWQCF_CM	Di(2-ethylhexyl)adipate	µg/L	1/23/2002	4/9/2002	4	0.51	0.51	0.51
NPDES	MWQCF_CM	Diazinon	µg/L	1/23/2002	12/14/2006	34	0.006	0.006	0.32
NPDES	MWQCF_CM	Dibenzo(a,h)anthracene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.04
NPDES	MWQCF_CM	Dibromo-3-Chloropropane, 1,2-(DBCP)	µg/L	1/23/2002	4/9/2002	4	0.007	0.007	0.007
NPDES	MWQCF_CM	Dibromochloromethane	µg/L	1/10/2002	12/14/2006	42	0.07	0.07	0.4
NPDES	MWQCF_CM	Dichlorobenzene, 1,2-	µg/L	1/10/2002	12/14/2006	40	0.05	0.05	0.5
NPDES	MWQCF_CM	Dichlorobenzene, 1,3-	µg/L	1/10/2002	12/14/2006	40	0.07	0.07	0.3
NPDES	MWQCF_CM	Dichlorobenzene, 1,4-	µg/L	1/10/2002	12/14/2006	40	0.06	0.06	0.3

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	Dichlorobromomethane	µg/L	1/7/2003	1/7/2003	1	0.2	0.2	0.2
NPDES	MWQCF_CM	Dichlorodifluoromethane	µg/L	1/7/2003	12/14/2006	30	0.06	0.06	0.3
NPDES	MWQCF_CM	Dichloroethane, 1,1-	µg/L	1/10/2002	12/14/2006	40	0.05	0.05	0.34
NPDES	MWQCF_CM	Dichloroethane, 1,2-	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.2
NPDES	MWQCF_CM	Dichloroethylene, trans 1,2-	µg/L	1/7/2003	1/7/2003	1	0.43	0.43	0.43
NPDES	MWQCF_CM	Dichlorophenol, 2,4-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.9
NPDES	MWQCF_CM	Dichloropropane, 1,2-	µg/L	1/10/2002	12/14/2006	42	0.05	0.05	0.5
NPDES	MWQCF_CM	Dichloropropene, cis 1,3-	µg/L	1/10/2002	12/14/2006	42	0.06	0.06	0.25
NPDES	MWQCF_CM	Dichloropropene, trans 1,3-	µg/L	1/7/2003	12/14/2006	31	0.05	0.05	0.3
NPDES	MWQCF_CM	Dichlorotrifluoroethane	µg/L	1/7/2003	12/14/2006	31	0.06	0.06	0.4
NPDES	MWQCF_CM	Dieldrin	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
NPDES	MWQCF_CM	Diethyl phthalate	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	0.9
NPDES	MWQCF_CM	Dimethyl phthalate	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	0.7
NPDES	MWQCF_CM	Dimethylphenol, 2,4-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	1.1
NPDES	MWQCF_CM	Di-n-butyl phthalate	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	1
NPDES	MWQCF_CM	Dinitro-2-methylphenol, 4,6-	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	2
NPDES	MWQCF_CM	Dinitrophenol, 2,4-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	2
NPDES	MWQCF_CM	Dinitrotoluene, 2,4-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.9
NPDES	MWQCF_CM	Dinitrotoluene, 2,6-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.6
NPDES	MWQCF_CM	Di-n-octyl phthalate	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	0.9
NPDES	MWQCF_CM	Dinoseb	µg/L	1/23/2002	12/18/2002	6	0.49	0.49	0.49
NPDES	MWQCF_CM	Diquat	µg/L	1/23/2002	1/7/2003	4	0.8	0.8	0.8
NPDES	MWQCF_CM	Dissolved Solids	mg/L	1/10/2002	1/21/2003	28	540	540	1100
NPDES	MWQCF_CM	Disulfoton	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
NPDES	MWQCF_CM	Electrical Conductance	µmhos/cm	1/7/2002	3/13/2003	28	752	752	1704
NPDES	MWQCF_CM	Endosulfan I	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
NPDES	MWQCF_CM	Endosulfan II	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	Endothal	µg/L	1/23/2002	1/7/2003	4	19	19	19
NPDES	MWQCF_CM	Endrin	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
NPDES	MWQCF_CM	Endrin Aldehyde	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Endrin Ketone	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
NPDES	MWQCF_CM	Ethion	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
NPDES	MWQCF_CM	Ethylbenzene	µg/L	1/10/2002	12/14/2006	40	0.06	0.06	0.4
NPDES	MWQCF_CM	Fluoranthene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.03
NPDES	MWQCF_CM	Fluorene	µg/L	1/23/2002	3/22/2006	8	0.02	0.02	0.03
NPDES	MWQCF_CM	Fluoride	mg/L	1/10/2002	1/7/2003	34	0.03	0.03	0.4
NPDES	MWQCF_CM	gamma-BHC (Lindane)	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Glyphosate	µg/L	1/23/2002	4/9/2002	4	4.6	4.6	4.6
NPDES	MWQCF_CM	hardness	mg/L	12/26/2001	12/15/2004	48	130	130	370
NPDES	MWQCF_CM	Hardness as CaCO3	mg/L	12/27/2001	5/8/2002	26	130	130	340
NPDES	MWQCF_CM	Heptachlor	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Heptachlor epoxide	µg/L	3/22/2006	3/22/2006	1	0.01	0.01	0.01
NPDES	MWQCF_CM	Hexachlorobenzene	µg/L	1/23/2002	3/22/2006	7	0.4	0.4	3.2
NPDES	MWQCF_CM	Hexachlorobutadiene	µg/L	1/23/2002	3/22/2006	8	0.2	0.2	0.8
NPDES	MWQCF_CM	Hexachlorocyclopentadiene	µg/L	1/23/2002	3/22/2006	7	0.1	0.1	3.2
NPDES	MWQCF_CM	Hexachloroethane	µg/L	1/23/2002	3/22/2006	8	0.2	0.2	0.9
NPDES	MWQCF_CM	HpCDD, 1,2,3,4,6,7,8-	pg/L	1/7/2003	3/17/2004	2	3.05	3.05	3.89
NPDES	MWQCF_CM	HpCDF, 1,2,3,4,6,7,8-	pg/L	1/7/2003	3/17/2004	2	1.05	1.05	2.11
NPDES	MWQCF_CM	HpCDF, 1,2,3,4,7,8,9-	pg/L	1/7/2003	3/17/2004	2	1.34	1.34	2.86
NPDES	MWQCF_CM	HxCDD, 1,2,3,4,7,8-	pg/L	1/7/2003	3/17/2004	2	2.4	2.4	2.9
NPDES	MWQCF_CM	HxCDD, 1,2,3,6,7,8-	pg/L	1/7/2003	3/17/2004	2	2.72	2.72	2.95
NPDES	MWQCF_CM	HxCDD, 1,2,3,7,8,9-	pg/L	1/7/2003	3/17/2004	2	2.53	2.53	2.84
NPDES	MWQCF_CM	HxCDF, 1,2,3,4,7,8-	pg/L	1/7/2003	3/17/2004	2	0.823	0.823	1.05
NPDES	MWQCF_CM	HxCDF, 1,2,3,6,7,8-	pg/L	1/7/2003	3/17/2004	2	0.829	0.829	1.02

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	HxCDF, 1,2,3,7,8,9-	pg/L	1/7/2003	3/17/2004	2	1.32	1.32	1.71
NPDES	MWQCF_CM	HxCDF, 2,3,4,6,7,8-	pg/L	1/7/2003	3/17/2004	2	0.955	0.955	1.27
NPDES	MWQCF_CM	Indeno(1,2,3-c,d)pyrene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.04
NPDES	MWQCF_CM	Iron	mg/L	1/10/2002	12/14/2006	48	0.03	0.03	4.4
NPDES	MWQCF_CM	Isophorone	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.8
NPDES	MWQCF_CM	Lead	µg/L	12/26/2001	12/14/2006	102	0.12	0.12	2.2
NPDES	MWQCF_CM	Lead	mg/L	10/21/1998	10/25/2005	54	0.001	0.001	0.99
NPDES	MWQCF_CM	Malathion	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.05
NPDES	MWQCF_CM	Manganese	µg/L	1/10/2002	1/21/2003	32	9.3	9.3	270
NPDES	MWQCF_CM	Manganese	mg/L	1/11/2002	12/14/2006	20	0.021	0.021	0.25
NPDES	MWQCF_CM	MBAS	mg/L	1/23/2002	1/21/2003	24	0.02	0.02	0.22
NPDES	MWQCF_CM	Mercury	µg/L	12/27/2001	12/14/2006	98	0.0016	0.0016	0.018
NPDES	MWQCF_CM	Mercury	mg/L	10/21/1998	10/25/2005	52	0.00002	0.00002	0.0082
NPDES	MWQCF_CM	Mercury, Methyl	ng/L	1/19/2005	12/14/2006	56	0.106	0.106	1.09
NPDES	MWQCF_CM	Methoxychlor	µg/L	3/22/2006	3/22/2006	1	0.015	0.015	0.015
NPDES	MWQCF_CM	Methylene Chloride	µg/L	1/10/2002	12/14/2006	42	0.07	0.07	0.4
NPDES	MWQCF_CM	Molinate	µg/L	1/23/2002	1/7/2003	6	0.03	0.03	0.28
NPDES	MWQCF_CM	Molybdenum	µg/L	12/26/2001	12/14/2006	92	1.6	1.6	8.2
NPDES	MWQCF_CM	Molybdenum	mg/L	10/21/1998	4/24/2001	52	0.005	0.005	0.007
NPDES	MWQCF_CM	MTBE	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.3
NPDES	MWQCF_CM	Naphthalene	µg/L	1/23/2002	3/22/2006	8	0.02	0.02	0.05
NPDES	MWQCF_CM	Nickel	µg/L	12/26/2001	12/14/2006	108	3	3	10
NPDES	MWQCF_CM	Nickel	mg/L	10/21/1998	4/24/2001	52	0.005	0.005	0.008
NPDES	MWQCF_CM	Nitrobenzene	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.7
NPDES	MWQCF_CM	Nitrophenol, 2-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	1.1
NPDES	MWQCF_CM	Nitrophenol, 4-	µg/L	1/23/2002	3/22/2006	7	0.2	0.2	4
NPDES	MWQCF_CM	Nitrosodi-n-propylamine, N-	µg/L	1/23/2002	3/22/2006	8	0.3	0.3	0.8

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	N-nitrosodimethylamine	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	0.6
NPDES	MWQCF_CM	N-nitrosodiphenylamine	µg/L	1/23/2002	3/22/2006	8	0.4	0.4	0.7
NPDES	MWQCF_CM	OCDD	pg/L	1/7/2003	3/17/2004	2	17.2	17.2	17.6
NPDES	MWQCF_CM	OCDF	pg/L	1/7/2003	3/17/2004	2	6.17	6.17	6.17
NPDES	MWQCF_CM	Oxamyl	µg/L	1/23/2002	4/9/2002	4	2.6	2.6	2.6
NPDES	MWQCF_CM	Parathion, Ethyl	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
NPDES	MWQCF_CM	Parathion, Methyl	µg/L	2/9/2005	12/14/2006	29	0.028	0.028	0.04
NPDES	MWQCF_CM	PCB AROCLOR 1016	µg/L	1/23/2002	3/22/2006	7	0.05	0.05	0.15
NPDES	MWQCF_CM	PCB AROCLOR 1221	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.25
NPDES	MWQCF_CM	PCB AROCLOR 1232	µg/L	1/23/2002	3/22/2006	8	0.04	0.04	0.3
NPDES	MWQCF_CM	PCB AROCLOR 1242	µg/L	1/23/2002	3/22/2006	8	0.042	0.042	0.2
NPDES	MWQCF_CM	PCB AROCLOR 1248	µg/L	1/23/2002	3/22/2006	8	0.025	0.025	0.052
NPDES	MWQCF_CM	PCB AROCLOR 1254	µg/L	1/23/2002	3/22/2006	8	0.063	0.063	0.3
NPDES	MWQCF_CM	PCB AROCLOR 1260	µg/L	1/23/2002	3/22/2006	8	0.05	0.05	0.15
NPDES	MWQCF_CM	PeCDD, 1,2,3,7,8-	pg/L	1/7/2003	3/17/2004	2	1.28	1.28	2.15
NPDES	MWQCF_CM	PeCDF, 1,2,3,7,8-	pg/L	1/7/2003	3/17/2004	2	2.06	2.06	2.59
NPDES	MWQCF_CM	PeCDF, 2,3,4,7,8-	pg/L	1/7/2003	3/17/2004	2	2.04	2.04	2.38
NPDES	MWQCF_CM	Pentachlorophenol	µg/L	1/23/2002	3/22/2006	13	0.02	0.02	3.9
NPDES	MWQCF_CM	Phenanthrene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.03
NPDES	MWQCF_CM	Phenol	µg/L	1/23/2002	3/22/2006	8	0.2	0.2	0.8
NPDES	MWQCF_CM	Picloram	µg/L	1/23/2002	12/18/2002	6	0.27	0.27	0.27
NPDES	MWQCF_CM	Pyrene	µg/L	1/23/2002	3/22/2006	8	0.03	0.03	0.03
NPDES	MWQCF_CM	Selenium	µg/L	11/27/2001	12/14/2006	134	0.3	0.3	6
NPDES	MWQCF_CM	Selenium	mg/L	10/21/1998	3/28/2001	50	0.005	0.005	0.005
NPDES	MWQCF_CM	Silver	µg/L	12/26/2001	12/14/2006	104	0.02	0.02	0.08
NPDES	MWQCF_CM	Silver	mg/L	10/21/1998	4/24/2001	52	0.0004	0.0004	0.005
NPDES	MWQCF_CM	Simazine	µg/L	1/23/2002	1/7/2003	6	0.02	0.02	0.38

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	MWQCF_CM	Strontium	mg/L	10/21/1998	3/28/2001	38	0.21	0.21	0.93
NPDES	MWQCF_CM	Styrene	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.4
NPDES	MWQCF_CM	TCDD, 2,3,7,8-	pg/L	1/7/2003	3/17/2004	2	0.903	0.903	1.01
NPDES	MWQCF_CM	TCDF, 2,3,7,8-	pg/L	1/7/2003	3/17/2004	2	1.28	1.28	1.53
NPDES	MWQCF_CM	Tetrachloroethane, 1,1,2,2-	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.34
NPDES	MWQCF_CM	tetrachloroethene	µg/L	1/10/2002	12/14/2006	30	0.06	0.06	0.44
NPDES	MWQCF_CM	Thallium	µg/L	12/26/2001	12/14/2006	106	0.01	0.01	0.2
NPDES	MWQCF_CM	Thallium	mg/L	10/21/1998	10/25/2005	54	0.002	0.002	0.092
NPDES	MWQCF_CM	Thiobencarb	µg/L	1/23/2002	1/7/2003	6	0.02	0.02	0.45
NPDES	MWQCF_CM	Toluene	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.32
NPDES	MWQCF_CM	Toxaphene	µg/L	1/23/2002	3/22/2006	7	0.2	0.2	0.75
NPDES	MWQCF_CM	Toxic Equivalent Quotient as 2,3,7,8-TCDD	pg/L	1/7/2003	3/17/2004	2	0.0241	0.0241	0.0407
NPDES	MWQCF_CM	TP, 2,4,5-	µg/L	1/23/2002	12/18/2002	6	0.42	0.42	0.42
NPDES	MWQCF_CM	trans-1,2-dichloroethene	µg/L	1/10/2002	12/14/2006	42	0.05	0.05	0.43
NPDES	MWQCF_CM	Tributyltin	µg/L	1/10/2002	1/7/2003	28	0.0014	0.0014	0.05
NPDES	MWQCF_CM	Trichlorobenzene, 1,2,4-	µg/L	1/10/2002	12/14/2006	41	0.05	0.05	0.4
NPDES	MWQCF_CM	Trichloroethane, 1,1,1-	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.49
NPDES	MWQCF_CM	Trichloroethane, 1,1,2-	µg/L	1/10/2002	12/14/2006	40	0.07	0.07	0.3
NPDES	MWQCF_CM	trichloroethene	µg/L	1/10/2002	12/14/2006	30	0.06	0.06	0.3
NPDES	MWQCF_CM	Trichlorofluoromethane	µg/L	1/10/2002	12/14/2006	41	0.05	0.05	0.48
NPDES	MWQCF_CM	Trichlorophenol, 2,4,6-	µg/L	1/23/2002	3/22/2006	8	0.2	0.2	2
NPDES	MWQCF_CM	Trichlorotrifluoroethane	µg/L	1/7/2003	12/14/2006	31	0.07	0.07	0.3
NPDES	MWQCF_CM	Vinyl Chloride	µg/L	1/10/2002	12/14/2006	40	0.05	0.05	0.47
NPDES	MWQCF_CM	Xylenes, total	µg/L	1/10/2002	12/14/2006	41	0.06	0.06	0.4
NPDES	MWQCF_CM	Zinc	µg/L	12/26/2001	12/14/2006	110	4	4	58
NPDES	MWQCF_CM	Zinc	mg/L	10/21/1998	4/9/2002	54	0.01	0.01	6

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	SRWTP NPDES	Ammonia as N	mg/L	1/3/2000	12/26/2007	400	0.02	0.02	1.5
NPDES	SRWTP NPDES	Chlorine (Residual)	mg/L	3/23/2000	12/26/2007	794	0	0	0
NPDES	SRWTP NPDES	ElectricalConductivity	µmhos/cm	1/5/2000	12/26/2007	840	72	72	270
NPDES	SRWTP NPDES	Nitrogen	mg/L	1/5/2000	10/15/2003	87	0.1	0.1	1.9
NPDES	SRWTP NPDES	Oxygen, Dissolved	mg/L	1/5/2000	12/26/2007	848	7.3	7.3	17
NPDES	SRWTP NPDES	pH	none	1/5/2000	12/26/2007	848	6.2	6.2	8.3
NPDES	SRWTP NPDES	Temperature	°F	1/5/2000	12/26/2007	848	42.3	42.3	75
NPDES	SRWTP NPDES	Turbidity	NTU	1/3/2000	12/26/2007	852	2.4	2.4	320
NPDES	SRWTP_P4 NPDES	Bromodichloromethane	µg/L	2/18/2004	2/18/2004	1	1.1	1.1	1.1
NPDES	SRWTP_P4 NPDES	Chloroform	µg/L	2/18/2004	2/18/2004	2	1.1	1.1	1.5
NPDES	TMUDSWMD	Ammonia as N	mg/L	11/7/1999	3/6/2002	59	0.12	0.12	4
NPDES	TMUDSWMD	BOD	mg/L	11/7/1999	3/6/2002	54	2	2	56
NPDES	TMUDSWMD	Bolstar	µg/L	2/17/2002	3/6/2002	6	0.1	0.1	1.4
NPDES	TMUDSWMD	Cadmium	µg/L	11/7/1999	3/6/2002	41	0.1	0.1	0.9
NPDES	TMUDSWMD	Chlorpyrifos	µg/L	2/10/2000	3/2/2001	3	0.051	0.051	0.1
NPDES	TMUDSWMD	Chromium	µg/L	11/7/1999	3/6/2002	71	1	1	9
NPDES	TMUDSWMD	COD	mg/L	11/7/1999	3/6/2002	62	5	5	380
NPDES	TMUDSWMD	Coliform	MPN/100 mL	2/10/2000	3/6/2002	84	400	400	1600000
NPDES	TMUDSWMD	Copper	µg/L	11/7/1999	3/6/2002	101	2	2	35
NPDES	TMUDSWMD	Demeton-s	µg/L	1/8/2001	12/5/2001	7	0.2	0.2	2.4
NPDES	TMUDSWMD	Diazinon	µg/L	11/7/1999	3/6/2002	30	0.045	0.045	1.6
NPDES	TMUDSWMD	Dichlorvos	µg/L	11/29/2001	11/29/2001	1	0.13	0.13	0.13
NPDES	TMUDSWMD	Dissolved Solids	mg/L	11/7/1999	3/6/2002	62	10	10	270
NPDES	TMUDSWMD	Disulfoton	µg/L	11/7/1999	3/2/2001	10	0.06	0.06	1.2
NPDES	TMUDSWMD	EPTC	µg/L	11/7/1999	11/7/1999	3	0.21	0.21	0.39
NPDES	TMUDSWMD	Fensulfothion	µg/L	11/29/2001	11/29/2001	6	0.36	0.36	3.4

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TMUDSWMD	Fenthion	µg/L	1/8/2001	1/8/2001	1	0.33	0.33	0.33
NPDES	TMUDSWMD	Hardness as CaCO3	mg/L	11/7/1999	3/6/2002	62	4.7	4.7	130
NPDES	TMUDSWMD	Lead	µg/L	11/7/1999	3/6/2002	46	1.1	1.1	45
NPDES	TMUDSWMD	Malathion	µg/L	11/7/1999	11/7/1999	2	0.47	0.47	0.56
NPDES	TMUDSWMD	Merphos	µg/L	1/8/2001	1/8/2001	5	1	1	2.7
NPDES	TMUDSWMD	Nickel	µg/L	11/7/1999	3/6/2002	100	1	1	22
NPDES	TMUDSWMD	Nitrate + Nitrite as N	mg/L	10/26/2000	3/6/2002	38	0.13	0.13	1.5
NPDES	TMUDSWMD	Nitrate as N	mg/L	11/7/1999	2/27/2000	21	0.04	0.04	2
NPDES	TMUDSWMD	Nitrogen	mg/L	11/7/1999	3/6/2002	49	0.06	-85.6	8.83
NPDES	TMUDSWMD	Nitrogen, Total Kjeldahl	mg/L	11/7/1999	3/6/2002	82	0.27	-87.73	8
NPDES	TMUDSWMD	OilandGrease	mg/L	11/7/1999	3/6/2002	47	1.1	1.1	33.3
NPDES	TMUDSWMD	Parathion, Methyl	µg/L	1/8/2001	1/8/2001	3	0.14	0.14	0.16
NPDES	TMUDSWMD	pH	SU	11/7/1999	3/6/2002	44	6.9	6.9	8.7
NPDES	TMUDSWMD	Phorate	µg/L	1/8/2001	1/8/2001	5	0.11	0.11	0.14
NPDES	TMUDSWMD	Phosphorus as P	mg/L	11/7/1999	3/6/2002	123	0.05	0.05	1.35
NPDES	TMUDSWMD	Prometon	µg/L	11/7/1999	11/7/1999	2	0.09	0.09	0.44
NPDES	TMUDSWMD	Prowl	µg/L	11/7/1999	2/10/2000	6	0.06	0.06	0.52
NPDES	TMUDSWMD	Solids	mg/L	11/7/1999	3/6/2002	59	5	5	490
NPDES	TMUDSWMD	Streptococcus	MPN/100 mL	2/10/2000	3/6/2002	47	800	800	1600000
NPDES	TMUDSWMD	Total Organic Carbon	mg/L	11/7/1999	3/6/2002	61	2.2	2.2	60
NPDES	TMUDSWMD	Trifluralin	µg/L	3/2/2001	3/2/2001	1	0.7	0.7	0.7
NPDES	TMUDSWMD	Zinc	µg/L	11/7/1999	3/6/2002	100	5	5	500
NPDES	TWWTP_CT	1,2-diphenylhydrazine	µg/L	6/10/2002	8/7/2007	45	0.5	-1.2	-1
NPDES	TWWTP_CT	1,3-dichloropropene(total)	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	2,4-D	µg/L	6/10/2002	10/21/2002	8	5	-10	-10
NPDES	TWWTP_CT	2-chloroethylvinyl ether	µg/L	6/10/2002	8/17/2007	49	0.5	-2	-1
NPDES	TWWTP_CT	3,3'-dichlorobenzidine	µg/L	6/10/2002	8/17/2007	48	2	-5	-4

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NPDES	TWWTP_CT	Acenaphthene	µg/L	6/10/2002	8/17/2007	48	0.5	-2.4	-1
NPDES	TWWTP_CT	Acenaphthylene	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Acetone	µg/L	6/10/2002	11/6/2002	6	5	-10	-10
NPDES	TWWTP_CT	Acrolein	µg/L	6/10/2002	8/17/2007	48	10	-20	-20
NPDES	TWWTP_CT	acrylonitrile	µg/L	6/10/2002	8/17/2007	48	1	-2	-2
NPDES	TWWTP_CT	Alachlor	µg/L	6/10/2002	3/17/2003	11	0.5	-1	-1
NPDES	TWWTP_CT	Aldrin	µg/L	6/10/2002	8/17/2007	48	0.05	-0.1	-0.1
NPDES	TWWTP_CT	Aluminum	µg/L	6/10/2002	8/17/2007	41	50	-100	4440
NPDES	TWWTP_CT	Ammonia	mg/L	12/5/2001	12/26/2007	961	0	-1	15.7
NPDES	TWWTP_CT	Ammonia as N	µg/L	6/10/2002	10/21/2002	8	250	-1000	-500
NPDES	TWWTP_CT	Anthracene	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Antimony	µg/L	6/10/2002	8/17/2007	49	0.5	-10	134
NPDES	TWWTP_CT	Arsenic	µg/L	6/10/2002	8/17/2007	49	0.5	-5	7.3
NPDES	TWWTP_CT	asbestos	µg/L	5/21/2002	5/14/2007	42	0	-0.2	1
NPDES	TWWTP_CT	Atrazine	µg/L	6/10/2002	3/17/2003	11	0.5	-1	-1
NPDES	TWWTP_CT	Barban	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Barium	µg/L	6/10/2002	8/17/2007	41	15	15	450
NPDES	TWWTP_CT	Bentazon	µg/L	6/10/2002	10/21/2002	8	1	-2	-2
NPDES	TWWTP_CT	Benz(a)anthracene	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Benzene	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	benzidine	µg/L	6/10/2002	8/7/2007	45	2.5	-6	-5
NPDES	TWWTP_CT	Benzo(a)pyrene	µg/L	6/10/2002	8/17/2007	48	1	-2.4	-2
NPDES	TWWTP_CT	Benzo(g,h,i)perylene	µg/L	6/10/2002	8/17/2007	48	1	-5	-2
NPDES	TWWTP_CT	Benzo(k)fluoranthene	µg/L	6/10/2002	8/17/2007	48	1	-3	-2
NPDES	TWWTP_CT	Beryllium	µg/L	6/10/2002	8/17/2007	49	0.05	-10	-0.1
NPDES	TWWTP_CT	BHC-alpha	µg/L	6/10/2002	8/17/2007	47	0.05	-0.1	-0.1
NPDES	TWWTP_CT	BHC-beta	µg/L	6/10/2002	8/17/2007	48	0.05	-0.1	-0.1

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	BHC-delta	µg/L	6/10/2002	8/17/2007	48	0.05	-0.2	-0.1
NPDES	TWWTP_CT	BHC-gamma (Lindane)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.2	0.13
NPDES	TWWTP_CT	Bicarbonate	mg/L	11/6/2002	11/6/2002	1	31	31	31
NPDES	TWWTP_CT	Bis(2-chloroethoxy)methane	µg/L	6/10/2002	8/17/2007	48	1	-5	-2
NPDES	TWWTP_CT	Bis(2-chloroethyl)ether	µg/L	6/10/2002	8/17/2007	48	0.5	-2.4	-1
NPDES	TWWTP_CT	Bis(2-chloroisopropyl) ether	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Bis(2-ethylhexyl)phthalate	µg/L	6/10/2002	8/17/2007	32	1	-5	19
NPDES	TWWTP_CT	Boron	µg/L	11/6/2002	8/17/2007	41	25	-100	11000
NPDES	TWWTP_CT	Bromobenzene	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Bromodichloromethane	µg/L	6/10/2002	8/17/2007	51	0.25	-0.5	22.4
NPDES	TWWTP_CT	Bromoform	µg/L	6/10/2002	8/17/2007	57	0.04	-2	0.3
NPDES	TWWTP_CT	Bromomethane	µg/L	6/10/2002	8/17/2007	49	0.5	-2	-1
NPDES	TWWTP_CT	Bromophenyl phenyl ether, 4-	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Butanone, 2-	µg/L	6/10/2002	8/17/2007	8	0.25	-1	-0.5
NPDES	TWWTP_CT	Butyl benzyl phthalate	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Butylbenzene, n-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Butylbenzene, sec-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Butylbenzene, tert-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Cadmium	µg/L	6/10/2002	8/17/2007	49	0.005	-2	0.2
NPDES	TWWTP_CT	Calcium	mg/L	11/6/2002	11/6/2002	1	6.4	6.4	6.4
NPDES	TWWTP_CT	Carbaryl	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Carbofuran	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Carbon tetrachloride	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	0.6
NPDES	TWWTP_CT	Carbonate	mg/L	11/6/2002	11/6/2002	1	10	-20	-20
NPDES	TWWTP_CT	Chlordane	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	Chloride	µg/L	6/10/2002	11/6/2002	8	88	88	207000
NPDES	TWWTP_CT	Chloride	mg/L	6/10/2002	10/21/2002	7	15	15	23000

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	Chloro-3-methylphenol, 4-	µg/L	6/10/2002	8/17/2007	48	2	-5	-4
NPDES	TWWTP_CT	Chlorobenzene	µg/L	6/10/2002	8/17/2007	37	0.5	-2	-1
NPDES	TWWTP_CT	Chloroethane	µg/L	6/10/2002	8/17/2007	49	0.25	-2	-0.5
NPDES	TWWTP_CT	Chloroform	µg/L	6/10/2002	8/17/2007	55	0.04	-2	31.5
NPDES	TWWTP_CT	Chloromethane	µg/L	6/10/2002	8/17/2007	48	0.5	-2	-1
NPDES	TWWTP_CT	Chloronaphthalene, 2-	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Chlorophenol, 2-	µg/L	6/10/2002	8/17/2007	48	1	-2.4	-2
NPDES	TWWTP_CT	Chlorophenyl phenyl ether, 4-	µg/L	6/10/2002	8/17/2007	48	1	-5	2.4
NPDES	TWWTP_CT	Chlorotoluene, 2-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Chlorotoluene, 4-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Chlorpropham	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Chlorpyrifos	µg/L	12/17/2001	10/30/2007	90	0.04	-1	-0.08
NPDES	TWWTP_CT	Chromium	µg/L	6/10/2002	8/17/2007	46	0.5	-20	9.7
NPDES	TWWTP_CT	Chromium VI	µg/L	6/10/2002	10/21/2002	8	0.25	-5	0.8
NPDES	TWWTP_CT	Chrysene	µg/L	6/10/2002	8/17/2007	48	1	-5	-2
NPDES	TWWTP_CT	cis-1,2-Dichloroethene	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Coliform	MPN/100 mL	12/17/2001	10/30/2007	95	23	23	2400
NPDES	TWWTP_CT	Copper	µg/L	6/10/2002	8/17/2007	59	0.0025	-10	17
NPDES	TWWTP_CT	Cyanide	µg/L	6/10/2002	8/17/2007	24	0.0025	-5	-0.005
NPDES	TWWTP_CT	Cyanide	mg/L	6/10/2002	8/7/2007	19	0.0025	-5	-0.005
NPDES	TWWTP_CT	Dalapon	µg/L	6/10/2002	10/21/2002	8	5	-10	10
NPDES	TWWTP_CT	DDD (unsp.)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	-0.1
NPDES	TWWTP_CT	DDE (unsp.)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.1	-0.1
NPDES	TWWTP_CT	DDT (unsp.)	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	0.5
NPDES	TWWTP_CT	Di(2-ethylhexyl)adipate	µg/L	6/10/2002	8/7/2007	43	2.5	-6	-5
NPDES	TWWTP_CT	Diazinon	µg/L	12/17/2001	10/30/2007	93	0.04	-0.5	-0.08
NPDES	TWWTP_CT	Dibenzo(a,h)anthracene	µg/L	6/10/2002	8/17/2007	48	0.2	-2.4	-0.4

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	Dibromo-3-Chloropropane, 1,2- (DBCP)	µg/L	6/10/2002	10/21/2002	8	0.005	-0.01	-0.01
NPDES	TWWTP_CT	Dibromochloromethane	µg/L	6/10/2002	8/17/2007	49	0.21	-0.5	7
NPDES	TWWTP_CT	Dibromomethane	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Dicamba	µg/L	6/10/2002	10/21/2002	8	0.75	-1.5	-1.5
NPDES	TWWTP_CT	Dichlorobenzene, 1,2-	µg/L	6/10/2002	8/17/2007	49	0.25	-2	-0.5
NPDES	TWWTP_CT	Dichlorobenzene, 1,3-	µg/L	6/10/2002	8/17/2007	49	0.5	-2	-1
NPDES	TWWTP_CT	Dichlorobenzene, 1,4-	µg/L	6/10/2002	8/17/2007	49	0.24	-2	0.53
NPDES	TWWTP_CT	Dichlorodifluoromethane	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Dichloroethane, 1,1-	µg/L	6/10/2002	8/17/2007	49	0.25	-2	-0.5
NPDES	TWWTP_CT	Dichloroethane, 1,2-	µg/L	6/10/2002	8/17/2007	49	0.25	-1	-0.5
NPDES	TWWTP_CT	Dichloroethylene, 1,1-	µg/L	6/10/2002	8/17/2007	49	0.25	-2	0.42
NPDES	TWWTP_CT	Dichloromethane	µg/L	6/10/2002	8/17/2007	49	0.3	-2	1.8
NPDES	TWWTP_CT	Dichlorophenol, 2,4-	µg/L	6/10/2002	8/17/2007	48	0.5	-3	-1
NPDES	TWWTP_CT	Dichloropropane, 1,2-	µg/L	6/10/2002	8/17/2007	49	0.25	-1	-0.5
NPDES	TWWTP_CT	Dichloropropane, 1,3-	µg/L	6/10/2002	11/6/2002	6	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Dichloropropane, 2,2-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Dichloropropene, 1,2-	µg/L	6/10/2002	11/6/2002	6	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Dieldrin	µg/L	6/10/2002	8/17/2007	48	0.05	-0.1	-0.1
NPDES	TWWTP_CT	Diethyl phthalate	µg/L	6/10/2002	8/17/2007	48	0.5	-2.4	4.9
NPDES	TWWTP_CT	Dimethoate	µg/L	6/10/2002	3/17/2003	11	5	-10	-10
NPDES	TWWTP_CT	Dimethyl phthalate	µg/L	6/10/2002	8/17/2007	48	1	-2.4	-2
NPDES	TWWTP_CT	Dimethylphenol, 2,4-	µg/L	6/10/2002	8/17/2007	48	1	-2.4	-2
NPDES	TWWTP_CT	Di-n-butyl phthalate	µg/L	6/10/2002	8/17/2007	48	0.5	-10	0.9
NPDES	TWWTP_CT	Dinitro-2-methylphenol, 4,6-	µg/L	6/10/2002	8/17/2007	48	5	-12.1	-10
NPDES	TWWTP_CT	Dinitrophenol, 2,4-	µg/L	6/10/2002	8/17/2007	48	2.5	-12.1	3.9
NPDES	TWWTP_CT	Dinitrotoluene, 2,4-	µg/L	6/10/2002	8/17/2007	48	1	-5	-2

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	Dinitrotoluene, 2,6-	µg/L	6/10/2002	8/17/2007	48	1	-5	-2
NPDES	TWWTP_CT	Di-n-octyl phthalate	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Dinoseb	µg/L	6/10/2002	10/21/2002	8	1	-2	-2
NPDES	TWWTP_CT	Diquat	µg/L	6/10/2002	10/21/2002	7	0.2	-0.4	-0.4
NPDES	TWWTP_CT	Dissolved Solids	µg/L	6/10/2002	11/6/2002	9	325	-10000	290000
NPDES	TWWTP_CT	Diuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	ElectricalConductivity	µmhos/cm	6/10/2002	11/6/2002	9	80	80	1480
NPDES	TWWTP_CT	Endosulfan I	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	-0.1
NPDES	TWWTP_CT	Endosulfan II	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	-0.1
NPDES	TWWTP_CT	Endosulfan sulfate	µg/L	6/10/2002	8/17/2007	48	0.15	-1	-0.3
NPDES	TWWTP_CT	Endothal	µg/L	6/10/2002	10/21/2002	7	22.5	-45	-45
NPDES	TWWTP_CT	Endrin	µg/L	6/10/2002	8/17/2007	48	0.05	-0.2	-0.1
NPDES	TWWTP_CT	Endrin Aldehyde	µg/L	6/10/2002	8/17/2007	48	0.05	-0.5	-0.1
NPDES	TWWTP_CT	Ethylbenzene	µg/L	6/10/2002	8/17/2007	49	0.25	-2	-0.5
NPDES	TWWTP_CT	Ethylene Dibromide	µg/L	6/10/2002	10/21/2002	8	0.01	-0.02	-0.02
NPDES	TWWTP_CT	Fenuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	Fluometuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	Fluoranthene	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Fluorene	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Fluoride	µg/L	10/21/2002	5/15/2006	6	50	-100	190
NPDES	TWWTP_CT	Glyphosate	µg/L	6/10/2002	10/21/2002	7	2.5	-5	-5
NPDES	TWWTP_CT	Hardness as CaCO3	mg/L	6/10/2002	3/30/2005	20	86	86	102000
NPDES	TWWTP_CT	Heptachlor	µg/L	6/10/2002	8/17/2007	48	0.05	-0.2	-0.1
NPDES	TWWTP_CT	Heptachlor epoxide	µg/L	6/10/2002	8/17/2007	48	0.05	-0.1	-0.1
NPDES	TWWTP_CT	Hexachlorobenzene	µg/L	6/10/2002	8/17/2007	48	0.5	-2.4	-1
NPDES	TWWTP_CT	Hexachlorobutadiene	µg/L	6/10/2002	8/17/2007	49	0.5	-1	-1
NPDES	TWWTP_CT	Hexachlorocyclopentadiene	µg/L	6/10/2002	8/17/2007	48	1	-5	-2

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	Hexachloroethane	µg/L	6/10/2002	8/17/2007	42	0.5	-2.4	-1
NPDES	TWWTP_CT	HpCDD, 1,2,3,4,6,7,8-	pg/L	6/10/2002	8/7/2007	42	0.24	-23	-0.48
NPDES	TWWTP_CT	HpCDF, 1,2,3,4,6,7,8-	pg/L	6/10/2002	8/7/2007	42	0.165	-22	-0.33
NPDES	TWWTP_CT	HpCDF, 1,2,3,4,7,8,9-	pg/L	6/10/2002	8/7/2007	42	0.18	-14	-0.36
NPDES	TWWTP_CT	HxCDD, 1,2,3,4,7,8-	pg/L	6/10/2002	8/7/2007	42	0.335	-18	0.85
NPDES	TWWTP_CT	HxCDD, 1,2,3,6,7,8-	pg/L	6/10/2002	8/7/2007	42	0.325	-15	-0.65
NPDES	TWWTP_CT	HxCDD, 1,2,3,7,8,9-	pg/L	6/10/2002	8/7/2007	42	0.315	-14	3.8
NPDES	TWWTP_CT	HxCDF, 1,2,3,4,7,8-	pg/L	6/10/2002	8/7/2007	42	0.195	-18	-0.39
NPDES	TWWTP_CT	HxCDF, 1,2,3,6,7,8-	pg/L	6/10/2002	8/7/2007	42	0.17	-15	-0.34
NPDES	TWWTP_CT	HxCDF, 1,2,3,7,8,9-	pg/L	6/10/2002	8/7/2007	42	0.21	-19	-0.42
NPDES	TWWTP_CT	HxCDF, 2,3,4,6,7,8-	pg/L	6/10/2002	8/7/2007	42	0.19	-18	-0.38
NPDES	TWWTP_CT	Hydroxide as CaCO3	mg/L	11/6/2002	11/6/2002	1	10	-20	-20
NPDES	TWWTP_CT	Indeno(1,2,3-c,d)pyrene	µg/L	6/10/2002	8/17/2007	48	0.25	-6	-0.5
NPDES	TWWTP_CT	Iron	µg/L	6/10/2002	8/17/2007	40	76	76	2800
NPDES	TWWTP_CT	Isophorone	µg/L	6/10/2002	8/17/2007	48	0.5	-2.4	-1
NPDES	TWWTP_CT	Isopropylbenzene	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Isopropyltoluene, p-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Lead	µg/L	6/10/2002	8/17/2007	48	0.25	-10	2
NPDES	TWWTP_CT	Linuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	Magnesium	µg/L	8/17/2004	8/7/2007	7	4.3	4.3	8200
NPDES	TWWTP_CT	Magnesium	mg/L	11/6/2002	8/17/2007	14	2.4	2.4	15000
NPDES	TWWTP_CT	Magnesium	NR	8/8/2005	5/14/2007	4	13.8	13.8	14000
NPDES	TWWTP_CT	Manganese	µg/L	6/10/2002	8/17/2007	41	7.9	-20	262
NPDES	TWWTP_CT	MBAS	mg/L	6/10/2002	11/6/2002	9	0.025	-0.05	-0.05
NPDES	TWWTP_CT	Mercury	µg/L	9/15/2003	5/14/2007	6	0.1	-1	0.56
NPDES	TWWTP_CT	Mercury, Methyl	ng/L	10/11/2004	10/11/2004	2	0.041	0.041	0.36
NPDES	TWWTP_CT	Mercury, Trace Level	ng/L	6/10/2002	8/7/2007	45	0.1	-0.5	16.4

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NPDES	TWWTP_CT	Methiocarb	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Methoxychlor	µg/L	6/10/2002	8/17/2007	48	0.5	-10	10
NPDES	TWWTP_CT	Methyl-2-pentanone, 4-	µg/L	6/10/2002	11/6/2002	6	5	-10	-10
NPDES	TWWTP_CT	Metolachlor	µg/L	6/10/2002	3/17/2003	11	5	-10	-10
NPDES	TWWTP_CT	Metribuzin	µg/L	6/10/2002	3/17/2003	11	0.5	-1	-1
NPDES	TWWTP_CT	Molinate	µg/L	6/10/2002	3/17/2003	11	1	-2	-2
NPDES	TWWTP_CT	Molybdenum	µg/L	11/6/2002	8/17/2007	33	0.4	-10	10
NPDES	TWWTP_CT	Monuron	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	MTBE	µg/L	6/10/2002	8/17/2007	49	0.5	-3	-1
NPDES	TWWTP_CT	Naphthalene	µg/L	6/10/2002	8/17/2007	47	0.44	-10	0.44
NPDES	TWWTP_CT	Neburon	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	Nickel	µg/L	6/10/2002	8/17/2007	47	0.5	-20	6.8
NPDES	TWWTP_CT	Nitrate as N	mg/L	6/10/2002	11/6/2002	9	0.5	-1	7
NPDES	TWWTP_CT	Nitrite as N	mg/L	6/10/2002	11/6/2002	9	0.2	-1	-0.4
NPDES	TWWTP_CT	Nitrobenzene	µg/L	6/10/2002	8/17/2007	42	1	-10	-2
NPDES	TWWTP_CT	Nitrophenol, 2-	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Nitrophenol, 4-	µg/L	6/10/2002	8/17/2007	48	5	-12.1	-10
NPDES	TWWTP_CT	Nitrosodi-n-propylamine, N-	µg/L	6/10/2002	8/17/2007	48	1	-5	-2
NPDES	TWWTP_CT	N-Nitrodimethylamine	µg/L	6/10/2002	8/7/2007	23	2.5	-5	-5
NPDES	TWWTP_CT	N-nitrosodimethylamine	µg/L	6/10/2002	8/7/2007	22	2.5	-6	-5
NPDES	TWWTP_CT	N-nitrosodiphenylamine	µg/L	6/10/2002	8/17/2007	48	0.5	-2.4	-1
NPDES	TWWTP_CT	OCDD	pg/L	6/10/2002	8/7/2007	42	1.05	-51	140
NPDES	TWWTP_CT	OCDF	pg/L	6/10/2002	8/7/2007	42	0.455	-23	-0.91
NPDES	TWWTP_CT	Oxamyl	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Oxygen, Dissolved	mg/L	1/12/2000	12/26/2007	1239	1.9	1.9	13.8
NPDES	TWWTP_CT	PCB AROCLOR 1016	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	PCB AROCLOR 1221	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	PCB AROCLOR 1232	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	PCB AROCLOR 1242	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	PCB AROCLOR 1248	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	PCB AROCLOR 1254	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	PCB AROCLOR 1260	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	PeCDD, 1,2,3,7,8-	pg/L	6/10/2002	8/7/2007	42	0.465	-19	-0.93
NPDES	TWWTP_CT	PeCDF, 1,2,3,7,8-	pg/L	6/10/2002	8/7/2007	42	0.295	-12	-0.59
NPDES	TWWTP_CT	PeCDF, 2,3,4,7,8-	pg/L	6/10/2002	8/7/2007	42	0.29	-12	-0.58
NPDES	TWWTP_CT	Pentachlorophenol	µg/L	6/10/2002	8/17/2007	48	0.5	-12.1	-1
NPDES	TWWTP_CT	pH	none	1/12/2000	12/26/2007	1248	6.1	6.1	8.9
NPDES	TWWTP_CT	Phenanthrene	µg/L	6/10/2002	8/17/2007	48	1	-8	-2
NPDES	TWWTP_CT	Phenol	µg/L	6/10/2002	8/17/2007	48	0.5	-3	-1
NPDES	TWWTP_CT	Phosphorus	µg/L	6/10/2002	11/6/2002	9	5	-10	5750
NPDES	TWWTP_CT	Picloram	µg/L	6/10/2002	10/21/2002	8	0.5	-1	-1
NPDES	TWWTP_CT	Potassium	mg/L	11/6/2002	11/6/2002	1	1.7	1.7	1.7
NPDES	TWWTP_CT	Prometryn	µg/L	6/10/2002	3/17/2003	11	1	-2	-2
NPDES	TWWTP_CT	Propachlor	µg/L	6/10/2002	3/17/2003	11	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Propham	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Propoxur	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Propylbenzene, n-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Pyrene	µg/L	6/10/2002	8/17/2007	48	1	-10	-2
NPDES	TWWTP_CT	Selenium	µg/L	6/10/2002	8/7/2007	17	0.5	-5	1
NPDES	TWWTP_CT	Selenium	mg/L	6/10/2002	8/17/2007	31	0.5	-5	3.5
NPDES	TWWTP_CT	Siduron	µg/L	6/10/2002	10/21/2002	7	5	-10	-10
NPDES	TWWTP_CT	Silica	mg/L	11/6/2002	11/6/2002	1	24	24	24
NPDES	TWWTP_CT	Silver	µg/L	6/10/2002	8/17/2007	49	0.5	-20	5
NPDES	TWWTP_CT	Simazine	µg/L	6/10/2002	3/17/2003	11	0.5	-4	-1

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	Sodium	mg/L	11/6/2002	11/6/2002	1	4.8	4.8	4.8
NPDES	TWWTP_CT	SpecificConductivity	µmhos/cm	12/5/2001	12/26/2007	961	3.1	3.1	1860
NPDES	TWWTP_CT	Styrene	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Sulfate	µg/L	6/10/2002	11/6/2002	9	3300	3300	278000
NPDES	TWWTP_CT	Sulfide as S	µg/L	6/10/2002	11/6/2002	9	500	-1000	-1000
NPDES	TWWTP_CT	Sulfite (SO3)	µg/L	6/10/2002	11/6/2002	9	1000	-5000	-2000
NPDES	TWWTP_CT	Swep	µg/L	6/10/2002	10/21/2002	7	2	-4	-4
NPDES	TWWTP_CT	TCDD, 2,3,7,8-	pg/L	6/10/2002	8/7/2007	42	0.255	-12	-0.51
NPDES	TWWTP_CT	TCDF, 2,3,7,8-	pg/L	6/10/2002	8/7/2007	42	0.325	-8.2	-0.65
NPDES	TWWTP_CT	Temperature	°F	1/12/2000	12/26/2007	1240	5.3	5.3	95
NPDES	TWWTP_CT	Tetrachloroethane, 1,1,1,2-	µg/L	6/10/2002	8/17/2007	8	0.25	-1	-0.5
NPDES	TWWTP_CT	Tetrachloroethane, 1,1,2,2-	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Tetrachloroethene (PCE)	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Thallium	µg/L	6/10/2002	8/17/2007	49	0.5	-200	1.2
NPDES	TWWTP_CT	Toluene	µg/L	6/10/2002	8/17/2007	49	0.5	-2	-1
NPDES	TWWTP_CT	Toxaphene	µg/L	6/10/2002	8/17/2007	48	0.25	-1	-0.5
NPDES	TWWTP_CT	TP, 2,4,5-	µg/L	6/10/2002	10/21/2002	8	0.5	-1	-1
NPDES	TWWTP_CT	trans-1,2-dichloroethene	µg/L	6/10/2002	8/17/2007	49	0.25	-1	-0.5
NPDES	TWWTP_CT	Tributyltin	µg/L	10/21/2002	11/6/2002	5	0.01	-0.02	-0.02
NPDES	TWWTP_CT	Tributyltin	ng/L	7/16/2002	7/16/2002	3	2	2	2
NPDES	TWWTP_CT	Trichlorobenzene, 1,2,3-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Trichlorobenzene, 1,2,4-	µg/L	6/10/2002	8/17/2007	49	0.5	-5	-1
NPDES	TWWTP_CT	Trichloroethane, 1,1,1-	µg/L	6/10/2002	8/17/2007	49	0.25	-2	-0.5
NPDES	TWWTP_CT	Trichloroethane, 1,1,2-	µg/L	6/10/2002	8/17/2007	49	0.25	-2	-0.5
NPDES	TWWTP_CT	Trichloroethylene	µg/L	6/10/2002	8/17/2007	49	0.5	-2	-1
NPDES	TWWTP_CT	Trichlorofluoromethane	µg/L	6/10/2002	8/17/2007	49	0.5	-5	-1
NPDES	TWWTP_CT	Trichlorophenol, 2,4,6-	µg/L	6/10/2002	8/17/2007	48	1	-10	-2

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
NPDES	TWWTP_CT	Trichloropropane, 1,2,3-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Trichlorotrifluoroethane	µg/L	6/10/2002	8/17/2007	48	0.38	-10	0.38
NPDES	TWWTP_CT	Trimethylbenzene, 1,2,4-	µg/L	6/10/2002	5/15/2006	7	0.5	-5	-1
NPDES	TWWTP_CT	Trimethylbenzene, 1,3,5-	µg/L	6/10/2002	11/6/2002	6	0.5	-1	-1
NPDES	TWWTP_CT	Turbidity	NTU	1/12/2000	12/26/2007	1240	0.6	0.6	326.2
NPDES	TWWTP_CT	Vinyl Chloride	µg/L	6/10/2002	8/17/2007	49	0.25	-0.5	-0.5
NPDES	TWWTP_CT	Xylene, m/p-	µg/L	6/10/2002	8/17/2007	49	0.25	-1	-0.5
NPDES	TWWTP_CT	Xylene, o-	µg/L	6/10/2002	8/17/2007	38	0.25	-1	-0.5
NPDES	TWWTP_CT	Zinc	µg/L	6/10/2002	8/17/2007	49	0.5	-1	639
NPDES	WDR_RVPW	Aluminum	µg/L	11/16/2002	1/17/2008	20	0.51	0.51	1800
NPDES	WDR_RVPW	Ammonia	mg/L	11/16/2002	1/17/2008	20	0.1	-0.5	16
NPDES	WDR_RVPW	Arsenic	µg/L	11/16/2002	1/17/2008	20	0.01	-0.5	2.4
NPDES	WDR_RVPW	Dissolved Solids	mg/L	11/16/2002	1/17/2008	19	75	75	575
NPDES	WDR_RVPW	Oxygen, Dissolved	mg/L	10/20/2006	1/18/2008	10	6.75	6.75	11.8
NPDES	WDR_RVPW	pH	none	10/20/2006	1/18/2008	10	7.05	7.05	9.06
NPDES	WDR_RVPW	SpecificConductivity	µmhos/cm	12/17/2001	2/14/2008	76	0.15	0.15	1090
NPDES	WDR_RVPW	Temperature	NR	10/20/2006	1/18/2008	10	6.2	6.2	17
NPDES	WDR_RVPW	Turbidity	NTU	10/20/2006	1/18/2008	10	4.73	4.73	46.6
RB5	03AG5001	Ammonia as N	mg/L	9/2/2003	10/7/2003	45	0.0005	-0.001	0.6
RB5	03AG5001	Ammonia as NH3	mg/L	3/26/2003	8/26/2003	129	0.05	-0.1	8
RB5	03AG5001	Chloride	mg/L	9/2/2003	10/7/2003	45	0.005	-0.01	0.73
RB5	03AG5001	Chlorpyrifos	µg/L	8/21/2003	9/15/2003	5	0.019	0.019	0.09
RB5	03AG5001	Diazinon	µg/L	8/21/2003	9/15/2003	5	0.0025	-0.005	0.008
RB5	AD_RB5S	Alkalinity as CaCO3	mg/L	11/3/1995	9/28/2000	59	17	17	220
RB5	AD_RB5S	Ammonia as N	mg/L	9/28/2000	3/29/2007	61	0.05	0.05	1.4
RB5	AD_RB5S	Boron	mg/L	10/2/1995	8/30/2007	11215	0.01	0.01	20

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	AD_RB5S	Calcium	mg/L	10/25/1995	9/28/2000	287	3.8	3.8	390
RB5	AD_RB5S	Chloride	mg/L	10/25/1995	9/28/2000	313	2.2	2.2	780
RB5	AD_RB5S	Chromium	µg/L	10/25/1995	9/28/2000	206	1	1	72
RB5	AD_RB5S	Copper	µg/L	10/25/1995	9/28/2000	241	1.1	1.1	49
RB5	AD_RB5S	Dissolved Solids	mg/L	11/3/1995	9/28/2000	114	44	44	4300
RB5	AD_RB5S	Hardness as CaCO3	mg/L	10/25/1995	9/28/2000	312	18	18	1500
RB5	AD_RB5S	Lead	µg/L	10/27/1995	4/29/1999	2	1	1	14
RB5	AD_RB5S	Magnesium	mg/L	10/25/1995	9/28/2000	287	1.7	1.7	120
RB5	AD_RB5S	Molybdenum	mg/L	10/25/1995	8/16/2007	710	0.00072	0.00072	0.048
RB5	AD_RB5S	Nickel	µg/L	10/25/1995	9/28/2000	112	4.6	4.6	100
RB5	AD_RB5S	Nitrate as N	mg/L	6/29/2000	6/21/2007	257	0.062	0.062	110
RB5	AD_RB5S	Nitrogen, Total Kjeldahl	mg/L	2/25/1999	6/21/2007	175	0.36	0.36	6.7
RB5	AD_RB5S	Nitrogen,Total	mg/L	11/29/2001	12/19/2002	36	0.76	0.76	15.2
RB5	AD_RB5S	OrthoPhosphate as P	mg/L	11/29/2001	6/21/2007	128	0.01	0.01	0.63
RB5	AD_RB5S	Phosphorus as P	mg/L	5/25/2000	6/21/2007	293	0.033	0.033	0.83
RB5	AD_RB5S	Potassium	mg/L	11/3/1995	11/20/2003	295	1	1	15
RB5	AD_RB5S	Selenium	µg/L	10/1/1995	10/25/2007	11710	0.04	0.04	134
RB5	AD_RB5S	Sodium	mg/L	11/3/1995	9/28/2000	58	2.4	2.4	860
RB5	AD_RB5S	SpecificConductivity	µS/cm	10/1/1995	11/1/2007	9797	46.3	46.3	9790
RB5	AD_RB5S	Sulfate	mg/L	10/25/1995	9/28/2000	313	2.7	2.7	2000
RB5	AD_RB5S	Suspended Solids	mg/L	9/26/1996	10/25/2007	823	7.1	7.1	420
RB5	AD_RB5S	Zinc	µg/L	10/25/1995	9/28/2000	159	2	2	120
RB5	SWAMP_RB2	Acenaphthene	µg/L	4/21/2003	6/13/2005	5	0.00859	0.00859	0.0375
RB5	SWAMP_RB2	Alkalinity as CaCO3	mg/L	9/17/2001	2/16/2006	166	27.2	27.2	540
RB5	SWAMP_RB2	Aluminum	µg/L	9/18/2001	6/14/2005	142	0.38	0.38	2618
RB5	SWAMP_RB2	Ammonia as N	mg/L	9/17/2001	6/14/2005	116	0.04	0.04	2.16

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	Anthracene	µg/L	6/18/2002	4/21/2003	2	0.0117	0.0117	0.035
RB5	SWAMP_RB2	Arsenic	µg/L	9/18/2001	2/16/2006	153	0.12	0.12	7.5
RB5	SWAMP_RB2	Benz(a)anthracene	µg/L	9/18/2001	6/13/2005	5	0.00949	0.00949	0.035
RB5	SWAMP_RB2	Benzo(a)pyrene	µg/L	10/2/2001	6/13/2005	4	0.0146	0.0146	0.0576
RB5	SWAMP_RB2	Benzo(b)fluoranthene	µg/L	10/2/2001	6/13/2005	10	0.0104	0.0104	0.0876
RB5	SWAMP_RB2	Benzo(e)pyrene	µg/L	10/2/2001	6/13/2005	5	0.0101	0.0101	0.134
RB5	SWAMP_RB2	Benzo(g,h,i)perylene	µg/L	6/17/2002	6/13/2005	7	0.0151	0.0151	0.255
RB5	SWAMP_RB2	Benzo(k)fluoranthene	µg/L	10/2/2001	6/13/2005	7	0.00877	0.00877	0.035
RB5	SWAMP_RB2	Biphenyl	µg/L	4/21/2003	6/13/2005	5	0.00891	0.00891	0.0232
RB5	SWAMP_RB2	Boron	mg/L	4/10/2002	6/14/2005	95	0.0434	0.0434	2.1
RB5	SWAMP_RB2	Cadmium	µg/L	9/18/2001	6/14/2005	124	0.002	0.002	1
RB5	SWAMP_RB2	Carbofuran	µg/L	1/10/2005	1/10/2005	3	0.103	0.103	0.46
RB5	SWAMP_RB2	Carbophenothion	µg/L	9/26/2001	9/26/2001	1	0.054	0.054	0.054
RB5	SWAMP_RB2	Chlordane, trans-	µg/L	4/22/2003	4/22/2003	1	0.001	0.001	0.001
RB5	SWAMP_RB2	Chlordene, gamma-	µg/L	6/18/2002	6/18/2002	1	0.0015	0.0015	0.0015
RB5	SWAMP_RB2	Chloride	mg/L	9/17/2001	2/16/2006	167	5.28	5.28	763
RB5	SWAMP_RB2	Chlorophyll a	µg/L	9/17/2001	2/16/2006	166	0.05	0.05	92
RB5	SWAMP_RB2	Chlorpyrifos	µg/L	9/26/2001	1/23/2003	6	0.057	0.057	0.11
RB5	SWAMP_RB2	Chromium	µg/L	9/18/2001	2/16/2006	152	0.039	0.039	30.6
RB5	SWAMP_RB2	Chrysene	µg/L	9/19/2001	6/13/2005	6	0.00743	0.00743	0.124
RB5	SWAMP_RB2	Chrysenes, C1 -	µg/L	4/21/2003	6/13/2005	4	0.00679	0.00679	0.156
RB5	SWAMP_RB2	Chrysenes, C2 -	µg/L	4/21/2003	6/13/2005	4	0.00612	0.00612	0.203
RB5	SWAMP_RB2	Chrysenes, C3 -	µg/L	4/21/2003	6/13/2005	3	0.00917	0.00917	0.38
RB5	SWAMP_RB2	Coliform	MPN/100 mL	8/7/2001	8/16/2005	370	2	2	30000
RB5	SWAMP_RB2	Copper	µg/L	9/18/2001	2/16/2006	153	0.05	0.05	30.9
RB5	SWAMP_RB2	Dacthal	µg/L	9/26/2001	6/18/2002	3	0.0015	0.0015	0.005
RB5	SWAMP_RB2	DDE(p,p')	µg/L	4/8/2002	4/22/2003	5	0.0015	0.0015	0.002

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RB5	SWAMP_RB2	DDMU(p,p')	µg/L	9/18/2001	6/18/2002	3	0.0015	0.0015	0.002
RB5	SWAMP_RB2	DDT(p,p')	µg/L	4/8/2002	4/22/2003	4	0.002	0.002	0.0035
RB5	SWAMP_RB2	Diazinon	µg/L	9/18/2001	2/16/2006	55	0.005	0.005	0.741
RB5	SWAMP_RB2	Dibenz(a,h)anthracene	µg/L	4/11/2002	4/21/2003	3	0.035	0.035	0.0448
RB5	SWAMP_RB2	Dibenzothiophene	µg/L	4/21/2003	4/21/2003	1	0.0933	0.0933	0.0933
RB5	SWAMP_RB2	Dibenzothiophenes, C1 -	µg/L	1/21/2003	4/12/2005	13	0.00641	0.00641	0.316
RB5	SWAMP_RB2	Dibenzothiophenes, C2 -	µg/L	1/21/2003	6/13/2005	17	0.00565	0.00565	0.623
RB5	SWAMP_RB2	Dibenzothiophenes, C3 -	µg/L	1/21/2003	4/12/2005	10	0.0075	0.0075	0.411
RB5	SWAMP_RB2	Dieldrin	µg/L	6/17/2002	6/3/2003	5	0.0015	0.0015	0.003
RB5	SWAMP_RB2	Dimethylnaphthalene, 2,6-	µg/L	4/12/2005	6/13/2005	2	0.0071	0.0071	0.0271
RB5	SWAMP_RB2	Dioxathion	µg/L	9/26/2001	9/26/2001	1	0.04	0.04	0.04
RB5	SWAMP_RB2	Dissolved Organic Carbon	mg/L	9/17/2001	6/14/2005	120	0.7	0.7	23.9
RB5	SWAMP_RB2	Dissolved Solids	mg/L	9/17/2001	2/16/2006	166	108	108	3220
RB5	SWAMP_RB2	Disulfoton	µg/L	9/26/2001	2/16/2006	17	0.012	0.012	0.037
RB5	SWAMP_RB2	Diuron	µg/L	1/11/2005	1/11/2005	2	1.77	1.77	1.8
RB5	SWAMP_RB2	E. coli	MPN/100 mL	8/7/2001	8/16/2005	230	2	2	24000
RB5	SWAMP_RB2	Endosulfan I	µg/L	9/18/2001	9/19/2001	2	0.007	0.007	0.008
RB5	SWAMP_RB2	Fluoranthene	µg/L	4/21/2003	6/13/2005	7	0.00544	0.00544	0.0794
RB5	SWAMP_RB2	Fluoranthene/Pyrenes, C1 -	µg/L	4/21/2003	6/13/2005	8	0.00516	0.00516	0.106
RB5	SWAMP_RB2	Fluorene	µg/L	9/19/2001	6/13/2005	5	0.013	0.013	0.035
RB5	SWAMP_RB2	Fluorenes, C1 -	µg/L	4/21/2003	4/12/2005	6	0.007	0.007	0.0568
RB5	SWAMP_RB2	Fluorenes, C3 -	µg/L	4/21/2003	4/12/2005	10	0.00714	0.00714	0.102
RB5	SWAMP_RB2	Fonofos	µg/L	6/17/2002	6/18/2002	4	0.03	0.03	0.03
RB5	SWAMP_RB2	Hardness as CaCO3	mg/L	9/17/2001	2/16/2006	154	34.6	34.6	1060
RB5	SWAMP_RB2	HCH, gamma	µg/L	10/2/2001	10/2/2001	1	0.003	0.003	0.003
RB5	SWAMP_RB2	Hexachlorobenzene	µg/L	4/8/2002	4/10/2002	2	0.00075	0.00075	0.00075
RB5	SWAMP_RB2	Indeno(1,2,3-c,d)pyrene	µg/L	4/21/2003	6/13/2005	4	0.0154	0.0154	0.131

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RB5	SWAMP_RB2	Lead	µg/L	9/18/2001	2/16/2006	142	0.002	0.002	17.6
RB5	SWAMP_RB2	Manganese	µg/L	9/18/2001	2/16/2006	153	0.06	0.06	1439
RB5	SWAMP_RB2	Mercury	ng/L	9/18/2001	2/16/2006	76	0.51	0.51	79.6
RB5	SWAMP_RB2	Methyldibenzothiophene, 4-	µg/L	4/12/2005	4/12/2005	1	0.00619	0.00619	0.00619
RB5	SWAMP_RB2	Methylfluorene, 1-	µg/L	4/12/2005	4/12/2005	1	0.0228	0.0228	0.0228
RB5	SWAMP_RB2	Methylnaphthalene, 1-	µg/L	9/18/2001	6/13/2005	9	0.0101	0.0101	0.0454
RB5	SWAMP_RB2	Methylnaphthalene, 2-	µg/L	4/21/2003	6/13/2005	8	0.0065	0.0065	0.0563
RB5	SWAMP_RB2	Methylphenanthrene, 1-	µg/L	4/21/2003	4/12/2005	2	0.0068	0.0068	0.0159
RB5	SWAMP_RB2	Mevinphos	µg/L	9/26/2001	9/26/2001	1	0.056	0.056	0.056
RB5	SWAMP_RB2	Naphthalene	µg/L	4/21/2003	6/14/2005	17	0.00537	0.00537	0.417
RB5	SWAMP_RB2	Naphthalenes, C1 -	µg/L	1/21/2003	6/13/2005	11	0.00705	0.00705	0.0993
RB5	SWAMP_RB2	Naphthalenes, C2 -	µg/L	1/21/2003	6/13/2005	15	0.00596	0.00596	0.1646
RB5	SWAMP_RB2	Naphthalenes, C3 -	µg/L	1/21/2003	4/12/2005	21	0.00594	0.00594	0.163
RB5	SWAMP_RB2	Naphthalenes, C4 -	µg/L	1/21/2003	4/12/2005	9	0.0098	0.0098	0.305
RB5	SWAMP_RB2	Nickel	µg/L	9/18/2001	2/16/2006	148	0.135	0.135	33.7
RB5	SWAMP_RB2	Nitrate as N	mg/L	9/17/2001	2/16/2006	166	0.0227	0.0227	8.52
RB5	SWAMP_RB2	Nitrite as N	mg/L	9/17/2001	2/16/2006	131	0.0051	0.0051	0.13
RB5	SWAMP_RB2	Nitrogen, Total Kjeldahl	mg/L	9/17/2001	2/16/2006	156	0.12	0.12	3.16
RB5	SWAMP_RB2	OrthoPhosphate as P	mg/L	9/17/2001	2/16/2006	166	0.007	0.007	1.99
RB5	SWAMP_RB2	Oxadiazon	µg/L	9/18/2001	6/14/2005	40	0.0015	0.0015	0.364
RB5	SWAMP_RB2	Parathion, Methyl	µg/L	9/26/2001	9/26/2001	1	0.03	0.03	0.03
RB5	SWAMP_RB2	PCB 005	µg/L	9/18/2001	9/18/2001	1	0.003	0.003	0.003
RB5	SWAMP_RB2	PCB 018	µg/L	9/19/2001	9/19/2001	1	0.002	0.002	0.002
RB5	SWAMP_RB2	PCB 101	µg/L	9/19/2001	9/19/2001	1	0.003	0.003	0.003
RB5	SWAMP_RB2	Perylene	µg/L	6/17/2002	4/21/2003	6	0.0334	0.0334	0.12
RB5	SWAMP_RB2	Phenanthrene	µg/L	6/18/2002	6/14/2005	11	0.0051	0.0051	0.0429
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C1 -	µg/L	1/21/2003	6/13/2005	17	0.0063	0.0063	0.139

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C2 -	µg/L	1/21/2003	6/13/2005	19	0.00601	0.00601	0.178
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C3 -	µg/L	1/21/2003	6/13/2005	12	0.0061	0.0061	0.186
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C4 -	µg/L	4/21/2003	6/13/2005	3	0.00643	0.00643	0.0699
RB5	SWAMP_RB2	Pheophytin a	µg/L	9/17/2001	6/19/2002	84	0.75	0.75	51
RB5	SWAMP_RB2	Phosphorus as P	mg/L	9/17/2001	2/16/2006	151	0.0302	0.0302	0.61
RB5	SWAMP_RB2	Propazine	µg/L	6/18/2002	6/18/2002	1	0.035	0.035	0.035
RB5	SWAMP_RB2	Pyrene	µg/L	6/17/2002	6/13/2005	9	0.00665	0.00665	0.126
RB5	SWAMP_RB2	Secbumeton	µg/L	6/18/2002	6/18/2002	1	0.035	0.035	0.035
RB5	SWAMP_RB2	Selenium	µg/L	9/18/2001	2/16/2006	153	0.165	0.165	18.8
RB5	SWAMP_RB2	Silver	µg/L	9/18/2001	6/14/2005	15	0.009	0.009	0.775
RB5	SWAMP_RB2	Simazine	µg/L	1/10/2005	1/11/2005	7	0.024	0.024	0.083
RB5	SWAMP_RB2	Sulfate	mg/L	9/17/2001	2/16/2006	166	4.18	4.18	1410
RB5	SWAMP_RB2	Suspended Sediment Concentration	mg/L	9/17/2001	2/16/2006	141	0.1	0.1	344.5
RB5	SWAMP_RB2	Suspended Solids	mg/L	9/17/2001	1/30/2002	23	1	1	53
RB5	SWAMP_RB2	Thiobencarb	µg/L	9/26/2001	9/26/2001	1	0.21	0.21	0.21
RB5	SWAMP_RB2	Total Organic Carbon	mg/L	9/17/2001	2/16/2006	159	0.9	0.9	58
RB5	SWAMP_RB2	Trimethylnaphthalene, 2,3,5-	µg/L	9/18/2001	4/12/2005	3	0.0149	0.0149	0.035
RB5	SWAMP_RB2	Zinc	µg/L	9/18/2001	2/16/2006	151	0.22	0.22	271
RB5	SWAMP_RB5L	Alkalinity as CaCO3	mg/L	11/3/2000	3/25/2002	221	7	7	218
RB5	SWAMP_RB5L	Aluminum	µg/L	4/6/2006	4/6/2006	1	21.1	21.1	21.1
RB5	SWAMP_RB5L	Ammonia as N	mg/L	1/17/2001	3/25/2002	68	0.008	0.008	0.411
RB5	SWAMP_RB5L	Arsenic	µg/L	4/6/2006	4/6/2006	1	3.45	3.45	3.45
RB5	SWAMP_RB5L	Chromium	µg/L	4/6/2006	4/6/2006	1	0.6	0.6	0.6
RB5	SWAMP_RB5L	Copper	µg/L	4/6/2006	4/6/2006	1	4.91	4.91	4.91
RB5	SWAMP_RB5L	Hardness as CaCO3	mg/L	11/3/2000	3/25/2002	222	2.9	2.9	480
RB5	SWAMP_RB5L	Lead	µg/L	4/6/2006	4/6/2006	1	0.25	0.25	0.25

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RB5	SWAMP_RB5L	Manganese	µg/L	4/6/2006	4/6/2006	1	87	87	87
RB5	SWAMP_RB5L	Nickel	µg/L	4/6/2006	4/6/2006	1	1.94	1.94	1.94
RB5	SWAMP_RB5L	Selenium	µg/L	4/6/2006	4/6/2006	1	0.33	0.33	0.33
RB5	SWAMP_RB5L	Zinc	µg/L	3/21/2005	4/6/2006	16	0.98	0.98	3.98
RB5	SWAMP_RB5S	Alkalinity as CaCO3	mg/L	10/24/2000	11/21/2002	545	10	10	1100
RB5	SWAMP_RB5S	Ammonia as N	mg/L	11/29/2000	12/19/2002	132	0.05	0.05	22
RB5	SWAMP_RB5S	Arsenic	µg/L	6/7/2001	6/26/2003	299	2	2	13
RB5	SWAMP_RB5S	BOD	mg/L	10/12/2000	1/30/2003	1538	0.1	0.1	33.5
RB5	SWAMP_RB5S	Boron	mg/L	10/24/2000	6/30/2003	534	0.05	0.05	8.4
RB5	SWAMP_RB5S	Cadmium	µg/L	6/7/2001	6/24/2003	16	0.11	0.11	60
RB5	SWAMP_RB5S	Calcium	mg/L	10/24/2000	6/30/2003	738	3.8	3.8	350
RB5	SWAMP_RB5S	Chloride	mg/L	10/24/2000	6/30/2003	716	2.2	2.2	690
RB5	SWAMP_RB5S	Chromium	µg/L	10/24/2000	6/26/2003	673	1	1	110
RB5	SWAMP_RB5S	Coliform	MPN/100 mL	3/11/2002	6/28/2007	1046	14.4	14.4	2419.6
RB5	SWAMP_RB5S	Copper	µg/L	10/24/2000	6/30/2003	1193	1	1	98
RB5	SWAMP_RB5S	Dissolved Solids	mg/L	10/5/2000	11/26/2002	518	24	24	4400
RB5	SWAMP_RB5S	E. coli	MPN/100 mL	3/11/2002	6/28/2007	1032	1	1	2419.6
RB5	SWAMP_RB5S	Hardness as CaCO3	mg/L	10/24/2000	6/30/2003	783	0.29	0.29	1300
RB5	SWAMP_RB5S	Lead	µg/L	10/24/2000	6/26/2003	38	5.2	5.2	46
RB5	SWAMP_RB5S	Magnesium	mg/L	10/24/2000	6/30/2003	738	1.3	1.3	110
RB5	SWAMP_RB5S	Mercury	ng/L	6/19/2002	6/26/2003	4	0.23	0.23	290
RB5	SWAMP_RB5S	Nickel	µg/L	10/24/2000	6/26/2003	273	2.7	2.7	180
RB5	SWAMP_RB5S	Nitrate as N	mg/L	10/24/2000	2/27/2003	272	1	1	77
RB5	SWAMP_RB5S	Nitrogen, Total Kjeldahl	mg/L	10/24/2000	2/27/2003	193	0.06	0.06	32
RB5	SWAMP_RB5S	Nitrogen,Total	mg/L	11/27/2001	12/19/2002	180	0.11	0.11	30.7
RB5	SWAMP_RB5S	OrthoPhosphate as P	mg/L	10/24/2000	12/19/2002	178	0.03	0.03	11
RB5	SWAMP_RB5S	Phosphorus as P	mg/L	10/24/2000	2/27/2003	400	0.02	0.02	12

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB5S	Potassium	mg/L	10/24/2000	2/27/2003	540	0.67	0.67	100
RB5	SWAMP_RB5S	Sodium	mg/L	10/24/2000	11/21/2002	571	2.1	2.1	800
RB5	SWAMP_RB5S	SpecificConductivity	µS/cm	10/5/2000	8/28/2002	638	43.1	43.1	5730
RB5	SWAMP_RB5S	Sulfate	mg/L	10/24/2000	6/30/2003	717	2.2	2.2	1900
RB5	SWAMP_RB5S	Suspended Solids	mg/L	10/5/2000	3/29/2007	1237	1	1	6200
RB5	SWAMP_RB5S	Total Organic Carbon	mg/L	10/12/2000	3/29/2007	1455	1	1	53
RB5	SWAMP_RB5S	Zinc	µg/L	10/24/2000	6/30/2003	860	2	2	240

Table 5. POD Contaminant Database – Sediment Chemistry Data. Summary of sediment chemistry results by data source, projectID and analyte name. Results are tabulated by start and end date of samples, result counts, minimum of results with non detects quantified as one half the MDL, minimum of result and maximum of result.

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
UCD RDC	04AG5001	Bifenthrin	µg/Kg	8/10/2004	12/7/2005	37	0.165	-0.33	286.39
UCD RDC	04AG5001	Chlordane, Alpha-	µg/Kg	8/10/2004	12/7/2005	37	0.3	-0.6	1.24
UCD RDC	04AG5001	Chlordane, gamma-	µg/Kg	8/10/2004	12/7/2005	37	0.15	-0.3	-0.3
UCD RDC	04AG5001	Chlorpyrifos	µg/Kg	8/10/2004	12/7/2005	37	0.22	-0.44	5.69
UCD RDC	04AG5001	Copper	mg/Kg	8/30/2004	8/10/2005	3	8.17	8.17	70.9
UCD RDC	04AG5001	DDD(p,p')	µg/Kg	8/10/2004	12/7/2005	37	0.2	-0.4	9.8
UCD RDC	04AG5001	DDE(p,p')	µg/Kg	8/10/2004	12/7/2005	37	0.185	-0.37	74.57
UCD RDC	04AG5001	DDT(p,p')	µg/Kg	8/10/2004	12/7/2005	37	0.195	-0.39	28.53
UCD RDC	04AG5001	Dieldrin	µg/Kg	8/10/2004	12/7/2005	37	0.305	-0.61	3.88
UCD RDC	04AG5001	Permethrin, total	µg/Kg	8/10/2004	12/7/2005	37	0.295	-0.59	23.6
UCD RDC	04AG5001	Permethrin-1	µg/Kg	8/10/2004	12/7/2005	37	0.14	-0.28	14.31
UCD RDC	04AG5001	Permethrin-2	µg/Kg	8/10/2004	12/7/2005	37	0.155	-0.31	10.26
UCD RDC	04AG5001	Selenium	mg/Kg	8/30/2004	8/10/2005	3	0.05	-0.1	0.24
BDAT	SJRDO UOP	Chlorophyll a	m/hr	6/14/2001	10/16/2001	12	0.0097	0.0097	0.1412
BDAT	SJRDO UOP	Chlorophyll a	mg/(m 2 hr)	6/14/2001	10/25/2001	27	0.1008	0.1008	1.7375
BDAT	SJRDO UOP	Chlorophyll a	mg/hr	6/14/2001	10/25/2001	13	0.3715	0.3715	4.2502
BDAT	SJRDO UOP	Chlorophyll a	mg/L	8/16/2000	11/9/2000	11	0.0039	0.0039	0.0288
BDAT	SJRDO UOP	Chlorophyll a + Pheophytin a	mg/L	8/16/2000	11/9/2000	7	0.007	0.007	0.0621
BDAT	SJRDO UOP	Pheophytin a	mg/hr	6/14/2001	10/25/2001	52	0.3769	0.3769	16.6951
BDAT	SJRDO UOP	Solids	g/(m 2 hr)	6/14/2001	10/25/2001	23	0.591	0.591	135.0473
BDAT	SJRDO UOP	Solids	m/hr	6/21/2001	10/25/2001	12	0.1332	0.1332	3.1095
BDAT	SJRDO UOP	Solids	mg/hr	6/14/2001	10/25/2001	17	0.6488	0.6488	156.0204
BDAT	SJRDO UOP	Solids	mg/L	8/16/2000	11/9/2000	9	2.5333	2.5333	30
BDAT	SJRDO USGS	Sediment	%	6/12/2001	9/19/2001	39	25	25	99
BDAT	SJRDO USGS	Sediment	mg/L	6/13/2001	9/20/2001	25	21	21	1160
RB5	SWAMP_RB2	Acenaphthene	ng/g dw	9/19/2001	4/12/2005	9	2.07	2.07	8.95

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	Acenaphthylene	ng/g dw	9/19/2001	4/12/2005	4	2	2	6.54
RB5	SWAMP_RB2	Aldrin	ng/g dw	4/12/2005	4/12/2005	1	0.326	0.326	0.326
RB5	SWAMP_RB2	Aluminum	mg/Kg dw	9/18/2001	4/12/2005	23	10150	10150	52811
RB5	SWAMP_RB2	Anthracene	ng/g dw	9/19/2001	4/12/2005	13	1.36	1.36	32.3
RB5	SWAMP_RB2	Arsenic	mg/Kg dw	9/18/2001	4/12/2005	23	1.04	1.04	12
RB5	SWAMP_RB2	Benz(a)anthracene	ng/g dw	9/18/2001	4/12/2005	20	1.11	1.11	149
RB5	SWAMP_RB2	Benzo(a)pyrene	ng/g dw	9/18/2001	4/12/2005	20	1.24	1.24	278
RB5	SWAMP_RB2	Benzo(b)fluoranthene	ng/g dw	9/18/2001	4/12/2005	23	1.21	1.21	351
RB5	SWAMP_RB2	Benzo(e)pyrene	ng/g dw	9/18/2001	4/12/2005	20	1.73	1.73	212
RB5	SWAMP_RB2	Benzo(g,h,i)perylene	ng/g dw	9/18/2001	4/12/2005	22	2.27	2.27	293
RB5	SWAMP_RB2	Benzo(k)fluoranthene	ng/g dw	9/19/2001	4/12/2005	17	1.67	1.67	131
RB5	SWAMP_RB2	Bifenthrin	ng/g dw	4/12/2005	4/12/2005	3	0.862	0.862	2.58
RB5	SWAMP_RB2	Biphenyl	ng/g dw	9/19/2001	4/12/2005	13	1.4	1.4	11.5
RB5	SWAMP_RB2	Cadmium	mg/Kg dw	9/18/2001	4/12/2005	23	0.07	0.07	1.24
RB5	SWAMP_RB2	Chlordane, cis-	ng/g dw	9/19/2001	4/12/2005	13	1.56	1.56	11.6
RB5	SWAMP_RB2	Chlordane, trans-	ng/g dw	9/19/2001	4/12/2005	16	0.62	0.62	16.5
RB5	SWAMP_RB2	Chlordene, alpha-	ng/g dw	6/17/2002	6/17/2002	1	2.74	2.74	2.74
RB5	SWAMP_RB2	Chlordene, gamma-	ng/g dw	6/17/2002	4/22/2003	2	1.02	1.02	2.16
RB5	SWAMP_RB2	Chlorpyrifos	ng/g dw	6/17/2002	4/21/2003	3	1.94	1.94	3.85
RB5	SWAMP_RB2	Chromium	mg/Kg dw	9/18/2001	4/12/2005	23	26.8	26.8	475
RB5	SWAMP_RB2	Chrysene	ng/g dw	9/18/2001	4/12/2005	22	2.1	2.1	204
RB5	SWAMP_RB2	Chrysenes, C1 -	ng/g dw	9/18/2001	4/12/2005	22	1.43	1.43	182
RB5	SWAMP_RB2	Chrysenes, C2 -	ng/g dw	9/18/2001	4/12/2005	22	1.32	1.32	318
RB5	SWAMP_RB2	Chrysenes, C3 -	ng/g dw	9/18/2001	4/12/2005	20	3.32	3.32	233
RB5	SWAMP_RB2	Clay <0.005 mm	%	9/18/2001	4/12/2005	14	0.91	0.91	37
RB5	SWAMP_RB2	Copper	mg/Kg dw	9/18/2001	4/12/2005	23	10.6	10.6	73.4
RB5	SWAMP_RB2	Cypermethrin, total	ng/g dw	4/12/2005	4/12/2005	1	32.28	32.28	32.28
RB5	SWAMP_RB2	Dacthal	ng/g dw	9/19/2001	6/18/2002	2	1.12	1.12	5.35

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	DDD(o,p')	ng/g dw	6/17/2002	4/12/2005	8	1.21	1.21	8.39
RB5	SWAMP_RB2	DDD(p,p')	ng/g dw	9/19/2001	4/12/2005	13	1.05	1.05	31.7
RB5	SWAMP_RB2	DDE(p,p')	ng/g dw	9/19/2001	4/12/2005	18	1.19	1.19	30
RB5	SWAMP_RB2	DDMU(p,p')	ng/g dw	4/12/2005	4/12/2005	2	1.95	1.95	5.03
RB5	SWAMP_RB2	DDT(o,p')	ng/g dw	6/17/2002	4/12/2005	3	1.7	1.7	4.42
RB5	SWAMP_RB2	DDT(p,p')	ng/g dw	6/17/2002	4/12/2005	9	3.4	3.4	22.4
RB5	SWAMP_RB2	Dibenz(a,h)anthracene	ng/g dw	9/18/2001	4/12/2005	21	1.83	1.83	86.8
RB5	SWAMP_RB2	Dibenzothiophene	ng/g dw	9/18/2001	4/12/2005	14	1.26	1.26	38.3
RB5	SWAMP_RB2	Dibenzothiophenes, C1 -	ng/g dw	9/18/2001	4/12/2005	18	1.7	1.7	142
RB5	SWAMP_RB2	Dibenzothiophenes, C2 -	ng/g dw	9/18/2001	4/12/2005	20	1.57	1.57	418
RB5	SWAMP_RB2	Dibenzothiophenes, C3 -	ng/g dw	9/18/2001	4/12/2005	19	1.87	1.87	694
RB5	SWAMP_RB2	Dieldrin	ng/g dw	9/26/2001	4/12/2005	11	0.795	0.795	12.6
RB5	SWAMP_RB2	Dimethylnaphthalene, 2,6-	ng/g dw	9/18/2001	4/12/2005	19	1.77	1.77	9.51
RB5	SWAMP_RB2	Dimethylphenanthrene, 3,6-	ng/g dw	4/12/2005	4/12/2005	7	1.55	1.55	9.42
RB5	SWAMP_RB2	Fine <0.075 mm	%	4/11/2005	4/12/2005	5	0.11	0.11	1.26
RB5	SWAMP_RB2	Fluoranthene	ng/g dw	9/18/2001	4/12/2005	22	1.43	1.43	468
RB5	SWAMP_RB2	Fluoranthene/Pyrenes, C1 -	ng/g dw	9/18/2001	4/12/2005	22	1.58	1.58	303
RB5	SWAMP_RB2	Fluorene	ng/g dw	9/19/2001	4/12/2005	11	1.66	1.66	10.5
RB5	SWAMP_RB2	Fluorenes, C1 -	ng/g dw	9/19/2001	4/12/2005	16	2.17	2.17	9.4
RB5	SWAMP_RB2	Fluorenes, C2 -	ng/g dw	9/18/2001	4/12/2005	12	1.6	1.6	26
RB5	SWAMP_RB2	Fluorenes, C3 -	ng/g dw	9/26/2001	4/12/2005	20	2.25	2.25	76.9
RB5	SWAMP_RB2	Gravel 4.75 to <75 mm	%	6/17/2002	4/12/2005	17	0.22	0.22	73.92
RB5	SWAMP_RB2	Heptachlor	ng/g dw	4/22/2003	4/22/2003	1	0.848	0.848	0.848
RB5	SWAMP_RB2	Heptachlor epoxide	ng/g dw	9/18/2001	4/12/2005	11	0.708	0.708	3.2
RB5	SWAMP_RB2	Hexachlorobenzene	ng/g dw	9/19/2001	4/12/2005	8	0.152	0.152	132
RB5	SWAMP_RB2	Indeno(1,2,3-c,d)pyrene	ng/g dw	9/18/2001	4/12/2005	22	1.91	1.91	377
RB5	SWAMP_RB2	Lead	mg/Kg dw	9/18/2001	4/12/2005	23	4.24	4.24	130
RB5	SWAMP_RB2	Manganese	mg/Kg dw	9/18/2001	4/12/2005	23	113	113	4655

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	Mercury	mg/Kg dw	9/18/2001	4/12/2005	17	0.006	0.006	1.171
RB5	SWAMP_RB2	Methyldibenzothiophene, 4-	ng/g dw	4/12/2005	4/12/2005	3	1.63	1.63	6.46
RB5	SWAMP_RB2	Methylfluoranthene, 2-	ng/g dw	4/11/2005	4/12/2005	8	1.57	1.57	8.52
RB5	SWAMP_RB2	Methylfluorene, 1-	ng/g dw	4/12/2005	4/12/2005	4	1.66	1.66	2.69
RB5	SWAMP_RB2	Methylnaphthalene, 1-	ng/g dw	9/19/2001	4/12/2005	18	1.5	1.5	8.76
RB5	SWAMP_RB2	Methylnaphthalene, 2-	ng/g dw	9/19/2001	4/12/2005	18	2.47	2.47	16.7
RB5	SWAMP_RB2	Methylphenanthrene, 1-	ng/g dw	9/19/2001	4/12/2005	17	1.89	1.89	49.4
RB5	SWAMP_RB2	Moisture	%	9/18/2001	4/12/2005	122	1.21	1.21	91.908
RB5	SWAMP_RB2	Naphthalene	ng/g dw	9/18/2001	4/12/2005	21	0.94	0.94	17.7
RB5	SWAMP_RB2	Naphthalenes, C1 -	ng/g dw	9/19/2001	4/12/2005	20	2.2	2.2	26.7
RB5	SWAMP_RB2	Naphthalenes, C2 -	ng/g dw	9/18/2001	4/12/2005	21	3.74	3.74	29.8
RB5	SWAMP_RB2	Naphthalenes, C3 -	ng/g dw	9/19/2001	4/12/2005	20	3.76	3.76	26.2
RB5	SWAMP_RB2	Naphthalenes, C4 -	ng/g dw	9/19/2001	4/12/2005	18	1.58	1.58	17
RB5	SWAMP_RB2	Nickel	mg/Kg dw	9/18/2001	4/12/2005	23	13.7	13.7	269
RB5	SWAMP_RB2	Nonachlor, cis-	ng/g dw	6/17/2002	4/12/2005	7	1.18	1.18	5.36
RB5	SWAMP_RB2	Nonachlor, trans-	ng/g dw	9/19/2001	4/12/2005	18	0.53	0.53	21.7
RB5	SWAMP_RB2	Oxadiazon	ng/g dw	9/18/2001	4/12/2005	15	1.65	1.65	267
RB5	SWAMP_RB2	Oxychlordane	ng/g dw	6/17/2002	4/12/2005	3	0.503	0.503	1.54
RB5	SWAMP_RB2	Parathion, Ethyl	ng/g dw	9/19/2001	9/19/2001	1	6.17	6.17	6.17
RB5	SWAMP_RB2	PCB 008	ng/g dw	6/17/2002	6/17/2002	1	0.508	0.508	0.508
RB5	SWAMP_RB2	PCB 018	ng/g dw	6/17/2002	4/12/2005	8	0.15	0.15	0.876
RB5	SWAMP_RB2	PCB 027	ng/g dw	9/18/2001	9/18/2001	1	0.212	0.212	0.212
RB5	SWAMP_RB2	PCB 028	ng/g dw	9/19/2001	4/12/2005	20	0.091	0.091	1.97
RB5	SWAMP_RB2	PCB 031	ng/g dw	9/18/2001	4/12/2005	16	0.163	0.163	1.22
RB5	SWAMP_RB2	PCB 033	ng/g dw	9/18/2001	4/12/2005	12	0.086	0.086	1.05
RB5	SWAMP_RB2	PCB 044	ng/g dw	9/19/2001	4/12/2005	17	0.191	0.191	1.76
RB5	SWAMP_RB2	PCB 049	ng/g dw	6/17/2002	4/12/2005	13	0.091	0.091	0.929
RB5	SWAMP_RB2	PCB 052	ng/g dw	9/19/2001	4/12/2005	22	0.211	0.211	2.75

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	PCB 056	ng/g dw	6/17/2002	4/12/2005	9	0.059	0.059	0.428
RB5	SWAMP_RB2	PCB 060	ng/g dw	6/17/2002	4/22/2003	6	0.066	0.066	0.368
RB5	SWAMP_RB2	PCB 066	ng/g dw	9/19/2001	4/12/2005	17	0.133	0.133	4.36
RB5	SWAMP_RB2	PCB 070	ng/g dw	9/19/2001	4/12/2005	16	0.152	0.152	1.65
RB5	SWAMP_RB2	PCB 074	ng/g dw	6/17/2002	4/12/2005	6	0.168	0.168	0.472
RB5	SWAMP_RB2	PCB 087	ng/g dw	9/18/2001	4/12/2005	19	0.114	0.114	2.56
RB5	SWAMP_RB2	PCB 095	ng/g dw	9/19/2001	4/12/2005	21	0.177	0.177	4.62
RB5	SWAMP_RB2	PCB 097	ng/g dw	6/17/2002	4/12/2005	13	0.062	0.062	1.85
RB5	SWAMP_RB2	PCB 099	ng/g dw	9/19/2001	4/12/2005	15	0.081	0.081	2.17
RB5	SWAMP_RB2	PCB 101	ng/g dw	9/18/2001	4/12/2005	23	0.148	0.148	5.58
RB5	SWAMP_RB2	PCB 105	ng/g dw	9/26/2001	4/12/2005	19	0.139	0.139	1.43
RB5	SWAMP_RB2	PCB 110	ng/g dw	9/18/2001	4/12/2005	23	0.147	0.147	5.63
RB5	SWAMP_RB2	PCB 114	ng/g dw	6/17/2002	4/12/2005	5	0.095	0.095	0.673
RB5	SWAMP_RB2	PCB 118	ng/g dw	9/18/2001	4/12/2005	23	0.224	0.224	5.43
RB5	SWAMP_RB2	PCB 128	ng/g dw	6/17/2002	4/12/2005	10	0.09	0.09	1.12
RB5	SWAMP_RB2	PCB 137	ng/g dw	6/17/2002	4/12/2005	4	0.155	0.155	0.627
RB5	SWAMP_RB2	PCB 138	ng/g dw	9/18/2001	4/12/2005	23	0.163	0.163	11.9
RB5	SWAMP_RB2	PCB 141	ng/g dw	6/17/2002	4/12/2005	9	0.075	0.075	2.12
RB5	SWAMP_RB2	PCB 149	ng/g dw	9/19/2001	4/12/2005	17	0.338	0.338	10.5
RB5	SWAMP_RB2	PCB 151	ng/g dw	9/26/2001	4/12/2005	14	0.131	0.131	3.96
RB5	SWAMP_RB2	PCB 153	ng/g dw	9/19/2001	4/12/2005	18	0.181	0.181	12.1
RB5	SWAMP_RB2	PCB 156	ng/g dw	6/17/2002	4/12/2005	9	0.061	0.061	1.1
RB5	SWAMP_RB2	PCB 157	ng/g dw	6/17/2002	4/12/2005	3	0.094	0.094	0.241
RB5	SWAMP_RB2	PCB 158	ng/g dw	6/17/2002	4/12/2005	9	0.152	0.152	0.956
RB5	SWAMP_RB2	PCB 170	ng/g dw	9/26/2001	4/12/2005	12	0.056	0.056	3.71
RB5	SWAMP_RB2	PCB 174	ng/g dw	9/26/2001	4/12/2005	10	0.301	0.301	4.46
RB5	SWAMP_RB2	PCB 177	ng/g dw	6/17/2002	4/12/2005	9	0.118	0.118	2.96
RB5	SWAMP_RB2	PCB 180	ng/g dw	9/19/2001	4/12/2005	18	0.187	0.187	9.2

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB2	PCB 183	ng/g dw	6/17/2002	4/12/2005	9	0.096	0.096	2.52
RB5	SWAMP_RB2	PCB 187	ng/g dw	9/19/2001	4/12/2005	15	0.065	0.065	5.69
RB5	SWAMP_RB2	PCB 194	ng/g dw	9/26/2001	4/12/2005	10	0.173	0.173	2.36
RB5	SWAMP_RB2	PCB 195	ng/g dw	6/17/2002	4/12/2005	6	0.135	0.135	0.94
RB5	SWAMP_RB2	PCB 200	ng/g dw	6/17/2002	4/12/2005	5	0.128	0.128	0.454
RB5	SWAMP_RB2	PCB 201	ng/g dw	9/26/2001	4/12/2005	11	0.225	0.225	2.53
RB5	SWAMP_RB2	PCB 203	ng/g dw	9/26/2001	4/12/2005	11	0.233	0.233	1.82
RB5	SWAMP_RB2	PCB 206	ng/g dw	9/26/2001	4/12/2005	9	0.097	0.097	1.23
RB5	SWAMP_RB2	PCB 209	ng/g dw	9/26/2001	4/12/2005	3	0.287	0.287	0.353
RB5	SWAMP_RB2	PCB AROCLOR 1248	ng/g dw	6/17/2002	6/17/2002	2	37	37	38
RB5	SWAMP_RB2	PCB AROCLOR 1254	ng/g dw	6/17/2002	4/12/2005	13	5	5	73
RB5	SWAMP_RB2	PCB AROCLOR 1260	ng/g dw	6/17/2002	4/12/2005	12	5	5	86
RB5	SWAMP_RB2	Permethrin, total	ng/g dw	4/11/2005	4/11/2005	1	6.43	6.43	6.43
RB5	SWAMP_RB2	Perylene	ng/g dw	9/18/2001	4/12/2005	20	2.94	2.94	56
RB5	SWAMP_RB2	Phenanthrene	ng/g dw	9/18/2001	4/12/2005	22	1.31	1.31	206
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C1 -	ng/g dw	9/18/2001	4/12/2005	23	1.68	1.68	142
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C2 -	ng/g dw	9/18/2001	4/12/2005	23	1.7	1.7	379
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C3 -	ng/g dw	9/18/2001	4/12/2005	22	1.27	1.27	521
RB5	SWAMP_RB2	Phenanthrene/Anthracene, C4 -	ng/g dw	9/18/2001	4/12/2005	20	1.6	1.6	388
RB5	SWAMP_RB2	Pyrene	ng/g dw	9/18/2001	4/12/2005	23	1.27	1.27	395
RB5	SWAMP_RB2	Sand 0.075 to <4.75 mm	%	9/18/2001	4/12/2005	67	0.04	0.04	96.73
RB5	SWAMP_RB2	Selenium	mg/Kg dw	4/11/2005	4/12/2005	10	0.06	0.06	0.59
RB5	SWAMP_RB2	Silt 0.005 to <0.075 mm	%	9/18/2001	4/12/2005	16	0.13	0.13	68.41
RB5	SWAMP_RB2	Silver	mg/Kg dw	9/18/2001	4/12/2005	23	0.0963	0.0963	0.499
RB5	SWAMP_RB2	Tedion	ng/g dw	9/19/2001	4/12/2005	6	1.52	1.52	44.4
RB5	SWAMP_RB2	Total Organic Carbon	%	9/18/2001	4/12/2005	23	0.16	0.16	9.42
RB5	SWAMP_RB2	Trimethylnaphthalene, 2,3,5-	ng/g dw	6/17/2002	4/12/2005	6	1.8	1.8	4.34
RB5	SWAMP_RB2	Zinc	mg/Kg dw	9/18/2001	4/12/2005	23	5.78	5.78	320

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB5L	Bifenthrin	ng/g dw	9/24/2004	11/7/2004	22	1.2	1.2	436.6
RB5	SWAMP_RB5L	Chlordane, cis-	ng/g dw	10/24/2004	10/24/2004	3	1.01	1.01	1.3
RB5	SWAMP_RB5L	Chlordane, trans-	ng/g dw	9/24/2004	10/24/2004	5	1.89	1.89	8.18
RB5	SWAMP_RB5L	Chlorpyrifos	ng/g dw	9/24/2004	10/24/2004	8	2.12	2.12	19.31
RB5	SWAMP_RB5L	Cyfluthrin, total	ng/g dw	9/24/2004	11/7/2004	15	0.9	0.9	179.9
RB5	SWAMP_RB5L	Cyhalothrin, lambda, total	ng/g dw	9/24/2004	11/7/2004	16	1.1	1.1	18.2
RB5	SWAMP_RB5L	Cypermethrin, total	ng/g dw	9/24/2004	11/7/2004	14	1.3	1.3	295.8
RB5	SWAMP_RB5L	DDD(p,p')	ng/g dw	9/25/2004	10/24/2004	3	1.17	1.17	5.11
RB5	SWAMP_RB5L	DDE(p,p')	ng/g dw	9/24/2004	10/24/2004	8	1.15	1.15	3.29
RB5	SWAMP_RB5L	DDT(p,p')	ng/g dw	9/24/2004	11/7/2004	20	1.3	1.3	30.68
RB5	SWAMP_RB5L	Deltamethrin	ng/g dw	9/24/2004	11/7/2004	12	1.8	1.8	48.04
RB5	SWAMP_RB5L	Dieldrin	ng/g dw	9/24/2004	11/7/2004	11	1.01	1.01	2.06
RB5	SWAMP_RB5L	Endrin	ng/g dw	10/24/2004	10/24/2004	2	1.06	1.06	1.1
RB5	SWAMP_RB5L	Endrin Aldehyde	ng/g dw	9/24/2004	9/24/2004	1	2.54	2.54	2.54
RB5	SWAMP_RB5L	Esfenvalerate/Fenvalerate, total	ng/g dw	9/24/2004	10/24/2004	4	2.5	2.5	5.8
RB5	SWAMP_RB5L	HCH, alpha	ng/g dw	9/25/2004	9/25/2004	1	2.38	2.38	2.38
RB5	SWAMP_RB5L	Methoxychlor	ng/g dw	9/24/2004	10/24/2004	3	1.65	1.65	7.63
RB5	SWAMP_RB5L	Permethrin-1	ng/g dw	9/24/2004	11/7/2004	20	0.28	0.28	231.5
RB5	SWAMP_RB5L	Permethrin-2	ng/g dw	9/24/2004	11/7/2004	20	0.31	0.31	106.5
RB5	SWAMP_RB5L	Total Organic Carbon	%	9/24/2004	11/7/2004	23	0.9	0.9	9.15
RB5	SWAMP_RB5S	Bifenthrin	ng/g dw	4/8/2003	9/19/2005	6	0.401	0.401	2.44
RB5	SWAMP_RB5S	Chlordane, cis-	ng/g dw	6/15/2005	6/15/2005	1	3.25	3.25	3.25
RB5	SWAMP_RB5S	Chlordane, trans-	ng/g dw	6/15/2005	6/15/2005	1	1.9	1.9	1.9
RB5	SWAMP_RB5S	Chlorpyrifos	ng/g dw	4/8/2003	6/15/2005	5	1.67	1.67	10.7
RB5	SWAMP_RB5S	Clay <0.005 mm	%	10/9/2001	9/19/2005	55	2.96	2.96	55
RB5	SWAMP_RB5S	Cyhalothrin, lambda, total	ng/g dw	4/8/2003	9/19/2005	6	0.432	0.432	2.19
RB5	SWAMP_RB5S	Cypermethrin, total	ng/g dw	6/15/2005	6/15/2005	1	3.88	3.88	3.88
RB5	SWAMP_RB5S	DDD(p,p')	ng/g dw	3/30/2005	6/15/2005	2	4.12	4.12	26.5

Data Source	ProjectID	Analyte Name	Unit	Start Date	End Date	Count of Results	Min of Result (MDL/2)	Min of Result	Max of Result
RB5	SWAMP_RB5S	DDE(o,p')	ng/g dw	6/15/2005	6/15/2005	1	6.21	6.21	6.21
RB5	SWAMP_RB5S	DDE(p,p')	ng/g dw	3/30/2005	6/15/2005	2	35.7	35.7	134
RB5	SWAMP_RB5S	DDMU(p,p')	ng/g dw	6/15/2005	6/15/2005	1	1.88	1.88	1.88
RB5	SWAMP_RB5S	DDT(o,p')	ng/g dw	3/30/2005	6/15/2005	2	4.22	4.22	17.7
RB5	SWAMP_RB5S	DDT(p,p')	ng/g dw	3/30/2005	3/30/2005	1	13.7	13.7	13.7
RB5	SWAMP_RB5S	Diazinon	ng/g dw	4/8/2003	4/9/2003	4	1.23	1.23	2.26
RB5	SWAMP_RB5S	Endosulfan II	ng/g dw	6/15/2005	6/15/2005	1	1.26	1.26	1.26
RB5	SWAMP_RB5S	Endrin	ng/g dw	6/15/2005	6/15/2005	1	1.29	1.29	1.29
RB5	SWAMP_RB5S	Esfenvalerate/Fenvalerate, total	ng/g dw	4/8/2003	4/9/2003	5	0.985	0.985	11.4
RB5	SWAMP_RB5S	Gravel 4.75 to <75 mm	%	10/9/2001	9/19/2005	16	0.14	0.14	33.51
RB5	SWAMP_RB5S	Moisture	%	4/8/2003	9/19/2005	21	21.5	21.5	56.4
RB5	SWAMP_RB5S	Nonachlor, trans-	ng/g dw	6/15/2005	6/15/2005	1	2.08	2.08	2.08
RB5	SWAMP_RB5S	Permethrin, total	ng/g dw	4/8/2003	4/9/2003	4	2.27	2.27	50.6
RB5	SWAMP_RB5S	Sand 0.075 to <4.75 mm	%	10/9/2001	9/19/2005	152	0.01	0.01	84.02
RB5	SWAMP_RB5S	Silt 0.005 to <0.075 mm	%	10/9/2001	9/19/2005	55	0.28	0.28	53.96
RB5	SWAMP_RB5S	Total Organic Carbon	%	10/9/2001	9/19/2005	55	0.12	0.12	4.3
RB5	SWAMP_RB5S	Toxaphene	ng/g dw	6/15/2005	6/15/2005	1	678	678	678
RB5	SWAMP_SB	Fine <0.0625 mm	%	11/16/2006	1/2/2007	10	39.17	39.17	96.83
RB5	SWAMP_SB	Total Organic Carbon	%	11/16/2006	1/2/2007	10	2.35	2.35	10.54
RB5	SWAMP_RB5S	Chlorpyrifos	µg/L	5/29/2002	6/15/2005	2	0.0561	0.0561	0.122
RB5	SWAMP_RB5S	Diazinon	µg/L	5/29/2002	9/19/2002	2	0.0365	0.0365	0.037

APPENDIX II

TABULATED TOXICITY RESULTS

Table 6. Acute toxicity to larval fathead minnow (*P. promelas*; 96 h survival) and TIE results; Irrigated Lands Regulatory Program (Ag Waiver), 2003-2008.

Map ID	Sampling Site	Sampling Date	% Survival	Survival, % Control	Follow-Up Testing
37	Kellogg Creek @ Hwy 4	21-Jun-05	20	20.5	
41	Littlejohns Creek @ Jack Tone Rd	16-Feb-05	70	70	
42	Livingston Canal at Cressey Way	10-Jul-07	65	65	
42	Livingston Canal at Cressey Way	24-Jul-07	20	20	
43	Lone Tree Creek @ Bernnan Rd	27-Feb-06	0	0	NOEC=25%, EC50=36.6%, 2.73 TUa. TIE 3/8/06: toxicity due to ammonia. Resampled, toxicity not persistent.
44	Lone Tree Creek @ Jack Tone Rd	16-Feb-05	0	0	
44	Lone Tree Creek @ Jack Tone Rd	23-Jan-08	75	75	Sample had strong odor of manure, contained high levels of suspended solids and had extremely high oxygen demand. Resampled on 01/30/08; toxicity was not persistent.
61	Sand Creek @ Hwy 4 Bypass	16-May-06	60	60	Resampled, toxicity not persistent.

Table 7. Toxicity to green algae (*S. capricornutum*; 96 h growth) and TIE results; Irrigated Lands Regulatory Program (Ag Waiver), 2003-2008.

Map ID	Sampling Site	Sampling Date	Final Cell Count	Growth, % Control	Follow-Up Testing
7	Calaveras River at Pezzi Rd	15-Feb-05	1822250	67.9	
7	Calaveras River at Pezzi Rd	17-Feb-05	1742425	53.7	
17	Delta Drain- Terminous Tract off Guard Rd	27-Feb-06	212500	13.77	Control failure CV>20% 2/28/06 test. Re-test outside hold time. Toxicity persistent TIE 3/10/06: tox due to non-polar organic/cationic compounds. Resampled, toxicity not persistent.
30	Duck Creek @ Hwy 4	28-Feb-07	793000	77.1	Resampled on 3/6/07
31	French Camp Slough @ Airport Way	16-Feb-05	1410000	78.3	
32	Grant Line Canal @ Clifton Court Rd	23-Jan-08	283081	19	TIE 2/1/08: cationic metals and non-polar organics were the probable cause of toxicity; Resampled on 01/30/08; toxicity not persistent.
33	Grant Line Canal nr Calpack Rd	16-Feb-05	129000	7.17	
33	Grant Line Canal nr Calpack Rd	11-Apr-07	378000	44	TIE: toxicity likely due to two types of contaminants, one organic and one cationic.
33	Grant Line Canal nr Calpack Rd	10-Jul-07	755273	79.18	Resampled
33	Grant Line Canal nr Calpack Rd	23-Jan-08	22561	1.51	TIE 2/1/08: cationic metals as probable cause of toxicity. Resampled on 01/30/08; toxicity was persistent.
33	Grant Line Canal nr Calpack Rd	30-Jan-08	110486	24.96	Follow-up resample due to *S. capricornutum* toxicity on 01/23/08; toxicity was persistent.
34	Hatch Drain @ Tuolumne Rd	24-Jan-08	833429	74.41	Resampled on 1/30/08; toxicity was not persistent
37	Kellogg Creek @ Hwy 4	16-Aug-05	92000	44.2	
41	Littlejohns Cr. @ Jack Tone Rd	24-Aug-04	910500	55.3	
41	Littlejohns Cr. @ Jack Tone Rd	10-Jul-07	628270	70.69	Resampled
43	Lone Tree Creek @ Bernnan Rd	27-Feb-06	1286750	73.63	Control failure CV>20% 2/28/06 test. Re-test outside hold time.
43	Lone Tree Cr. @ Bernnan Rd	15-Mar-06	680250	56.02	Resampled, toxicity marginally persistent.
44	Lone Tree Cr. @ Jack Tone Rd	16-Feb-05	1380000	76.7	
44	Lone Tree Cr. @ Jack Tone Rd	15-Mar-06	753750	56.68	Resampled, toxicity not persistent.
44	Lone Tree Cr. @ Jack Tone Rd	28-Feb-07	353000	30.5	TIE indicates organic and or cationic contaminants w. additive

Map ID	Sampling Site	Sampling Date	Final Cell Count	Growth, % Control	Follow-Up Testing
					toxicity
44	Lone Tree Cr. @ Jack Tone Rd	23-Jan-08	742247	61.28	Strong odor of manure, high levels of suspended solids and extremely high oxygen demand. Resampled on 01/30/08; tox. was not persistent.
46	Lower Lateral 2 at Grayson Road	05-Sep-07	897000	75.06	
50	Mokelumne River @ Bruella Rd	24-Aug-04	835000	50.7	
50	Mokelumne River @ Bruella Rd	10-Jul-07	507779	57.13	Resampled
52	Mormon Slough @ Jack Tone Rd	10-Jul-07	543601	61.16	Resampled
59	Roberts Island Drain @ Holt Rd	10-Jul-07	289594	40.07	Resampled
59	Roberts Island Drain @ Holt Rd	23-Jan-08	16283	1.34	TIE 2/1/08: non-polar organics were probable toxicants; resampled on 01/30/08; toxicity not persistent.
64	Sycamore Slough at Cotta Road (nr Guard Rd)	08-Aug-07	352750	31.29	TIE: cationic metal(s).
65	Terminous Tract Drain @ Hwy12	16-Feb-05	334000	18.6	
65	Terminous Tract Drain @ Hwy12	23-Jan-08	103973	8.32	TIE 2/1/08. Toxicity not persistent. Resampled on 01/30/08; toxicity was not persistent.
68	Ulatis Creek at Brown Road	08-Aug-07	886500	78.63	
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	11-Feb-07	475000	53	
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	21-Feb-07	926000	83	Resampled on 2/11/07.
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	28-Feb-07	55300	4.8	NOEC <6.25%, EC50=5.95%, TUa=16.8; TIE indicates organic and cationic contaminants that are additive in toxicity
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	07-Mar-07	504000	57	Resampled due to toxicity in sample from 2/28/07; toxicity persistent
78	Westport Drain @ Vivian Rd	15-May-07	778069	73.31	
80	Winters Canal at Road 86A	26-Jan-05	225600	32.4	
80	Winters Canal at Road 86A	28-Jan-05	210125	8.7	
80	Winters Canal at Road 86A	16-Feb-05	388750	10.5	TIE: herbicides
80	Winters Canal at Road 86A	18-Feb-05	499725	12	

Table 8. Sediment toxicity to H. azteca (10-d survival/growth); Irrigated Lands Regulatory Program (Ag Waiver), 2003-2008.

Map ID	Sampling Site	Sampling Date	Endpoint	Result	% Control	Follow-Up
9	Del Puerto Creek at Frank Cox Rd	07-Dec-05	Survival (%)	1.25	1	
10	Del Puerto Creek at intersection Hwy 33 and Mulberry Rd	07-Dec-05	Survival (%)	0	0	
11	Del Puerto Creek at Loquat #1	07-Dec-05	Survival (%)	0	0	
12	Del Puerto Creek at Loquat #2	07-Dec-05	Survival (%)	1.25	1	
14	Del Puerto Creek at Vineyard	07-Dec-05	Survival (%)	0	0	
16	Delta Drain- Terminous Tract off Glasscock Rd	17-May-05	Growth (weight)	0.13	66	
17	Delta Drain- Terminous Tract off Guard Rd	17-May-05	Growth (weight)	0.11	55.3	
31	French Camp Slough @ Airport Way	17-May-05	Growth (weight)	0.16	79.8	
31	French Camp Slough @ Airport Way	09-Aug-07	Survival (%)	32	34	Resampled
32	Grant Line Canal @ Clifton Court Rd	17-May-05	Growth (weight)	0.14	70.7	
32	Grant Line Canal @ Clifton Court Rd	20-Sep-05	Survival (%)	8.8	9	
33	Grant Line Canal near Calpack Rd	17-May-05	Growth (weight)	0.05	25	
33	Grant Line Canal near Calpack Rd	17-May-05	Survival (%)	43.8	44.3	
33	Grant Line Canal near Calpack Rd	19-Jul-05	Survival (%)	68.8	70.6	
33	Grant Line Canal near Calpack Rd	27-Apr-06	Survival (%)	20	20.5	
33	Grant Line Canal near Calpack Rd	07-Mar-07	Survival (%)	12.5	12.8	
33	Grant Line Canal near Calpack Rd	29-Mar-07	Survival (%)	5	5.26	
34	Hatch Drain @ Tuolumne Rd	16-Aug-07	Survival (%)	0	0	Resampled
34	Hatch Drain @ Tuolumne Rd	11-Sep-07	Survival (%)	0	0	Resampled; toxicity persistent.
37	Kellogg Creek @ Hwy 4	17-May-05	Growth (weight)	0.14	71.7	
37	Kellogg Creek @ Hwy 4	19-Jul-05	Growth (weight)	0	0	
37	Kellogg Creek @ Hwy 4	19-Jul-05	Survival (%)	0	0	
37	Kellogg Creek @ Hwy 4	20-Sep-05	Survival (%)	57.5	59	
38	Kellogg Creek along Hoffman Ln	09-Aug-07	Survival (%)	0	0	Resampled
38	Kellogg Creek along Hoffman Ln	31-Aug-07	Survival (%)	0	0	
41	Littlejohns Creek @ Jack Tone Rd	17-May-05	Growth (weight)	0.16	78.1	
41	Littlejohns Creek @ Jack Tone Rd	06-Mar-07	Survival (%)	78.8	79.8	

Map ID	Sampling Site	Sampling Date	Endpoint	Result	% Control	Follow-Up
44	Lone Tree Creek @ Jack Tone Rd	23-Sep-04	Growth (weight)	0.09	75.1	
44	Lone Tree Creek @ Jack Tone Rd	17-May-05	Growth (weight)	0.11	54.2	
47	Marsh Creek @ Balfour Ave	17-May-05	Growth (weight)	0	0	
47	Marsh Creek @ Balfour Ave	17-May-05	Survival (%)	0	0	
47	Marsh Creek @ Balfour Ave	19-Jul-05	Growth (weight)	0	0	
47	Marsh Creek @ Balfour Ave	19-Jul-05	Survival (%)	0	0	
47	Marsh Creek @ Balfour Ave	20-Sep-05	Survival (%)	0	0	
47	Marsh Creek @ Balfour Ave	27-Apr-06	Survival (%)	57.5	57.5	
48	Marsh Creek @ Concord Ave	06-Mar-07	Survival (%)	50	51.3	
55	Pixley Slough at Eightmile Rd	12-Apr-05	Growth (weight)	0.06	70	
59	Roberts Island Drain @ Holt Rd	15-Aug-06	Survival (%)	70	74	Resampled; toxicity persistent.
59	Roberts Island Drain @ Holt Rd	19-Sep-06	Survival (%)	11.25	12	Resampled; toxicity persistent.
60	Roberts Island Drain along House Rd	06-Mar-07	Survival (%)	5	5.13	
60	Roberts Island Drain along House Rd	29-Mar-07	Survival (%)	17.5	18.4	
61	Sand Creek @ Hwy 4 Bypass	15-Aug-06	Survival (%)	0	0	Resampled; toxicity persistent.
61	Sand Creek @ Hwy 4 Bypass	19-Sep-06	Survival (%)	0	0	Resampled; toxicity persistent.
61	Sand Creek @ Hwy 4 Bypass	06-Mar-07	Survival (%)	3.75	3.85	
61	Sand Creek @ Hwy 4 Bypass	29-Mar-07	Survival (%)	2.5	2.6	
61	Sand Creek @ Hwy 4 Bypass	09-Aug-07	Survival (%)	0	0	Resampled
61	Sand Creek @ Hwy 4 Bypass	31-Aug-07	Survival (%)	0	0	
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	09-Aug-07	Survival (%)	57	61	Resampled

Table 9. State Water Ambient Monitoring Program (SWAMP). Samples showing significant toxicity in either acute 96-h toxicity tests with fathead minnow larvae (FHM) or acute 48-h toxicity tests with *C. dubia*; October 23, 2000-November 21, 2002. Results of both species are presented for comparison.

Map ID	Station Code	Sampling Date	Station Name (# Samples Tested)	Fathead Minnow 96-h survival (%)	Significance (FHM)	*C. dubia* 48-h survival (%)	Significant (*C. dubia*)
FHM Toxicity							
68	STC504	6/28/2001	SJR @ Crows Landing (18)	75	SG	100	NS
63	SAC001	1/29/2002	Cosumnes River at Twin Cities Road (8)	55	SL	100	NS
69	STC513	1/30/2002	Tuolumne River at Shiloh Fishing Access (12)	75	SG	100	NS
79	SJC001	2/20/2001	New Jerusalem Tile Drain (14)	70	SG	90	NS
***C. dubia* Toxicity**							
A2	CAL005	9/3/2002	Mokelumne River @ Camanche Rec. S. Shore (1)	95	NS	70	SL
A13	SJC512	9/3/2002	Mokelumne River @ Van Assen Co. Park (1)	100	NS	70	SG
79	SJC001	12/26/2001	New Jerusalem Tile Drain (14)	95	NS	70	SG

SG: significantly different from control; greater than 80% survival compared to the control.

SL: significantly different from control; less than 80% survival compared to the control.

NS: not significantly different from control.

Table 10. State Water Ambient Monitoring Program (SWAMP): Acute toxicity to larval fathead minnow (*P. promelas*), *C. dubia* (48 h), and 96-h growth of green algae (*S. capricornutum*); in 2004-2005; NT=not toxic, NA=not tested.

			TOXIC SAMPLES			
Map ID	Site Code	Site Name	#Samples Collected	*P. promelas* (96 h)	*C. dubia* (48 h)	Algae (96 h)
65	SJC503	Lone Tree Ck @ Austin Rd.	11	NT	1/26/05	NA
A11	SJC504	French Camp Sl. @ Airport Way	11	2/23/05 4/26/05 6/28/05	NT	NA
63	SAC001	Cosumnes River @ Twin Cities Rd.	9	4/26/05	NT	NA
A12	SJC507	Pixely Sl. @ Davis Rd.	10	NT	NT	NT
A15	SJC515	Bear Ck. @ Lower Sacramento Rd.	9	4/26/05	NT	
67	STC501	TID 5 Harding Drain	10	NT	NT	NT
A7	MER576	Turner Slough @ Fourth Ave.	10	NT	NT	NA
	MER007 (no coord.)	Bear Ck. @ Bert Crane Rd.	10	1/27/05 2/24/05 5/26/05	NT	NA
76	STC019	Orestimba Ck. @ River Rd.	11	NT	5/18/05	NA
A19	STC030	Grayson Rd. Drain @ Grayson	7	NT	3/22/05	NA
A20	STC040	Ingram Ck. @ River Rd.	11	NT		NA
A21	STC042	Hopital Ck. @ River Rd.	9	NT	3/23/05 4/19/05 8/16/05	NA
A23	STC515	Salado Ck. @ Hwy 33	11	NT	NT	NA
	STC522 (no coord.)	CCID Main @ J.T. Crow Rd.	11	NT	NT	NA
A24	STC523	Del Puerto Ck. @ Hwy 33	11	NT	12/21/04	12/21/04 2/15/05 3/23/05

			TOXIC SAMPLES			
Map ID	Site Code	Site Name	#Samples Collected	*P. promelas* (96 h)	*C. dubia* (48 h)	Algae (96 h)
						6/21/05
	STC525 (no coord.)	Del Puerto Creek @ Del Puerto Rd mi 3.9	9	NA	NA	4/19/05 5/17/05 6/21/05 7/19/05 8/16/05 9/20/05
	STC527 (no coord.)	Del Puerto Ck. @ Deer Creek camp	11	NA	NA	12/21/04 5/17/05 8/16/05 9/20/05
A25	STC531	Drain next to SJR @ Maze	9	NT	2/16/05	NA
79	SJC001	New Jerusalem Tile Drain	10	NT		NA
A16	SJC516	Unnamed Supply channel @ Howard Rd.	5	NT	3/23/05	4/19/05
A17	SJC517	Mid Roberts Island Drain @ Woodsbro	5	NT	3/23/05	NT

Table 11. State Water Ambient Monitoring Program (SWAMP): 7-day acute and chronic toxicity to fathead minnow (FHM, *P. promelas*) and *C. dubia* (CD); all samples listed are significantly different from controls (p<0.05) in 2004-05; NT=not toxic, NA=not tested.

				TOXIC SAMPLES			
Map ID	Site Code	Site Name	#Samples Collected (FHM/CD)	*P. promelas* (7 d survival)	*P. promelas* (7d growth)	*C. dubia* (7 d surv)	*C. dubia* (7d reproduction)
68	STC(SJR)504	SJR @ Crows Landing	7/0	11/18/04 12/22/04 5/19/05 9/22/05	NT NT NT 9/22/05	NA	NA
A10	SJC/SJR507	SJR @ Patterson	7/11	11/18/04 NT NT 5/19/05 NT	NT NT NT NT 9/22/05	NT NT NT NT NT	NT 2/17/05 4/21/05 5/19/05 9/22/05
A13/77	STC/SJR512	SJR @ Hills Ferry	7/0	NT NT 5/19/05 9/22/05	12/22/04 2/17/05 NT 9/22/05	NA	NA
71	MER522	SJR @ Lander Ave	7/0	NT 2/17/05 3/24/05 5/19/05	1/20/05 2/17/05 NT 5/19/05	NA	NA
A9	SJR/SJC501	SJR @ Airport Way	8/11	NT NT NT NT NT 5/19/05 NT	12/22/04 1/20/05 NT NT NA 5/19/95 9/22/05	NT NT NT NT NT NT NT	12/22/04 NT 2/17/05 3/24/05 4/21/05 5/19/05 9/22/05

Map ID	Site Code	Site Name	#Samples Collected (FHM/CD)	TOXIC SAMPLES			
				P. promelas (7 d survival)	*P. promelas* (7d growth)	*C. dubia* (7 d surv)	*C. dubia* (7d reproduction)
	STC013/STC513 (no coordinates)	Tuolumne River @ Shiloh	9/0	NT 12/29/04 2/24/05 3/29/05 5/26/05 6/30/05 NT 9/29/05	11/23/04 NT 2/24/05 3/29/05 5/26/05 6/30/05 8/25/05 NT	NA	NA
	STC014 (no coordinates)	Stanislaus River @ Caswell	9/0	11/22/04 4/26/05 8/23/05	11/22/04 4/26/05 8/23/05	NA	NA
66	MER546	Merced River @ River Rd.	9/0	NT 3/29/05 5/26/05 8/25/05	11/23/04 NT 5/26/05 8/25/05	NA	NA
A2	SAC002	Mokelumne River @ New Hope Rd.	8/0	NT 2/23/05 5/24/05 NT	11/23/04 2/23/05 NT 8/23/05	NA	NA

Table 12. State Water Ambient Monitoring Program (SWAMP): Acute and chronic toxicity to *C. dubia*; all samples listed are significantly different from controls (p<0.05); San Francisco Bay Area.

Map ID	Station	Sample Date	Endpoint	Unit	Mean	SD	Signif.
20	204SLE030	19/Sep/2001	Young/female	Num/Rep	16	11	SL
34	207KIR020	21/Jan/2003	Survival	%	0	0	SL
35	207KIR115	21/Jan/2003	Young/female	Num/Rep	8	3	SL
35	207KIR115	21/Jan/2003	Survival	%	60	51.6	SL
36	207MTD010	21/Jan/2003	Young/female	Num/Rep	14	5	SL
37	207MTD100	21/Jan/2003	Young/female	Num/Rep	15	2	SL
26	205PER070	23/Jan/2003	Survival	%	70	48.3	SL
28	205STE060	23/Jan/2003	Survival	%	70	48.3	SL
14	204AVJ020	11/Jan/2005	Young/female	Num/Rep	18.8	2.74	SL
14	204AVJ020	11/Jan/2005	Young/female	Num/Rep	20.3	2.36	SL
13	204AMO070	14/Jun/2005	Young/female	Num/Rep	22.5	12.41	SL

Table 13. State Water Ambient Monitoring Program (SWAMP): Acute and chronic toxicity to *P. promelas*; all samples listed are significantly different from controls (p<0.05); San Francisco Bay Area.

Map ID	Station	Sample Date	Endpoint	Unit	Mean	SD	Signif.
28	205STE060	11/Apr/2002	Survival	%	73	5	SL
36	207MTD010	21/Apr/2003	Growth (wt/surv indiv)	mg/ind	0.42	0.091	SL
21	204SMA020	22/Apr/2003	Survival	%	62	19.73	SL
23	204SMA080	22/Apr/2003	Survival	%	57.5	20.62	SL
24	204SMA110	22/Apr/2003	Survival	%	64.5	18.65	SL
23	204SMA080	03/Jun/2003	Survival	%	60	8.16	SL
14	204AVJ020	11/Jan/2005	Biomass (wt/orig indiv)	mg/ind	0.53	0.05	SL
9	203TEM090	14/Jun/2005	Biomass (wt/orig indiv)	mg/ind	0.68	0.13	SL

SL: significantly different from control; less than 80% survival compared to the control.

Table 14. State Water Ambient Monitoring Program (SWAMP): Acute and chronic toxicity to S. capricornutum; all samples listed are significantly different from controls (p<0.05); San Francisco Bay Area.

Map ID	Station	Sample Date	Unit	Mean	SD	Signif.
12	204ALP110	18/Sep/2001	cells/ml	4118000	163707	SL
29	206SPA020	26/Sep/2001	cells/ml	2673000	251064	SL
10	204ALP010	08/Apr/2002	cells/ml	2738000	108934	SL
11	204ALP100	08/Apr/2002	cells/ml	3523000	264512	SL
12	204ALP110	08/Apr/2002	cells/ml	2463000	122610	SL
29	206SPA020	09/Apr/2002	cells/ml	2783000	173109	SL
30	206SPA070	09/Apr/2002	cells/ml	2378000	182209	SL
25	205PER010	10/Apr/2002	cells/ml	2948000	71181	SL
25	205PER010	10/Apr/2002	cells/ml	2518000	119443	SL
26	205PER070	11/Apr/2002	cells/ml	2583000	262996	SL
27	205STE020	11/Apr/2002	cells/ml	1228000	101980	SL
25	205PER010	17/Jun/2002	cells/ml	3040000	76594	SL
26	205PER070	17/Jun/2002	cells/ml	3140000	96609	SL
35	207KIR115	21/Jan/2003	cells/ml	1125000	90000	SL
36	207MTD010	21/Jan/2003	cells/ml	3655000	186458	SL
25	205PER010	23/Jan/2003	cells/ml	2745000	365650	SL
26	205PER070	23/Jan/2003	cells/ml	3420000	400999	SL
27	205STE020	23/Jan/2003	cells/ml	4715000	444934	SL
34	207KIR020	21/Apr/2003	cells/ml	3801500	89195	SL
34	207KIR020	21/Apr/2003	cells/ml	3988000	153188	SL
35	207KIR115	21/Apr/2003	cells/ml	1683000	124766	SL
23	204SMA080	22/Apr/2003	cells/ml	3988000	376298	SL
34	207KIR020	02/Jun/2003	cells/ml	3178000	781537	SL
24	204SMA110	03/Jun/2003	cells/ml	4428000	453137	SL
14	204AVJ020	11/Jan/2005	cells/ml	2927325	224828	SL
6	203COD020	12/Apr/2005	cells/ml	2627500	614132	SL
7	203LOB020	16/Feb/2006	cells/ml	674250	54854	SL

SL: significantly different from control; less than 80% survival compared to the control.

Table 15. State Water Ambient Monitoring Program (SWAMP): Acute and chronic sediment toxicity to H. azteca; all samples listed are significantly different from controls (p<0.05); San Francisco Bay Area.

Map ID	Station	Sampling Date	Endpoint	Unit	Mean	SD	Sign.
19	204SLE030	19/Sep/2001	Survival	%	0	0	SL
76	541STC019	09/Oct/2001	Growth (wt/surv indiv)	mg/ind	0.13	0.04	SL
76	541STC019	09/Oct/2001	Survival	%	65	26.7	SL
78	541STC516	09/Oct/2001	Survival	%	0	0	SL
25	205PER010	17/Jun/2002	Survival	%	73	18.3	SL
25	205PER010	17/Jun/2002	Growth (wt/surv indiv)	mg/ind	0.222	0.087	SL
25	205PER010	17/Jun/2002	Growth (wt/surv indiv)	mg/ind	0.222	0.037	SL
18	205STE020	17/Jun/2002	Growth (wt/surv indiv)	mg/ind	0.219	0.07	SL
33	206WIL020	17/Jun/2002	Growth (wt/surv indiv)	mg/ind	0.157	0.049	SL
34	207KIR020	21/Apr/2003	Survival	%	0	0	SL
34	207KIR020	21/Apr/2003	Survival	%	0	0	SL
36	207MTD010	21/Apr/2003	Growth (wt/surv indiv)	mg/ind	0.338	0.206	SL
36	207MTD010	21/Apr/2003	Survival	%	66	0.31	SL
21	204SMA020	22/Apr/2003	Survival	%	18	0.17	SL
21	204SMA020	22/Apr/2003	Growth (wt/surv indiv)	mg/ind	0.15	0.12	SL
45	519LSAC30	24/Sep/2004	Growth (wt/surv indiv)	mg/ind	0.038	0.006	SL
45	519LSAC30	24/Sep/2004	Survival	%	75	8	SL
51	519LSAC37	24/Sep/2004	Survival	%	0	0	SL
53	519LSAC39	24/Sep/2004	Survival	%	0	0	SL
54	519LSAC40	24/Sep/2004	Growth (wt/surv indiv)	mg/ind	0.038	0.023	SL
54	519LSAC40	24/Sep/2004	Survival	%	63	18	SL
55	519LSAC41	24/Sep/2004	Survival	%	8	12	SL
48	519LSAC34	25/Sep/2004	Survival	%	1	4	SL
56	519LSAC42	25/Sep/2004	Survival	%	0	0	SL
57	519LSAC43	25/Sep/2004	Survival	%	0	0	SL
57	519LSAC43	25/Sep/2004	Survival	%	0	0	SL
59	519LSAC45	25/Sep/2004	Survival	%	1	4	SL
46	519LSAC31	24/Oct/2004	Survival	%	45	40	SL
46	519LSAC31	24/Oct/2004	Growth (wt/surv indiv)	mg/ind	0.042	0.009	SL
49	519LSAC35	24/Oct/2004	Survival	%	5	11	SL
58	519LSAC44	24/Oct/2004	Growth (wt/surv indiv)	mg/ind	0.039	0.007	SL
58	519LSAC44	24/Oct/2004	Survival	%	66	15	SL

Map ID	Station	Sampling Date	Endpoint	Unit	Mean	SD	Sign.
59	519LSAC45	24/Oct/2004	Survival	%	1	4	SL
61	519LSAC47	24/Oct/2004	Survival	%	3	5	SL
62	519LSAC48	24/Oct/2004	Survival	%	10	11	SL
62	519LSAC48	24/Oct/2004	Survival	%	16	17	SL
46	519LSAC31	07/Nov/2004	Survival	%	10	12	SL
7	203LOB020	11/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.48	0.06	SL
15	204ISL050	11/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.505	0.123	SL
4	203BAX030	12/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.529	0.131	SL
4	203BAX030	12/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.335	0.093	SL
6	203COD020	12/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.398	0.072	SL
8	203STW010	12/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.467	0.038	SL
14	204AVJ020	12/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.404	0.081	SL
18	204SAU030	12/Apr/2005	Growth (wt/surv indiv)	mg/ind	0.398	0.179	SL

SL: significantly different from control; less than 80% survival compared to the control.

Table 16. Sediment Quality Objectives - Phase II, 9/17/2007-10/16/ 2007; San Francisco Estuary Institute, Oakland, CA. Results of 10-d sediment toxicity test using *Hyalella azteca* and *Chironomus dilutus* (SFEI, 2007). Out of 100 samples tested for toxicity, sixteen stations showed significant toxicity in both significant effects tests (T-test & MSD-threshold). Note that only 50 stations were tested for toxicity to *C. dilutus*.

Station Code	Station Name	*C. dilutus* % Survival	*C. dilutes* Growth (mg/ind)	*H. azteca* % Survival	*H. azteca* Growth (mg/ind)
EMP-0005	Whiskey Slough				0.15
EMP-0006	Latham Slough			63	0.15
EMP-0012	Sherman Lake	na	na		0.15
EMP-0022	Latham Slough		1.4		
EMP-0024	New York Slough				0.14
EMP-0026	Old River				0.15
EMP-0049	Indian Slough			60	
EMP-0089	Fishermans Cut				0.1
EMP-0105	Sand Mound Slough		1.3		
EMP-0113	Bacon Canal				0.06
EMP-0149	San Joaquin River	na	na		0.1
EMP-0150	Mildred Island	69		46	
MR05	Mokelumne River N Fork				0.1
P8	San Joaquin River				0.1
SJR01	San Joaquin River @ Stockton Channel				0.11
STC01	San Joaquin River		1.67		0.11

Growth: weight/survival individual.

Table 17. Sacramento River Watershed Program 2006-2007: Water samples from Sacramento River at Freeport (in chronological order) showing significant acute and/or chronic toxicity to *C. dubia* (7-8 d), *S. capricornutum* (4 d) and *P. promelas* (7 d).

Sampling Date	Test species	Toxicity Endpoint	Percent of Control	Significance Level
04/21/2006	*Selenastrum capricornutum*	cells/ml	331.8	SG
07/26/2006	*Ceriodaphnia dubia*	%	20	SL
07/26/2006	*Ceriodaphnia dubia*	Young/female (#)	9	SL
07/31/2006	*Ceriodaphnia dubia*	%	22	SL
08/24/2006	*Ceriodaphnia dubia*	%	25	SL
08/24/2006	*Ceriodaphnia dubia*	Young/female (#)	27	SL
08/30/2006	*Ceriodaphnia dubia*	%	70	SL
08/30/2006	*Ceriodaphnia dubia*	Young/female (#)	41	SL
10/25/2006	*Pimephales promelas*	%	70	SL
10/25/2006	*Pimephales promelas*	mg/ind	75	SL
12/12/2006	*Ceriodaphnia dubia*	%	0	SL
12/12/2006	*Ceriodaphnia dubia*	Young/female (#)	11	SL
12/12/2006	*Pimephales promelas*	%	5	SL
12/12/2006	*Pimephales promelas*	mg/ind	5	SL
04/25/2007	*Ceriodaphnia dubia*	%	30	SL
04/25/2007	*Ceriodaphnia dubia*	Young/female (#)	27	SL
06/28/2007	*Pimephales promelas*	mg/ind	80	SL
07/26/2007	*Pimephales promelas*	mg/ind	75	SL
05/01/2007	*Ceriodaphnia dubia*	Young/female (#)	88	SG

SG: significantly different from control; greater than 80% survival compared to the control.

SL: significantly different from control; less than 80% survival compared to the control.

Table 18. Data sources, test type and test period for ambient toxicity monitoring covered in this review.

Data Source (Agency/City)	Program	Period Covered	Frequency of Testing	Test Species	Endpoints
CVRWQCB	SWAMP	2001-2003	approx. monthly	*P. promelas*	96 hr, survival
CVRWQCB	SWAMP	2001-2003	approx. monthly	*C. dubia*	48 hr, survival
CVRWQCB	SWAMP	2004	approx. monthly	*P. promelas*	7 day, survival/growth
CVRWQCB	SWAMP	2004	approx. monthly	*Ceriodaphnia dubia*	6-7 day, survival/ reproduction
CVRWQCB	SWAMP	2005	approx. monthly	*Selenastrum capricornutum*	growth
CVRWQCB	SWAMP	2005	approx. monthly	*C. dubia*	48 hr / 6-7 day survival/ reproduction
CVRWQCB	SWAMP	2005	approx. monthly	*P. promelas*	96 hr / 7 day, growth/ survival
CVRWQCB	Sacramento River Watershed Program	2000-2007	approx. monthly	*C. dubia, P. promelas, S. capricornutum*	7 or 4 day, survival, reproduction, growth
UCD AEAL[1] / MLJ-LLC	Irrigated Lands Program	2003-2008	varies	*C. dubia*	4 day, survival
UCD AEAL / MLJ-LLC	Irrigated Lands Program	2003-2008	varies	*P. promelas*	4 day, survival
UCD AEAL / MLJ-LLC	Irrigated Lands Program	2003-2008	varies	*S. capricornutum*	4 day, survival
UCD Aquatic Toxicology Laboratory	Irrigated Lands Program	Mar. 24- Oct. 7, 2003	approx. every 3 weeks	*C. dubia, P. promelas*	4 day, survival
San Francisco Estuary Institute	Sediment Quality Objectives, Phase II	Sep.17-Oct.16, 2007	one time only	*H. azteca Chironomus dilutus*	10 day, survival/growth
UCD ATL[2]	Interagency Ecological Program: POD	2005-2008	biweekly	*H. azteca*	10 day, survival/growth
UCD ATL	Interagency Ecological Program: POD	2005-2008	biweekly, April-June	Striped bass (*Morone saxitalis*)	7 day, survival
UCD ATL	Interagency Ecological Program: POD	2005-2008	biweekly, April-June	Delta smelt (*Hypomesus transpacificus*)	7 day, survival

[1]Aquatic Ecosystems Analysis Laboratory, UC Davis
[2]Aquatic Toxicology Laboratory, UC Davis

Table 19. Data sources, test type and test period for NPDES testing covered in this review.

Data Source (Agency/City)	Program	Period Covered	Frequency of Testing	Test Species	Endpoints
Rio Vista WWTP	NPDES	2007	April/May	*C. dubia*	7 day, survival / reproduction
Rio Vista WWTP	NPDES	2007	April/May	*P. promelas*	7 day survival / growth
Rio Vista WWTP	NPDES	2007	April/May	*S. capricornutum*	4 day, growth
Stockton WWTP	NPDES	2000-2004 & 2006-2008	quarterly and/or more	*C. dubia*	7 day, survival / reproduction
Stockton WWTP	NPDES	2000-2004 & 2006-2008	quarterly and/or more	*P. promelas*	7 day survival / growth
Stockton WWTP	NPDES	2000-2004 & 2006-2008	quarterly and/or more	*S. capricornutum*	4 day, growth
Brentwood WWTP	NPDES	2003-2006	quarterly	*C. dubia*	7 day, survival / reproduction
Brentwood WWTP	NPDES	2003-2006	quarterly	*P. promelas*	4 day, survival or 7 day, survival / growth
Brentwood WWTP	NPDES	2003-2006	quarterly	*S. capricornutum*	4 day, growth
Discovery Bay WWTP	NPDES	12/2006-2007	monthly	*C. dubia*	7 day, survival / reproduction
Discovery Bay WWTP	NPDES	12/2006-2007	monthly	*P. promelas*	4 day, survival or 7 day, survival / growth
Discovery Bay WWTP	NPDES	12/2006-2007	monthly	*S. capricornutum*	4 day, growth
Merced WWTP	NPDES	2007	varies	*C. dubia*	Chronic, survival / reproduction
Merced WWTP	NPDES	2007	varies	*P. promelas*	Chronic, survival / growth
Merced WWTP	NPDES	2007	varies	*S. capricornutum*	Chronic, growth
Tracy WWTP	NPDES	2000-2007	quarterly	*C. dubia*	6 day LC50 / reproduction
Tracy WWTP	NPDES	2000-2007	quarterly	*P. promelas*	7 day LC50
Tracy WWTP	NPDES	2000-2007	quarterly	*S. capricornutum*	96 hr LC50
Turlock WWTP	NPDES	2000-2007	quarterly	*C. dubia*	6 day LC50
Turlock WWTP	NPDES	2000-2007	quarterly	*P. promelas*	7 day LC50
Turlock WWTP	NPDES	2000-2007	quarterly	*S. capricornutum*	NR

Table 20. Irrigated Lands Regulatory Program (Ag Waiver), list of sampling sites and map ID numbers (see Figure 1).

Station Code	Station Name	Latitude	Longitude	Map ID
544EMARBR	8 Mile & Rio Blanco Rds	38.050500	-121.417500	1
531XNSJ32	Bear Creek at Alpine Rd	38.074020	-121.210930	2
531XNSJ34	Bear Creek at Harney Ln.	38.101712	-121.176429	3
544BSABRD	Beaver Slough @ Blossom Rd	38.204210	-121.447100	4
531XCRABI	Calaveras River @ Belotta Intake	37.961250	-121.204400	5
531XNSJ04	Calaveras River at Clements Rd.	38.045627	-121.076605	6
531XNSJ31	Calaveras River at Pezzi Rd	38.045360	-121.199820	7
511CAHWRD	Creek @ Hawkins Rd.	38.358650	-121.848500	8
541XSED44	Del Puerto Creek at Frank Cox Road	37.531300	-121.138050	9
541XNSJ17	Del Puerto Creek at intersection Hwy 33 and Mulberry Rd	37.514210	-121.158750	10
541XSED41	Del Puerto Creek at Loquat #1	37.538560	-121.123890	11
541XSED42	Del Puerto Creek at Loquat #2	37.538760	-121.123630	12
541XSED45	Del Puerto Creek at Rodgers	37.499360	-121.177610	13
541XSED43	Del Puerto Creek at Vineyard	37.521400	-121.148660	14
541XSED46	Del Puerto Creek at Zacharins Road	37.493940	-121.193860	15
544XTTGLR	Delta Drain- Terminous Tract off Glasscock Rd	38.125500	-121.489360	16
544XTTGUR	Delta Drain- Terminous Tract off Guard Rd	38.116700	-121.421100	17
544DABWMR	Drain @ Bowman Road	37.862670	-121.325100	18
511DAMBLV	Drain @ Mace Blvd.	38.511600	-121.695200	19
511DARBRD	Drain @ Robben Rd.	38.416280	-121.786100	20
511DARRMR	Drain @ Robben Rd. & Midway Rd.	38.380110	-121.786300	21
511DAUCWY	Drain @ Ulatis Creek & Hwy 113	38.338380	-121.823300	22
544DRAWLR	Drain @ Wing Levee Rd	37.856590	-121.378000	23
544XSED10	Drain to Brack Dr at Woodbridge Rd	38.152700	-121.498900	24
544XXXD02	Drain to Grant Line Canal off Wing Levee Rd.	37.820500	-121.403500	25
544XSED11	Drain to North Canal along Bonetti Drive	37.864300	-121.520000	26
544XXXD03	Drain to North Canal at South Bonetti Rd.	37.871500	-121.525600	27

Station Code	Station Name	Latitude	Longitude	Map ID
531XSED09	Drain to Pixley Slough at Davis Rd	38.056400	-121.333200	28
544XXXD01	Drain to San Joaquin R. @ South Manthey Rd.	37.823400	-121.298500	29
531XDCAHF	Duck Creek @ Hwy 4	37.949100	-121.181200	30
531SJC504	French Camp Slough @ Airport Way	37.881720	-121.249330	31
544XGLCAA	Grant Line Canal @ Clifton Court Rd	37.841400	-121.528800	32
544XGLCCR	Grant Line Canal near Calpack Rd	37.820500	-121.499900	33
535XHDATR	Hatch Drain @ Tuolumne Rd	37.514870	-121.012210	34
541XSED12	Hospital Creek at Rd. 33	37.612300	-121.259700	35
541STC042	Hospital Creek at River Road	37.610556	-121.228611	36
544XKCHWF	Kellogg Creek @ Hwy 4	37.889240	-121.619010	37
544XKCAHL	Kellogg Creek along Hoffman Ln	37.881880	-121.652210	38
535XSSJ17	Lateral 5 at Paradise Road	37.614530	-121.143800	39
531LJCANR	Little John Creek @ Newcastle Rd	37.876300	-121.210700	40
531XLCAJR	Littlejohns Creek @ Jack Tone Rd	37.889600	-121.146100	41
535XSSJ15	Livingston Canal at Cressey Way	37.478640	-121.406050	42
535XLTABR	Lone Tree Creek @ Bernnan Rd	37.825520	-121.015910	43
531XLTCJR	Lone Tree Creek @ Jack Tone Rd	37.837600	-121.143760	44
531LTCANR	Lone Tree Creek @ Newcastle Rd.	37.862200	-121.210100	45
535XSSJ18	Lower Lateral 2 at Grayson Road	37.565220	-121.138460	46
544XMCABA	Marsh Creek @ Balfour Ave	37.925590	-121.710200	47
544XMCACA	Marsh Creek @ Concord Ave	37.903930	-121.716270	48
544SJC517	Mid Roberts Island Drain at Woodsbro Road	37.941630	-121.369300	49
531XMRABR	Mokelumne River @ Bruella Rd	38.160150	-121.205100	50
531XMRAFH	Mokelumne River @ Fish Hatchery	38.226390	-121.026390	51
544MSAJTR	Mormon Slough @ Jack Tone Rd	37.964700	-121.148800	52
531XNSJ06	Mormon Slough on Jack Tone Rd	37.965046	-121.147934	53
531XNSJ38	Paddy Creek at Jack Tone Rd.	38.117898	-121.149731	54
531XNSJ28	Pixley Slough at Eightmile Rd	38.057650	-121.313503	55
531XNSJ36	Pixley Slough at Ham Ln	38.074740	-121.286298	56

Station Code	Station Name	Latitude	Longitude	Map ID
544XPSAHT	Potato Slough @ Hwy 12	38.111180	-121.499530	57
544RIDAMR	Return Irrigation Drain @ MCD Rd.	37.969830	-121.462300	58
544RIDAHT	Roberts Island Drain @ Holt Rd	37.955600	-121.422300	59
544RIDAHR	Roberts Island Drain along House Rd	37.970200	-121.407400	60
544SCAHFB	Sand Creek @ Hwy 4 Bypass	37.947500	-121.743000	61
544SJRSWC	SJR Source water to Canal @ Holt & Nueger Rds	37.994020	-121.420500	62
544SDMCSC	Storm Drain to Marsh Creek @ Sand Creek Rd	37.946030	-121.702710	63
544XXXD04	Sycamore Slough at Cotta Road (nr Guard Rd)	38.137940	-121.421440	64
544XTTHWT	Terminous Tract Drain @ Hwy 12	38.116580	-121.493690	65
544TPSELR	Tom Pain Sl. @ El Rancho Rd	37.768980	-121.374500	66
544XSED07	Tom Paine Slough at Paradise Rd.	37.771600	-121.386000	67
511ULCABR	Ulatis Creek at Brown Road	38.307000	-121.794200	68
544SJC516	Unnamed Canal at Howard Road	37.876960	-121.376560	69
544XNSJ03	Unnamed canal at west end of Woodbridge Rd	38.152657	-121.498601	70
510XXXSSI	Unnamed Drain Along Sutter Island X Rd	38.295720	-121.592630	71
531UDLTAJ	Unnamed Drain to Lone Tree Cr @ Jack Tone Rd	37.853600	-121.145700	72
544USAWRD	Unnamed Slough @ Woodsbro Rd & Burns Cutoff Levee	37.941740	-121.369100	73
531XSED08	Unnamed Slough at Wildwood Rd	37.863300	-121.128200	74
535WSAWAV	Walthal Slough @ Woodward Ave	37.770460	-121.292300	75
519WDADPR	West Drainage @ Del Paso Rd.	38.656300	-121.560600	76
535WDAJRD	Westport Drain @ Jennings Rd	37.536740	-121.066800	77
535XWDAVR	Westport Drain @ Vivian Rd	37.536820	-121.048610	78
511XXSS03	Willow Slough at Road 99	38.604707	-121.784218	79
511XXSS06	Winters Canal at Road 86A	38.663660	-122.016090	80

Table 21. Acute toxicity to *C. dubia* (96 h Survival) and TIE results; Irrigated Lands Regulatory Program (Ag Waiver), 2003-2008.

Map ID	Sampling Site	Sampling Date	% Survival	Survival %Control	Follow-Up Testing
2	Bear Creek at Alpine Rd	27-Jul-05	0	0	Acute TIE performed
2	Bear Creek at Alpine Rd	08-Aug-07	70	70	
7	Calaveras River at Pezzi Rd	13-Jul-05	0	0	TIE: OP pesticides as toxicants
25	Drain to Grant Line Canal off Wing Levee Rd.	26-Jan-05	40	40	
30	Duck Creek @ Hwy 4	19-Sep-06	0	0	NOEC=50%, EC50=68.3%, TU=1.5. TIE 9/25/06; toxicant: OP pesticide. Resampled, tox. not persistent
31	French Camp Slough @ Airport Way	15-Mar-06	0	0	NOEC=25%, EC50=61.6%, 1.62 TU. TIE 3/18/06: toxicity persistent, due to non-polar organics but not metabolically activated compounds. Resampled, toxicity not persistent.
31	French Camp Slough @ Airport Way	11-Feb-07	0	0	NOEC = 50%, EC50 = 68.3%, 1.5 TU. TIE: C18 column/PBO removed toxicity; toxicant: OP pesticide.
32	Grant Line Canal @ Clifton Court Rd	23-Jan-08	0	0	TIE: toxicant: OP pesticide. Resampled on 01/30/08; toxicity not persistent.
33	Grant Line Canal near Calpack Rd	21-Mar-05	75	78.9	
33	Grant Line Canal nr Calpack Rd	16-Aug-05	5	5	
33	Grant Line Canal near Calpack Rd	16-May-06	10	10.53	TIE 5/20/06: tox. due to hydrophobic, nonpolar organic. Some tox. due to metabolically activated compounds. Resampled, toxicity not persistent.
37	Kellogg Creek @ Hwy 4	16-Feb-05	0	0	
38	Kellogg Creek along Hoffman Ln	15-Mar-06	10	10.5	TIE 3/21/06: toxicity not persistent. Labile contaminants. Resampled, toxicity not persistent.
38	Kellogg Creek along Hoffman Ln	11-Apr-07	45	50	A TIE was initiated but toxicity did not persist to identify its cause. Labile contaminants.
42	Livingston Canal at Cressey Way	10-Jul-07	45	45	
42	Livingston Canal at Cressey Way	24-Jul-07	0	0	TIE: possibility that multiple toxicants present, with one likely to be a pyrethroid.
43	Lone Tree Creek @ Bernnan Rd	27-Feb-06	0	0	NOEC=50%,EC50=70.7%,1.41TUa. TIE 3/8/06: toxicity due to ammonia. Resampled, toxicity not persistent.

Map ID	Sampling Site	Sampling Date	% Survival	Survival %Control	Follow-Up Testing
44	Lone Tree Creek @ Jack Tone Rd	23-Jan-08	0	0	Strong odor of manure, high levels of suspended solids and extremely high oxygen demand. Low DO. Resampled on 01/30/08; toxicity was not persistent.
47	Marsh Creek @ Balfour Ave	21-Jun-05	45	47.4	
47	Marsh Creek @ Balfour Ave	15-Mar-06	60	63.2	Resampled, toxicity not persistent.
48	Marsh Creek @ Concord Ave	20-Jun-07	0	0	100% mortality on Day 3; TIE 6/25/07: non-polar organic chemicals were the cause of toxicity.
50	Mokelumne River @ Bruella Rd	23-Sep-04	5	5.3	TIE: toxicity not persistent; PBO potentiated the toxicity of the sample
50	Mokelumne River @ Bruella Rd	21-Mar-05	35	36.8	
50	Mokelumne River @ Bruella Rd	21-Jun-05	35	36.8	
50	Mokelumne River @ Bruella Rd	27-Feb-06	5	5	TIE 3/2/06, toxicity not persistent. Labile contaminants. Resampled, toxicity persistent.
50	Mokelumne River @ Bruella Rd	10-Mar-06	5	5	Resample, significant toxicity - persistent. Control failure CV>20% 3/11/06 test. Retest run outside hold time for sample.
52	Mormon Slough @ Jack Tone Rd	04-Sep-07	0	0	TIE 09/06/07, toxicity was due to non-polar organic chemicals; 3.2 TU
57	Potato Slough @ Hwy 12	16-Feb-05	30	31.6	
57	Potato Slough @ Hwy 12	27-Feb-06	15	15	TIE 3/2/06: toxicity not persistent. Labile contaminants. Resampled, toxicity persistent.
57	Potato Slough @ Hwy 12	10-Mar-06	15	10	Resample, significant toxicity - persistent. Control failure CV>20% 3/11/06 test. Re-test outside hold time.
59	Roberts Island Drain @ Holt Rd	10-Jul-07	0	0	100% mortality on Day 3; TIE 7/14/07: Tox. due to non-polar organic chemicals, 1.7 TU
61	Sand Creek @ Hwy 4 Bypass	16-May-06	15	15.79	TIE 5/20/06. Tox. persistent and due to particulate-associated contaminant and nonpolar organic. Some tox due to metabolically activated compunds. Resampled, toxicity not persistent.
61	Sand Creek @ Hwy 4 Bypass	20-Jun-06	0	0	Exceeded holding time.
61	Sand Creek @ Hwy 4 Bypass	18-Jul-06	0	0	NOEC=50%,EC50=70.7%,1.4TUa. TIE 7/23/06, toxicity persistent. C18SPE, PBO removed toxicity, due to nonpolar organics or metabolically activated compounds. Resampled, toxicity not persistent.

Map ID	Sampling Site	Sampling Date	% Survival	Survival %Control	Follow-Up Testing
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	11-Feb-07	0	0	NOEC = 50%, EC50 = 68.3%, 1.5 TU. TIE: C18SPE, PBO removed toxicity, due to nonpolar organics or metabolically activated compounds.
72	Unnamed Drain to Lone Tree Creek @ Jack Tone Rd	23-Jan-08	0	0	TIE 1/27/08: OP insecticides was the probable cause of toxicity. Resampled on 01/30/08; toxicity was not persistent.

Table 22. State Water Ambient Monitoring Program (SWAMP), list of sampling sites and map ID numbers (see Figure 2).

Map ID	Station Code	Latitude	Longitude
4	203BAX030	37.918280	-122.325870
5	203CER020	37.898210	-122.303900
6	203COD020	37.881880	-122.306920
7	203LOB020	37.788270	-122.483930
8	203STW010	37.867900	-122.286900
9	203TEM090	37.843590	-122.226860
10	204ALP010	37.697080	-121.849640
11	204ALP100	37.707390	-121.753250
12	204ALP110	37.705280	-121.754170
13	204AMO070	37.676720	-121.814520
14	204AVJ020	37.762530	-122.175390
15	204ISL050	37.741690	-122.442930
16	204LME100	37.817260	-122.261070
17	204PRL020	37.778100	-122.218120
18	204SAU030	37.785660	-122.224240
19	204SLE030	37.725560	-122.183610
20	204SLE230	37.797780	-122.075280
21	204SMA020	37.570280	-122.318610
22	204SMA060	37.562130	-122.328840
23	204SMA080	37.557220	-122.341940
24	204SMA110	37.532330	-122.350880
25	205PER010	37.421180	-122.086730
26	205PER070	37.329410	-122.085860
27	205STE020	37.413570	-122.068650
28	205STE060	37.335030	-122.063840
29	206SPA020	37.967500	-122.365830
30	206SPA070	37.962780	-122.332780
31	206SPA200	37.891390	-122.193890
32	206SPA220	37.886110	-122.192780
33	206WIL020	37.957780	-122.373890
34	207KIR020	38.016500	-121.838810
35	207KIR115	37.991010	-121.894570
36	207MTD010	38.018610	-122.026020
37	207MTD100	37.935700	-121.938860
38	207SUI010	38.218330	-122.104440
39	207SUI020	38.244720	-122.111940
40	207SUI060	38.274580	-122.122750
41	207SUI110	38.330860	-122.138580
42	519LSAC13	38.812400	-121.424500
43	519LSAC14	38.795900	-121.355500
44	519LSAC15	38.805500	-121.308700
45	519LSAC30	38.802350	-121.329500
46	519LSAC31	38.801430	-121.339680
47	519LSAC32	38.812190	-121.451280
48	519LSAC34	38.802760	-121.338420

Map ID	Station Code	Latitude	Longitude
49	519LSAC35	38.804070	-121.328320
50	519LSAC36	38.783820	-121.357480
51	519LSAC37	38.775290	-121.342030
52	519LSAC38	38.766520	-121.339440
53	519LSAC39	38.763770	-121.326190
54	519LSAC40	38.764820	-121.325690
55	519LSAC41	38.764150	-121.322800
56	519LSAC42	38.761340	-121.340190
57	519LSAC43	38.759060	-121.332510
58	519LSAC44	38.794700	-121.346050
59	519LSAC45	38.790470	-121.334150
60	519LSAC46	38.770000	-121.313500
61	519LSAC47	38.769320	-121.299450
62	519LSAC48	38.766920	-121.282520
63	531SAC001	38.290833	-121.375833
64	531SAC003	38.500556	-121.045000
65	531SJC503	37.855556	-121.185000
66	535MER546	37.349722	-120.957778
67	535STC501	37.464444	-121.030280
68	535STC504	37.431944	-121.011667
69	535STC513	37.603056	-121.131667
70	535STC514	37.702500	-121.177222
71	541MER522	37.295278	-120.850278
72	541MER536	37.254167	-120.906944
73	541MER538	37.309444	-120.929167
74	541MER542	37.263889	-120.906111
75	541SJC501	37.675556	-121.264167
76	541STC019	37.413889	-121.014167
77	541STC512	37.342500	-120.977222
78	541STC516	37.521389	-121.148611
79	544SJC001	37.708889	-121.298611
80	544SJC505	37.774167	-121.382222
81	544SJC509	37.785556	-121.534722
82	UP101	37.379170	-122.069670
83	UP102	38.179370	-122.131830
84	UP103	37.318480	-121.784740
85	UP11	38.643680	-121.078960
86	UP22	37.336980	-121.867920
87	UP26	38.033780	-121.332120
88	UP4	38.137370	-122.207980
89	UP47	38.159970	-122.244960
90	UP50	37.918320	-121.714150
91	UP51	38.542190	-121.276160
92	UP57	38.410930	-121.384240
93	UP7	37.977290	-122.353660

Table 23. State Water Ambient Monitoring Program (SWAMP), list of sampling sites and map ID numbers (see Figure 3).

Map ID	Station Code	Latitude	Longitude
A1	AMA002	38.392500	-120.801389
A2	CAL005	38.219722	-120.937770
A3	CAL007	38.170556	-120.807500
A4	CAL008	38.148330	-120.825560
A5	ELD004	38.550833	-120.849722
A6	MER522	37.295278	-120.850278
A7	MER576	37.320556	-120.889167
A8	SAC002	38.236110	-121.418890
A9	SJC 501	37.675556	-121.264167
A10	SJC 507	38.056110	-121.333056
A11	SJC504	37.881667	-121.249444
A12	SJC507	38.056110	-121.333056
A13	SJC512	38.222778	-121.034700
A14	SJC513	38.051390	-121.187780
A15	SJC515	38.042778	-121.321390
A16	SJC516	37.876960	-121.376560
A17	SJC517	37.941630	-121.369300
A18	STC019	37.413889	-121.014162
A19	STC030	37.561944	-121.174167
A20	STC040	37.431944	-121.011667
A21	STC042	37.610556	-121.228611
A22	STC510	37.641944	-121.227770
A23	STC515	37.481389	-121.135550
A24	STC523	37.513820	-121.159860
A25	STC531	37.640530	-121.229310

Figure 1. Irrigated Lands Regulatory Program: Sampling sites in the legal Delta + 30 miles (for detailed site information, see Table 20).

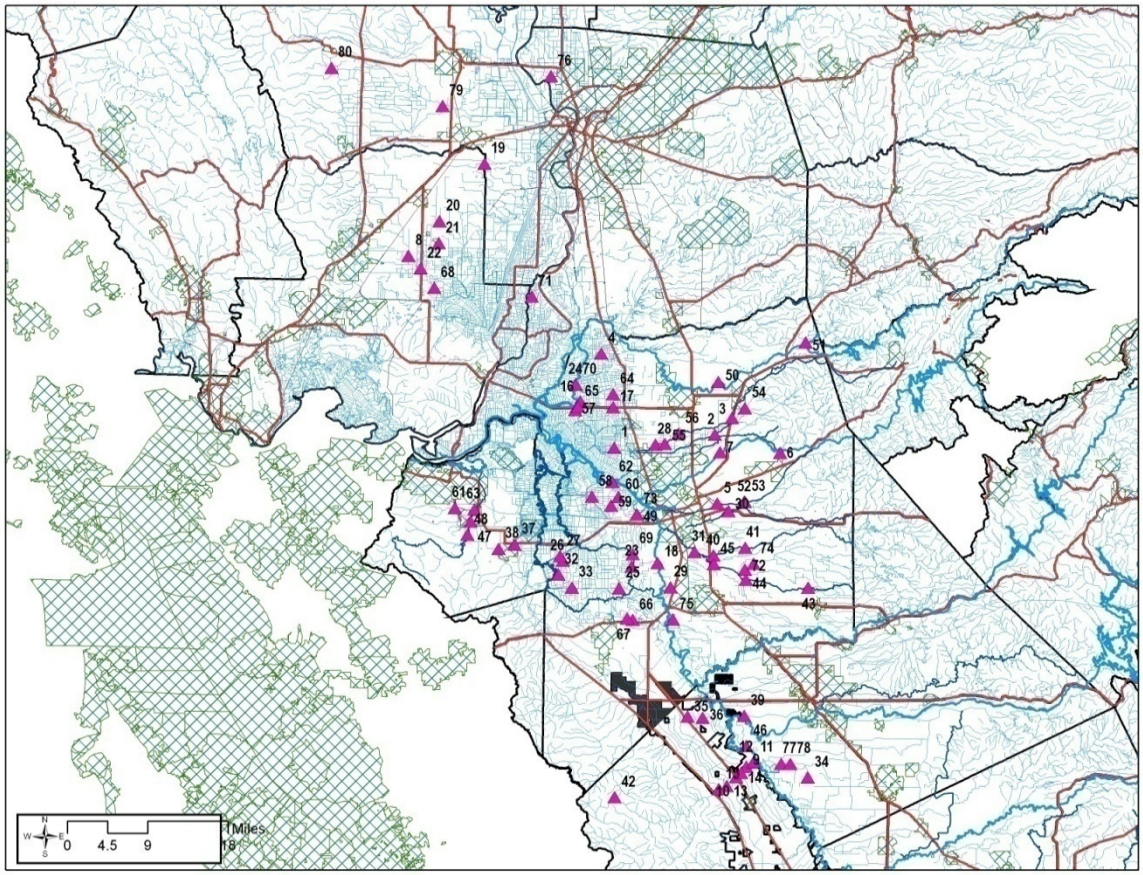

Figure 2. State Water Ambient Monitoring Program (SWAMP): Sampling sites within the legal Delta + 30 miles (for detailed site information, see Table 22)

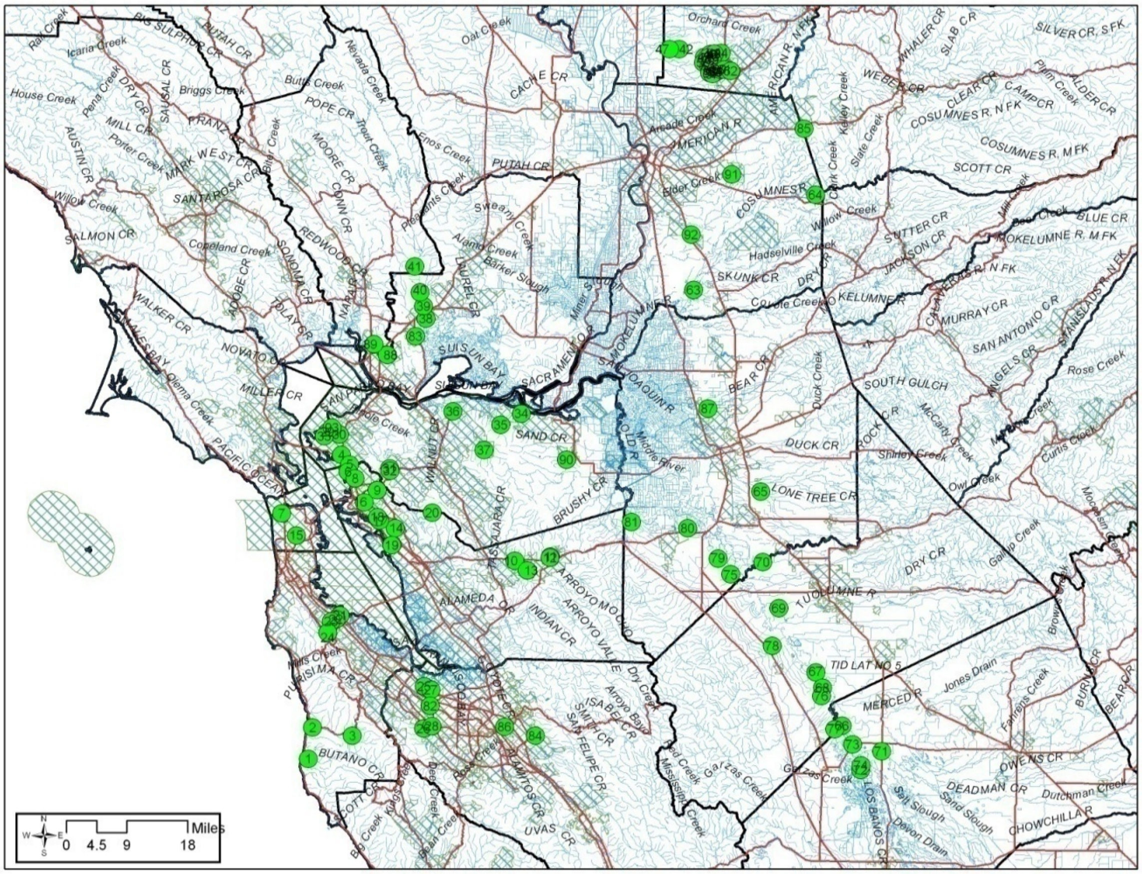

Figure 3. State Water Ambient Monitoring Program (SWAMP): Additional sampling sites 2000-2004 within the legal Delta + 30 miles (for detailed site information, see Table 23).

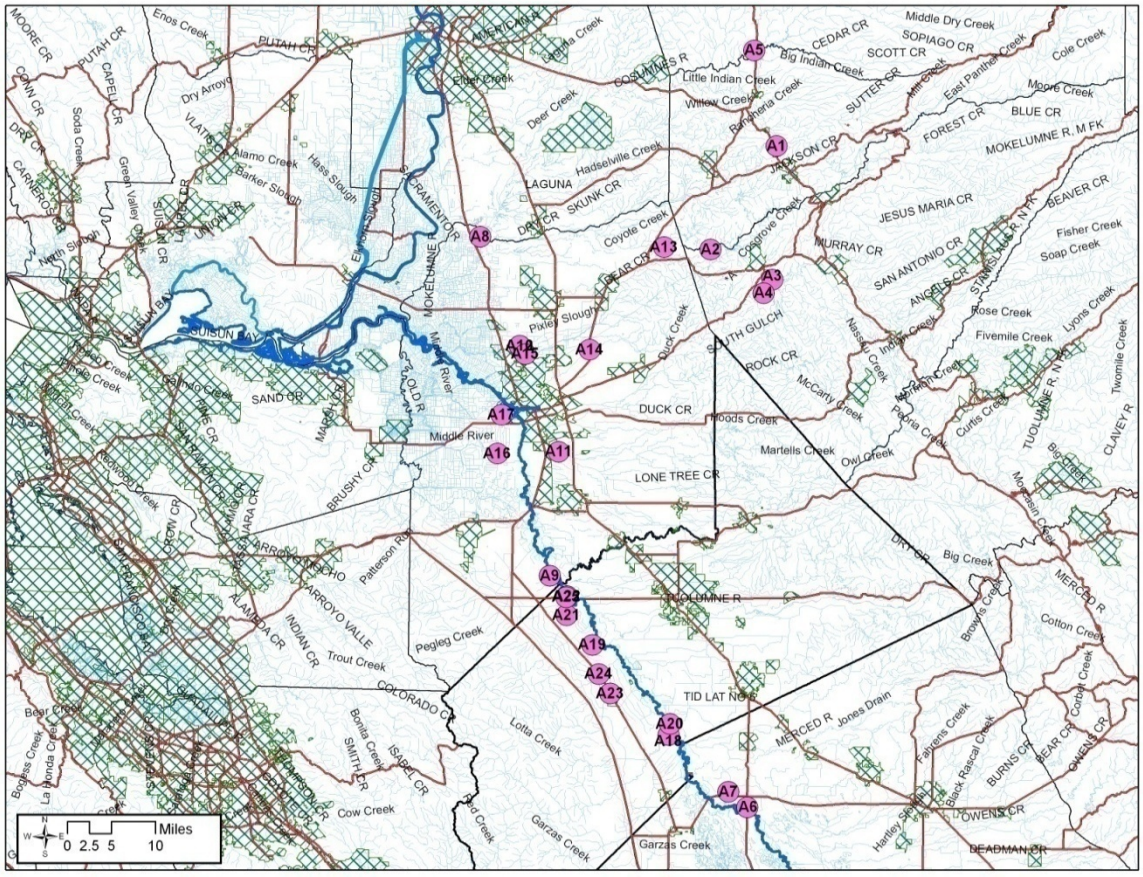

Figure 4. Pelagic Organism Decline (POD) Ambient Toxicity Monitoring: Sampling sites 2005 (for detailed site information, see Table 24).

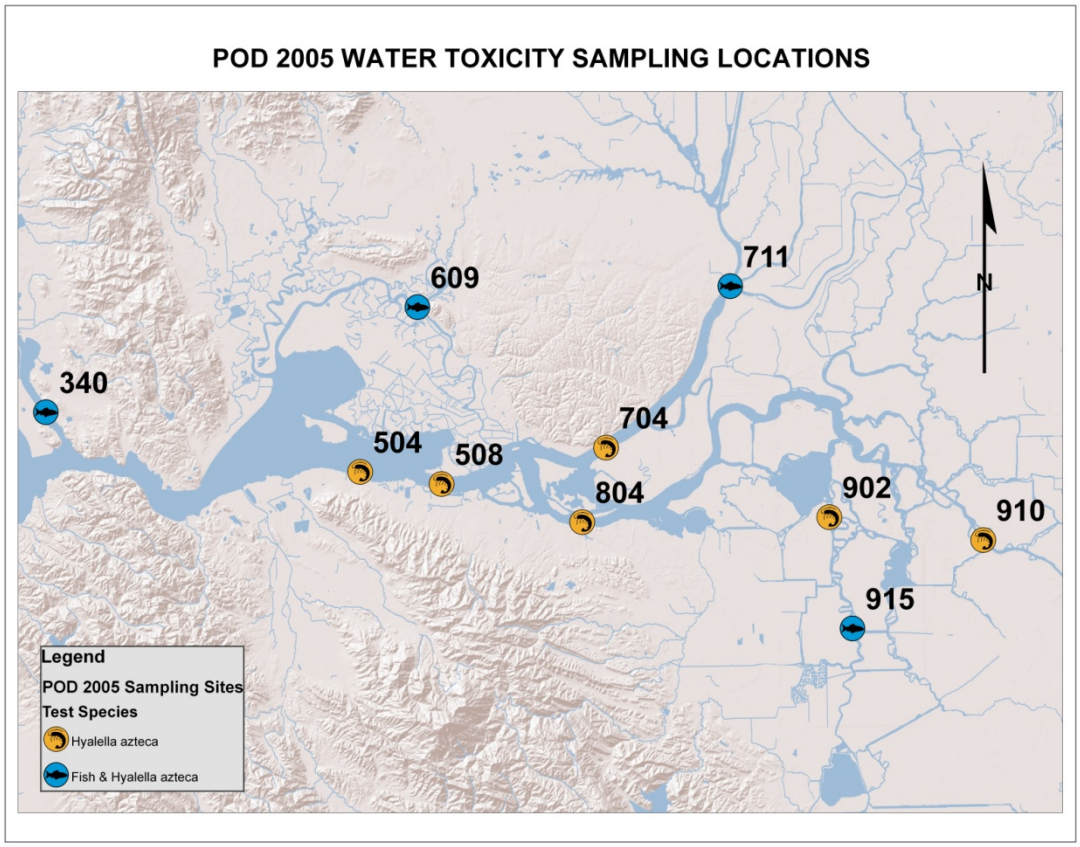

Figure 5. Pelagic Organism Decline (POD) Ambient Toxicity Monitoring: Sampling sites 2006-2007 (for detailed site information, see Table 24).

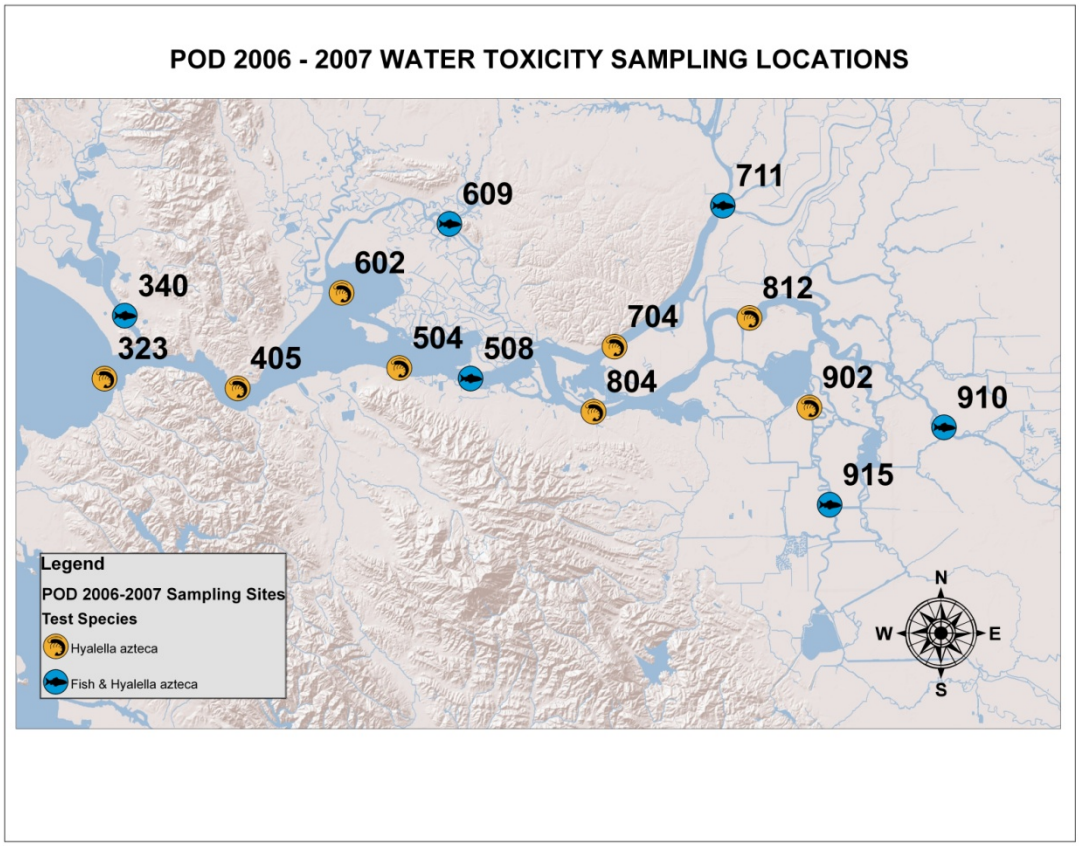

Figure 6. Pelagic Organism Decline (POD) Ambient Toxicity Monitoring: Sampling sites 2008 (for detailed site information, see Table 25).

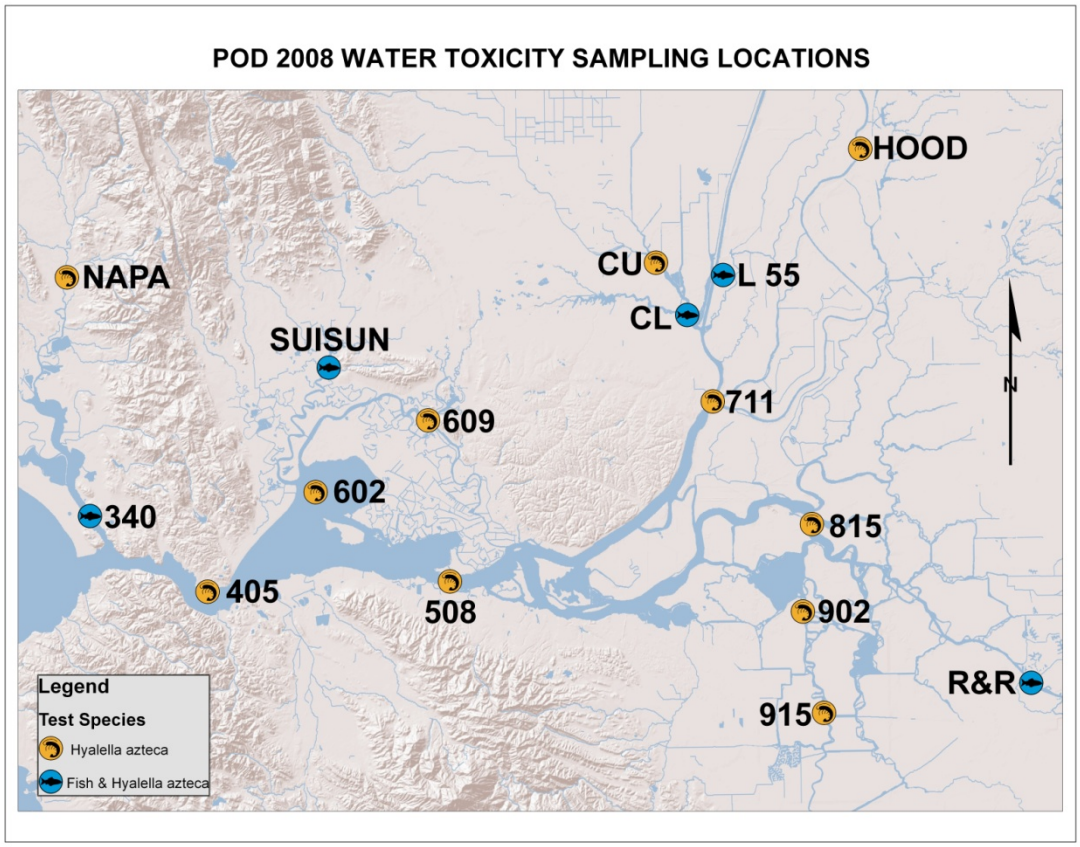

Figure 7. SFEI-Sediment Quality Objectives, sites where 10-d exposure to sediment reduced *H. azteca* or *C. dilutus* survival or growth. Only EMP 0049, 0006 and 0150 caused significant reduction in survival.

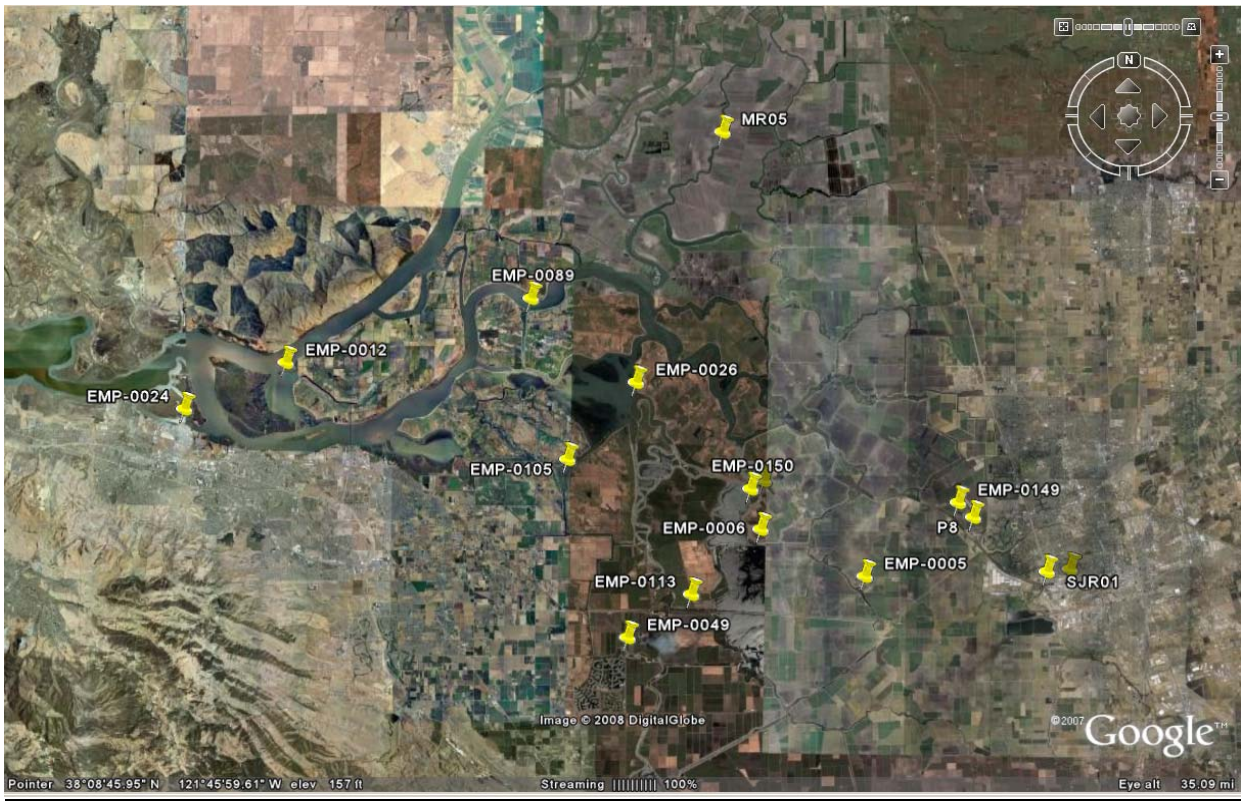

Table 24. Interagency Ecological Program: Pelagic Organism Decline (POD) Ambient Toxicity Monitoring: Sampling stations and GPS coordinates during the 2005-2007 project period.

Station	Location	Latitude	Longitude
323	San Pablo Bay, Rodeo Flats opposite end of rock wall.	38-02'-53.9"N	122-16'-58.1"W
340	Napa River along Vallejo seawall and park.	38-05'-51"N	122-15'-43.9"W
405	Carquinez Straight, just west of Benicia army dock.	38-02'-22.9"N	122-09'-01.8"W
504	Suisun Bay, east of middle point.	38-03'-16.2"N	121-59'-22.2"W
508	Suisun Bay, off Chipps Island, opposite Sacramento North ferry slip.	38-02-'43.8"N	121-55'-07.7"W
602	Grizzly Bay, northeast of Suisun Slough at Dolphin.	38-06'-50.4"N	122-02'-46.3"W
609	Montezuma Slough at Nurse Slough.	38-10'-01.9"N	121-56'-16.8"W
704	Sacramento River, north side across from Sherman Lake.	38-04'-09"N	121-46-'31"W
711	Sacramento.River at the tip of Grand Island.	38-10'43.7"N	121-39'-55.1"W
804	Middle of Broad Slough, west end.	38-01'-05.5"N	121-47'-49.2"W
812	San Joaquin River, just west of Oulton Point.	38-05'-25.1"N	121-38'-25.8"W
902	Old River at mouth of Holland Cut.	38-01'-09.1"N	121-34'-55.9"W
910	San Joaquin River, between Hog and Turner Cut.	38-0'-06.5"N	121-26'-55.3"W
915	Old River-Western arm at railroad bridge.	37-56'-33"N	121-33'-48.6"W
Light 55	Sacramento River Deep Water Channel at Light 55	38-16'-26.5"N	121-39'-42.9"W
Hood	DWR Water Quality Monitoring Station	38-22'-03.6"N	121-31'-13.6"W
Stockton Port	Downstream of Stockton Waste Water Treatment Plant	37-56'-05.7"N	121-19'-48.2"W
Vernalis	DWR Water Quality Monitoring Station, San Joaquin River	37-40'-45.8"N	121-31'-13.6"W

Table 25. Interagency Ecological Program: Pelagic Organism Decline (POD) Ambient Toxicity Monitoring: Sampling stations and GPS coordinates during the 2008 project period.

Station	Location	Latitude	Longitude
340	Napa River, Historic 340 at the seawall	38-05'-51"N	122-15'-43.9"W
405	Carquinez Straight, just west of Benicia arm dock	38-02'-22.9"N	122-09'-01.8"W
Suisun	Suisun at Public Dock	38-13'57.5"N	122-02'14.1"W
Suisun	Suisun Slough at Rush Ranch	38-12'-28.2"N	122-01'56.9"W
508	Suisun Bay, off Chipps Island, opposite Sac. North Ferry Slip	38-02'-43.8"N	121-55'-07.7"W
602	Grizzly Bay, northeast of Suisun Slough at Dolphin	38-06'-50.4"N	122-55'-46.3"W
609	Montezuma Slough at Nurse Slough	38-10'-01.9"N	121.56'-16.8"W
711	Sacramento River at the tip of Grand Island	38-10'-43.7"N	121-56'-55.1"W
Light 55	Sacramento River Deep Water Channel at Light 55	38-16'-26.5"N	121-39'-13.6"W
Hood	DWR water quality monitoring station	38-22'-03.6"N	121-31'-13.6"W
Cache-Lin	Confluence of Lindsey Slough/Cache Slough	38-14'-39.2"N	121-41'-19.5"W
Cache-Ul	Upper Cache Slough, mouth of Ulatis Creek	38-17'-02.7"N	121-43'-04.3"W
815	San Joaquin, Confluence of Potato Slough	38-17'-01.5"N	121-34'-21.5"W
902	Old River at mouth of Holland Cut	38-01'-09.1"N	121-34'-55.9"W
915	Old River, western arm at railroad bridge	37-56'-33"N	121-33'-48.6"W
R&R	San Joaquin, Rough & Ready Island	37-57'45.4"N	121-21'55.9"W
Napa	Napa River in Napa City at end of River Park Blvd.	38-16'-39.7"N	122-16'-56.9"W

APPENDIX III

AVAILABLE MODELS FOR FISH POPULATIONS

FRANK LOGE

TABLE OF CONTENTS

INTRODUCTION

Recent declines in the abundance indices of four pelagic fish species in the San Francisco Bay and Sacramento - San Joaquin Delta area (Bay-Delta) have prompted further investigation into causal reasons for this decline. Termed the pelagic organism decline (POD), recent focus has been upon four species of pelagic fish; Delta Smelt, Treadfin Shad, Longfin Smelt, and Striped Bass. Long-standing abundance indices for all four species, tabulated by the Interagency Ecological Program (IEP), revealed a near simultaneous decline beginning in late 2000, with record or near record lows being recorded for all four species in the subsequent years (Baxter 2008).

Many factors have been identified as possible reasons for the POD, although none appear to be a sole cause. Important factors in the POD described recently by the *2007 Pelagic Organism Decline Progress Report* include: (i) previous abundance levels, (ii) habitat, (iii) top-down effects and (iv) bottom-up effects. Previous abundance levels describe the links between adult abundance and juvenile production and how survival between various life stages may have changed since the beginning of the POD. Habitat describes the role that water quality constituents, including toxic contaminants, may play in the POD. Top-down effects describe the role of predation and water project/diversion entrainment upon mortality rates (potentially life-stage specific mortality rates), associated with the POD. Similarly, bottom-up effects describe food web interactions, particularly the availability and suitability of prey, and the influence this may have upon the POD (Baxter 2008).

Since numerous factors have been implicated in the POD, it is helpful to place these factors in a model framework that allows for the evaluation of each factor, relative to each other, to ascertain the factor's effect upon the fish population dynamics. The objective of this document is to review and evaluate existing fish population models relevant to the POD species.

1.0 GENERAL FISH MODELS

1.1 REGRESSION MODELS

Several researchers (Jenkins 1968, 1976; Aggus and Lewis 1978) have related standing crops of fish in reservoirs to environmental variables such as morphological features (surface area, depth, shoreline development), hydrologic variables (storage ratio, water level, surface area, discharge), and productivity (dissolved solids, growing season, reservoir age) (Johnson 1993). These studies resulted in regression models that were variable for different fish species with multiple correlation coefficients (R^2) generally less than 0.5, however hydrologic variables and dissolved solids were most consistently correlated with standing fish crops (Johnson 1993). Further relationships were developed by Ploskey et al. (1984) who regressed reservoir storage ratio and surface area against fish densities of 33 different fish species. Reservoirs were classified into hydropower storage, hydropower mainstream and flood control which were characterized by water retention time and water lever fluctuations. Depending upon the classification, the densities of different species were positively or negatively correlated with surface area changes and/or inflow and typically varied by season. While the Bay-Delta system is not a hydropower production or water storage system, the method of reservoir classification permits these regression relationships to be applied in areas sharing similar hydrologic properties (Johnson 1993). Given the hydrodynamics of the Bay-Delta system, applying these regression relationships to this region may only be useful for approximation purposes. For example, historic hydrodynamic data throughout the Bay-Delta system is known for roughly the last 30 years and applying the regression relationships developed by Ploskey et al. (1984) to the Bay-Delta system would likely only aid in addressing some top-down impacts (such as water diversion changes) upon the POD population dynamics. These regression models described above only assess the mean standing crops of adult fish and may be unsuitable at explaining the dynamics of fish abundance (Johnson 1993). However, they may be useful to indicate specific processes that could be targeted for study or to prioritize studies in the Bay-Delta system.

An adult model originally proposed by Horst (1975, 1978) and subsequently modified by Goodyear (1978) and MacCall et al. (1983), assess the egg and larval entrainment in power plants and subsequently estimates the effect on the fish population. The model requires the number of fish lost to entrainment, survival rates between life stages (i.e. egg to larvae survival), and an estimation of the fecundity of the females (Johnson 1993). The modifications by Goodyear (1978) and MacCall et al. (1983) separated larvae into size classes with different mortality rates and derived a per capita rate of entrainment, respectively. This model assumes the population is in equilibrium with constant annual recruitment and no density dependant or compensatory processes (Johnson 1993). While this model was developed for power plant entrainment, it can be theoretically applied to any source of mortality that can be measured (Johnson 1993). This model may provide a first approximation to the effect of a particular mortality source, although realistic estimates of the loss of eggs or larvae due to a particular mortality source would first need to be known (Johnson 1993). Moreover, applicability of this model to the Bay-Delta system appears limited since the onset of the POD, the fisheries do not appear to be in equilibrium and potential density dependence or compensatory processes appear to be operating. Measuring egg or larval mortalities due to specific stressors associated with habitat and bottom-up factors would likely prove difficult. The model may have some usefulness as a tool to help determine bounds of potential losses under optimistic to worst-case scenarios (Johnson 1993), particularly related to water project diversions in the Bay-Delta system where some degree of estimation of egg and larval mortalities is possible.

1.2 MATRIX PROJECTION MODEL

The matrix modeling approach of Kareiva et al. (2000) aims to develop a steady-state projection of the population dynamics of the species based upon survival, fecundity and growth estimates. In this matrix model, fish populations can be considered to be in different life-stages (i.e. eggs, larvae, juveniles and adults). The matrix model is defined as follows:

$$\vec{n}_{t+1} = [A]\vec{n}_t$$

Where *n* is the vector containing population at each stage and [A] is the population projection matrix defined as:

$$[A] = \begin{bmatrix} S_e & 0 & F_j & F_a \\ G_e & S_l & 0 & 0 \\ 0 & G_l & S_j & 0 \\ 0 & 0 & G_j & S_a \end{bmatrix}$$

In which, subscripts *e, l, j,* and *a* refer to egg, larvae, juvenile and adults respectively, and S, F, and G are respective probabilities of survival in the current stage, fecundity rate at each life stage, and growth to the next stage. The values of the above mentioned population transition coefficients are required inputs and can often be obtained from literature, field studies or outputs of other population models. Results from this matrix modeling approach can be used to determine the effect of various factors on growth or decline of the whole population.

Taken with the initial assumption that this matrix modeling approach assumes a steady-state population, the application of this approach to the POD species in the Bay-Delta is relatively straightforward. As long as reliable estimates of survival, growth and fecundity are known, a projection matrix similar to that shown above could be developed. In any event, it is important to remember that for the POD species in the Bay-Delta the existence of a steady-state population dynamics is highly questionable, especially in light of the recent declines over the past several years, thus such an approach must only be used where appropriate.

1.3 MECHANISTIC MODELS

Generally, for the Bay-Delta system and the POD, the most applicable fishery models are mechanistic models that aim to simulate processes that occur on individual fish or fish populations and output results relevant to the

population dynamics of the fish species. Simulated processes typically will include growth over time, fecundity and mortality, and can also include other environmental features such as contaminant accumulation, density dependence, habitat suitability, and species interactions (Johnson 1993). Typically with these models, the greater detailed inputs produces greater detailed outputs, allowing for a broader interpretation of the population dynamics. Three broad ranges of mechanistic models were defined by Johnson 1993 to include: Population Dynamics Models, Bioenergetics Models and Individual Based Models. This list can be expanded to include contaminant accumulation models, which may be particularly important in the Bay-Delta system.

1.3.1 POPULATION DYNAMICS MODELS

Simply put, population dynamics models evaluate the basic functions of population growth, including (but not limited to), recruitment, growth, fecundity and mortality. Species specific parameters are often developed to allow model application to a broad range of fish species.

The Generalized Inland Fishery Simulator (GIFSIM) developed by Taylor (1981) is capable of handling simple to complex age-structured fish populations (Johnson 1993). The model simulation is annual but can be broken down monthly to assign different growth and mortality rates for each month. The model allows for coverage of 15 different age classes, a time span sufficient for most of the POD species, with striped bass being a possible exception. The model allows for the incorporation of recruitment, density dependence and normally distributed random effects into the functions which describe growth, mortality and egg survival (Johnson 1993). The required minimum inputs into the model include population level, age specific mortality rates, initial population numbers, length-weight relationships, weight-fecundity relationships, and mean length-at-age estimates. If data is available on seasonal growth or density dependence related to growth, such features can also be incorporated into the model (Johnson 1993). The end output of the model is temporal coverage of mean population size, biomass by age, and mean recruitment (Johnson 1993). The GIFSM does not explicitly include any environmental effects that could change over time and impact population levels (e.g. hydrodynamics, water diversions, toxic contaminants, food web interactions, and predation). Such environmental effects could be incorporated through manipulating input parameters (e.g. lowered food supply may reduce growth or length-at-age), however, if the affect of an environmental factor on growth, for example, were unknown, it would be problematic to incorporate such an effect. The model is expandable/re-programmable so with some modification, some basic environmental factors may be incorporated (Johnson 1993).

Another similar age structured population dynamics model is the RAMAS Metapop 5.0 (Applied Biomathematics, Setauket, NY). RAMAS is designed broadly and is not designed specifically for any single fish species. The model is described herein as it would apply to any of the POD species. One major difference between GIFSM and RAMAS, is that RAMAS incorporates a spatial component of a metapopulation and allows for user configuration of fish populations, into multiple patches if desired. The benefits of a spatial component allow for different age structures, and different fecundity and mortality rates to be defined in each location. RAMAS does not require any length-at-age (growth) input data and as such does not output any information on growth. The required inputs to RAMAS include (not all are required): survival/mortality rates, fecundity rates, age structure, sex structure, density dependence information, carrying capacity abilities and natural random variations. RAMAS also can utilize

temporal trends in mortality rates, fecundity rates, and carrying capacities. RAMAS can handle multiple different populations of a single species at one time and can simulate up to 500 time steps. The time steps of RAMAS depend upon the input data and age structure of the modeled fish species. For most of the POD species, this time step would likely be on the order of several months to a year, given the available data sources. The outputs of RAMAS are similar to the GIFSM and essentially include population abundances over time. Like the GIFSM, RAMAS is not written to explicitly evaluate environmental effects upon a fish population or sub-population. However, given the flexibility of RAMAS, input variables may be manipulated to begin to address an environmental effect upon the population.

1.3.2 BIOENERGETICS MODELS

Bioenergetics models evaluate the growth and food consumption of fish over time by tracking energy budgets (Johnson 1993). The underpinnings of bioenergetics are rooted in the laws of thermodynamics and based upon energetic principles developed by Kitchell et al. 1977 (Hanson et al. 1997). Alone, they are not very useful in predicting population dynamics but can be coupled with population dynamic or individual-based models to aid in estimating biomass and consumption over time (Johnson 1993).

A generalized bioenergetics model was developed by Hewett and Johnson (1987) and subsequently expanded, culminating in *Fish Bioenergetics 3.0* (Hanson et al. 1997). This generalized model can be applied to any fish species and models the physiology and growth of a fish based upon several core bioenergetic processes and caloric densities of the fish and its prey (Johnson 1993, Hanson et al. 1997). The core bioenergetic processes include consumption, respiration waste losses and reproduction. Water temperature effects all of the bioenergetic processes and is also an important factor in the bioenergetic model.

While the *Fish Bioenergetics 3.0* model can be applied to any fish, it requires an extensive set of parameters to accurately describe the physiology of each fish and its subsequent growth. This model can also then be subdivided by life-stage, assuming parameters differ, to evaluate the effect of feeding at various life-stages upon growth. The parameter dataset required for this general bioenergetics model is usually not considered site specific (Hanson et al. 1997, Johnson 1993) so parameters established outside the Bay-Delta area should still be applicable. The bioenergetics model is not age structured and therefore does not require age-specific data, however, if age-specific parameters are available, the model can be run as such. Bioenergetic models can simulate the effect of bottom-up effects from a change in food supply, for example, to the degree that it affects the growth of the fish, since the output of the model is growth (i.e. weight change) per time step per fish. Other environmental effects arising from the top-down and habitat factors are more difficult to model with bioenergetic processes, unless these factors conclusively effect growth. Moreover, population level dynamics are not explicitly represented in a bioenergetics modeling approach unless size-dependant mortalities are apparent. However, modeled growth information can be useful in the development of other population dynamic or individual based models that then can give estimates to population level changes.

Bioenergetic models specific to each POD species will be discussed below, but in general, the required inputs for such models include water temperature, diet composition, consumption and the caloric density of the diet. Much of this data is site-specific and has temporal variations, therefore necessitating a sizable dataset on water temperature and diet to ensure accuracy.

1.3.3 INDIVIDUAL-BASED MODELS

Individual-based models (IBMs) aim to simulate population or age classes of fish by modeling biological processes on each individual fish (Johnson 1993). Modeled biological process can encompass feeding, swimming ability, movement, competitive interactions, contaminant accumulation, mortality, and fecundity, among other processes. Population dynamics of the modeled population is then determined by the collective mortalities and fecundities of the modeled individuals. To date, the majority of IBMs developed for fish populations are limited to a particular lifestage and focus on growth (Tyler and Rose 1994, Johnson 1993). The modeled lifestages usually cover those critical to survival or to establishing recruitment (Tyler and Rose 1994). Environmental effects arising from prior abundance levels, habitat, top-down, and bottom-up factors can all be incorporated into IBM's as long as there is a method to describe how these factors influence the biological process of the individual.

The basic structure of IBMs typically include an initial population estimate and definition of environmental constituents (e.g. temperature, salinity, food availability, etc), then at each timestep, the model determines how the defined biological processes affect the individual. Environmental constituents can change on a frequent basis and, if the data exists, can be incorporated into the model (Tyler and Rose 1994). Once the model framework is built, components of the model can be elaborated to address specific questions. For example, Trebitz (1991) considers the effect of different spawning rules for largemouth bass on population survivorship, Madenjian (1991) and Madenjian *et al.* (1991) consider the effect of foraging on the growth and size distribution of young-of-the-year (YOY) walleye populations, and Rose and Cowan (1993) examine the effect of foraging success, seasonal changes in the environment, and size dependent mortality on YOY striped bass survival (adapted from Tyler and Rose 1994).

Some IBMs have broken down the spatial homogeneity that is often assumed in other models, allowing varying habitat effects (i.e. a heterogeneous environment) to be incorporated on individual fish. A model developed by Bartsch et al. (1989) describes distribution of herring larvae in the North Sea by combining a hydrodynamic modeling and IBM techniques. Waiters et al. (1992) develop a model similar to the model of Bartsch et al. that describes the distribution of English sole larvae in the Hecate Strait, British Colombia. Smallmouth bass populations in streams were described Jager et al. (1993) in an IBM that incorporated biological detail of past IBMs along with spatial details found in the Bartsch et al. (1989) and Waiters et al. (1992) models (Tyler and Rose 1994). The inclusion of habitat descriptions in an IBM is usually guided by available data sources and the fish species being modeled. Since the Bay-Delta system is mostly a heterogeneous environment and the POD species are widespread throughout this system, an effective IBM for this system should include a detailed habitat description to the degree possible.

Since IBMs assess biological processes upon individual fish one major process, fish movement, can be explicitly evaluated. Without the use of an IBM, movement rules and descriptions would need to be applied to a large cohort or entire population of fish. The advantage of moving individual fish allows for the definition of different environmental conditions spatially, and can subsequently elicit different physiological responses in the individual modeled fish, potentially resulting in changes to the population dynamics. Using partial differential equations (PDEs), Kareiva and Odell (1987) and Turchin (1989) developed models that specifically addressed individual movement. Results to the population then stem from results of the individuals' actions rather than vice versa (Tyler and Rose 1994). These movement models do not explicitly address population dynamics, but rather provide population distributions over a time period based upon individuals' movement (Tyler and Rose 1994). Numerous other methods of describing individual movement have been developed, with biased random-walk methods involving the fewest assumptions. Biased random-walk methods only assume movement towards a location (DeAngelis and Yeh 1984) with a pre-defined bias (Tyler and Rose 1994). The inclusion of movement into an IBM for modeling POD species is likely important and the selection of a movement modeling approach may be species dependant due to differing life strategies.

1.3.4 CONTAMINANT ACCUMULATION MODELS

The bioaccumulation of contaminants in fish species has been the focus of numerous research studies and numerous fields studies have been conducted throughout the Bay-Delta region where various contaminant concentrations have been recorded. With this ever growing contaminant database, modeling contaminant accumulation has become more widely applicable. Fish accumulate contaminants across their gills and through dietary sources, with the bulk of accumulation postulated to occur through dietary uptake (Hanson et al. 1997).

Three general simplified contaminant accumulation models are presented by Hanson et al. 1997. These three models assume that contaminant uptake across the gills is negligible compared to dietary uptake. The first model is the most simple and assumes accumulation is a constant fraction of contaminant consumption and elimination is constant. The required inputs are prey consumption per time step, contaminant concentration in consumed prey and an assimilation efficiency which accounts for all losses. The resulting output is a time rate of change in contaminant concentration in the modeled fish. The second model presented by Hanson et al. 1997 expands upon the first model by explicitly accounting for a mass specific contaminant elimination rate from the fish. The third and final model presented by Hanson et al. 1997 expands upon the second model by accounting for the contaminant elimination being both mass and water temperature specific (Hanson et al. 1997). As the complexities of the models develop, the more input data is required.

A more integrated approach comes from the Canadian Centre for Environmental Modeling and Chemistry (CEMC). The CEMC has developed a fish model that is a single organism bioaccumulation model which accounts for a steady-state uptake and loss of an organic contaminant by a fish. Uptake processes occur through respiration through the gills and dietary sources. Losses occur through gill transfer, egestion in feces, metabolic conversion, and growth dilution (CEMC Fish Model 2004). According to the CEMC Fish Model description, "this model is useful for estimating the likely extent of bioaccumulation in the fish and biomagnification from the food to the fish, and the relative importance of each uptake and loss process. The results of changes to chemical, fish, and

environmental properties may be explored by modifying the input data. Incremental changes in input properties can be used to obtain a sensitivity analysis." The required major inputs into the model include: the chemical properties of the contaminant, dietary and water contaminant concentration, fish size, fish lipid fraction, consumption rate of prey, contaminant assimilation efficiencies, growth rate and elimination rate (CEMC Fish Model 2004). This model is useful to POD species for organic contaminants found throughout the Bay-Delta system, assuming the required input data is known. This model or a modification of this model could then be integrated into an individual-based model to determine contaminant affects upon individuals and in turn a modeled population. Alternatively, this model would be useful for evaluating the relative importance of various organic contaminants to individual fish to help guide further data collections, laboratory studies and/or model development.

Thomann (1981) uses a general mass balance approach where the change in contaminant concentration in a fish is equal to the amount of the contaminant absorbed from the water plus the contaminant concentration absorbed from the food minus the contaminant concentration eliminated by the body. There is also a growth effect term wherein as the fish grows dilution of the chemical occurs. This equation assumes an equal distribution of the contaminant throughout the body of this fish. The growth rate and weight specific realized consumption are required inputs and are identical to outputs that can be derived from a bioenergetics model. Similar to the CEMC Fish model described above, required inputs include the dietary and water contaminant concentration, as well as assimilation efficiencies from each sources. An estimation of the elimination rate of the contaminant is also required. This modeling approach of Thomann (1981) is described in further detail in the below section.

2.0 STRIPED BASS SPECIFIC FISH MODELS

2.1 STRIPED BASS POPULATION DYNAMICS MODELS

Logan (1985) related mortality of Hudson River young striped bass to their inverse size. The modeled result showed that year-class strength responded more strongly to growth and hatch length then egg mortality. Additionally, as fish grow the effect of changes in hatch length and growth rate were found to diminish. Environmental effects are not directly incorporated into this model, however the disappearance of Hudson River larvae during the spring of 1976 was attributed to lowered water temperatures, which decreased growth rates and allowed for an accurate mortality prediction from the model on the population. Sublethal exposure to toxicants can also reduce growth rate and hatch length, therefore increasing mortality according to the model (Logan 1985). The required model inputs include an age-structured dataset containing length at age (for early life-stages) and an initial population estimate. Logan (1985) postulates that a similar approach to modeling population dynamics developed here should be valid for other estuarine and marine species. While this model was developed upon an East Cost stock of striped bass, its applicability to the Bay-Delta region is likely valid. Beginning in the late 1960's and continuing through the early 1990's the California Department of Fish and Game (DFG) began an extensive striped bass egg and larval survey where numerical estimates of eggs and larvae were taken during the spring months at stations throughout the Delta. This dataset would likely be an ideal dataset to adapt and/or to expand Logan's (1985) approach.

2.2 STRIPED BASS BIOENERGETICS MODELS

Several bioenergetics models for striped bass have been developed for east coast stocks of striped bass (Overton et al. 2005, Hanson et al. 1997; Hartman and Brandt 1995 a & b). Most of the existing models are similar in construction and utilize some different parameters depending upon location and the desired outcomes of the bioenergetics model. Currently there is a bioenergetics model under development for the striped bass stocks in the Bay-Delta as part of a larger life-cycle individual based model. This bioenergetics model follows Hartman and Brandt (1995) and Hanson et al. (1997) and is described in detail below.

The overall bioenergetics equation formulated to evaluate the weight change of the Bay-Delta striped bass over time period (t):

(1) $$W_t = W_{t-1} + \left(G \times e_{dens}\right) - S_p$$

Where, W_t is the fish weight (g_{bass}) at time t (day), W_{t-1} is the fish weight at the previous time step, G is specific growth ($g_{prey} * g_{bass}^{-1} * d^{-1}$), e_{dens} is the energy density ratio of striped bass prey to the striped bass and S_p is the weight lost due spawning (egg release) (g_{bass}). The specific growth, energy density ratio and spawning terms are described in further detail below.

2.2.1 SPECIFIC GROWTH

The specific growth rate as described in Equation 1 above, is a function of realized consumption, respiration/metabolism, egestion, excreation and specific dynamic action (Hartman and Brandt 1995):

(2) $G=C_r-(R+SDA)-F-U$

where, G= Specific growth, C_r= Specific realized consumption, R= Specific metabolism, F= Specific egestion, E= Specific excretion, SDA=Specific dynamic action. All units in this equation are in terms of grams prey consumed per gram striped bass per day ($g_{prey} * g_{bass}^{-1} * d^{-1}$). Specific egestion, excretion and SDA terms are constants in this model formulation with their values obtained from laboratory experiments on striped bass. The specific realized consumption and metabolism parameters are more complex and are discussed below.

2.2.2 CONSUMPTION

MAX CONSUMPTION

The realized consumption rate (C_r), as noted in Equation 1.2, is defined as the proportion of a maximum consumption rate, C_{max} ($g_{prey}\ g_{bass}^{-1}\ d^{-1}$) that depends upon water temperature and striped bass weight. The maximum consumption rate is defined by the allometric equation:

(3) $$C_{\max} = CA * W^{CB} * f(T)$$

where, *CA* is the intercept of the allometric mass function and *CB* is the slope of the allometric mass function, and both terms are life-stage specific constants obtained from Hartman 1995 (Table 4, appendix). The maximum consumption rate also depends upon the fish weight (*W*) in grams and a temperature dependent function (*f(T)*), based upon the Thornton and Lessem (1978) algorithm:

(4) $f(T)=K_a(T) * K_b(T)$

$$\mathrm{K_a}(\theta) = \frac{K_1 e^{\gamma_1 (T-\theta_1)}}{1 + K_1 (e^{\gamma_1 (T-\theta_1)} - 1)}$$

$$\gamma_1 = \frac{1}{\theta_2 - \theta_1} \ln \frac{K_2 (1-K_1)}{K_1 (1-K_2)}$$

$$\mathrm{K_b}(\theta) = \frac{K_4 e^{\gamma_2 (\theta_4 - T)}}{1 + K4 (e^{\gamma_2 (\theta_4 - T)} - 1)}$$

$$\gamma_2 = \frac{1}{\theta_4 - \theta_3} \ln \frac{K_3 (1-K_4)}{K_4 (1-K_3)}$$

where, T is the water temperature in degrees C and all other values are life-stage specific constants, obtained for striped bass from Hartman and Brandt 1995.

REALIZED CONSUMPTION

The daily realized consumption rate (C_r) is a proportion of the above C_{max} (Equation 1.3) and can be described as:

(5) $C_r = p * C_{max}$

where, C_r is again the realized consumption ($g_{prey}\ g_{bass}^{-1}\ d^{-1}$) and *p* is the coefficient of consumption. For a single source of prey, the coefficient of consumption is a function of the prey density and the half saturation constant of the prey:

(6) $$p = \frac{\lambda}{\lambda + K_s}$$

where, λ is the prey density ($g_{prey}\ m^{-3}$) and K_s is the prey half-saturation constant in ($g_{prey}\ m^{-3}$).

When multiple food sources are considered, several modifications to Equation 1.6 are necessary, depending on the way the predator responds to limitation in one or more prey. Here, the coefficient of consumption described above was modified to reflect the fraction of each prey type *j* (*j*=1,2,…, *n*) consumed out of the total prey types consumed. Additionally, eutrophic conditions (*p*=1) are obtained with plentiful supply (with respect to individual half-saturation coefficients) of any of the individual prey; this is also reflected in the below description. Thus a Type II functional response equation for multiple prey types (Megrey et al. 2006, Rose et al. 1999) is employed:

(7) $$p = \sum_{j=1}^{n} p_j$$

where,

(8) $$p_j = \frac{\lambda_j / K_{s,j}}{1 + \sum_{m=1}^{n} \lambda_m / K_{s,m}}$$

Where λ_j is the density of prey type *j* ($g_{prey}\ m^{-3}$) and $K_{s,j}$ is the half-saturation constant of prey type *j* ($g_{prey}\ m^{-3}$). The summation term in the denominator of Equation 1.8, $\sum_{m=1}^{n} \lambda_m / K_{s,m}$, represents the feeding on prey type *m* (*m*=1,2,…,*j*,…,*n*) The formulation of Equation 1.8 assumes that the vulnerabilities of prey type *j* to predation by striped bass are equal.

IDEAL DETERMINATION OF λ_J AND $K_{S,J}$

Striped bass life-stages have been defined in part by changes in prey preference that occurs as the fish matures. Therefore, for each life-stage, different prey types may be considered. For example, young striped bass feed

primarily upon small zooplankton and related organisms while mature adults feed largely upon other small fish. The types of prey important to each life-stage will be inferred from literature and gut content analysis of striped bass. For each life-stage, prey densities of essential prey should be defined as per available data. Spatial variants in prey densities is also important and should defined by region within the Bay-Delta, as data permits.

Once prey types are determined, half saturation constants for each prey type can be determined by calibration to field data. Gut content analysis of striped bass will be used to iteratively solve for $K_{s,j}$ by incorporating prey densities from field data. This approach assumes that the organisms found in the gut analysis are representative of the type of prey the fish eats and the proportion of each prey it eats. Alternatively, some half saturation constants may be available from laboratory experiments.

ACTUAL DETERMINATION OF λ_j AND $K_{S,J}$

Unfortunately, the needed data to follow the above approach is not yet available to the degree necessary. Prey densities could be selected for various regions throughout the Bay-Delta to match observed striped bass growth, however then this leaves the half saturation constants without a calibration data source. Estimated half saturation constants may be obtained from literature and East coast stocks of striped bass, however, this is not an ideal case. To simplify this process, both the half saturation constants and prey densities can be removed and a straightforward calibration on *p* (the coefficient of consumption) to match field growth data can be preformed. This approach obviously involves several inherent assumptions, but different p-values can still be defined for each life-stage and can vary by space and time as well, if there is adequate data to substantiate such a dependence.

MULTI-SPECIES REALIZED CONSUMPTION

The daily realized consumption rate for each prey type *j* (C_j), as is a proportion of C_{max} and can be described as:

(9) $$C_j = C_{\max} p_j$$

And the total daily realized consumption rate is:

(10) $$C_r = \sum_{j=1}^{n} C_j = C_{\max}\left[\sum_{j=1}^{n} p_j\right] = C_{\max}\left[\sum_{j=1}^{n} \frac{\lambda_j / K_{s,j}}{1+\sum_{m=1}^{n} \lambda_m / K_{s,m}}\right]$$

where, C_r is the realized consumption ($g_{prey}\ g_{bass}^{-1}\ d^{-1}$) and *p* is the coefficient of consumption.

METABOLISM

The metabolism term (R) in Equation 2 is described by:

(11) $R=RA*(W^{RB})*(e^{RQ*T})*ACT * 5.258$

where,

RA, RB. RQ= Species specific constants

W= fish weight (g_{bass})

T= Temperature (ºC)

ACT= activity multiplier constant

The final value in Equation 1.11, 5.258 coverts the metabolism equation from $g_{O2}*g_{bass}^{-1}*d^{-1}$ into $g_{prey}*g_{bass}^{-1}*d^{-1}$ using the following conversion:

$$\frac{13560J}{g_{O_2}} \times \frac{1g_{zoop}}{2580J} = 5.258 g_{zoop}\, g_{O_2}$$

Note that this conversion is only for zooplankton and other food types will need their own conversion factors. For the interim, this value has been assumed for all prey types.

2.2.3 CONVERSION OF SPECIFIC-GROWTH (G) "NON-SPECIFIC" UNITS

Growth as determined by Equation 2 has units of grams prey consumed per gram striped bass per day ($g_{prey}*g_{bass}^{-1}*d^{-1}$). This unit of specific growth is not useful in determining the weight change of striped bass per unit time (i.e. dW/dt). To covert the units of gram prey per gram bass per day we employ a ratio (e_{dens}) of energy density in the prey (i.e. J/g_{prey}) to energy density in the striped bass (i.e. J/g_{bass}).

The energy density of fish is a measure of the energy content, essentially a caloric content, of that fish, often determined by bomb calorimetry (Hartman and Brandt 1995). This process is equivalent to the determining the

caloric content of a food item on a per gram basis. Therefore, in order to provide the necessary energy to grow the fish, we need to determine the energy content of the striped bass prey in relation to its own energy content.

By taking the ratio of prey energy (e_p) density to the striped bass energy density (e_s) and multiplying with G, we obtain units of grams striped bass per gram striped bass per day ($g_{bass} * g_{bass}^{-1} * d^{-1}$).

(12) $$G \times e_{dens} = \left[G \times \frac{e_p}{e_s} \right] = \left[\left(\frac{g_{prey}}{g_{bass} \times d} \right) \times \left(\frac{J/g_{prey}}{J/g_{bass}} \right) \right] = \left[\frac{g_{bass}}{g_{bass} \times d} \right]$$

ENERGY DENSITIES OF STRIPED BASS

From a literature and available data review, the energy density of striped bass can be obtained in several ways. The most straightforward approach, follows a striped bass bioenergetics model developed for Chesapeake Bay striped bass, estimated seasonal and age-specific energy densities for striped bass (Hartman 1995b). These energy density estimates were calculated from species-specific and generic models of fish energy density and seasonal means of percent dry weight for each age class with interpolation between observations (Hartman 1995b). This allowed for the development of simple linear regression for age 0 and age 1 striped bass and piece-wise linear functions for age 2 and age 3+ striped bass.

ENERGY DENSITIES OF PREY

Multiple sources of data exist for the energy densities of prey sources (Nelson 2006, Vatland et al. 2008, Hartman 1995a,b,c, Steimle and Terranova 1985, Chips and Bennett 2002, Pope et al. 2001). Some of these densities may fluctuate by season but are assumed constant for the interim.

COMPUTATION OF ENERGY DENSITIES OF MULTIPLE PREY TYPES

Similar to the coefficient of consumption term in Equation 1.10, the realized energy density of the prey depends upon the portion of that prey consumed. Functionally, this is the energy density of the prey type $e_{p,j}$, (J/g_{prey}), multiplied with the fractional consumption of prey type j.

(13) $$e_p = \sum_{j=1}^{n} e_{p,j} \left(C_j / C_r \right)$$

2.2.4 SPAWNING

Next, a component is added to adjust the weight of spawning females after spawning. The weight loss of the female is due to the release of eggs is assumed equal to the weight of the eggs. This can easily be obtained by multiplying the number of eggs released (as determined through the fecundity relationship) by the average weight of a striped bass egg. Since we are only interested in knowing the weight reduction due to spawning when the fish actually spawns, we will account for this reduction in the spawning module of IBM.

(14) $\mathbf{S_p=(F*W_{egg})}$

where,

S_p= total weight of eggs

F= fecundity (total # of eggs) per fish

W_{egg}= average weight of a striped bass egg

2.2.5 FINAL GROWTH FORMULATION

Therefore, returning to Equation 1.1, the weight at time *t* is the weight at time *t-1* plus the weight gain due to bioenergetics relationships minus weight loss due to release of eggs:

(15) $$W_t = W_{t-1} + (G \times e_{dens}) - S_p$$

or, expanding:

(15a) $$W_t = W_{t-1} + \left(\frac{1}{e_s} \sum_{j=1}^{n} e_{p,j} \left(C_j / C_r \right) \right) \left(C_{\max} \sum_{j=1}^{n} p_j - R - SDA - F - U) \right) - S_p$$

2.3 STRIPED BASS CONTAMINANT ACCUMULATION MODEL

Since contaminant concentrations, such as PCB's, vary temporally and spatially throughout the Bay-Delta, a contaminant accumulation/removal model is necessary to simulate the uptake process by striped bass. Additionally, this model needs to account for the uptake of contaminants via respiration and the uptake of contaminants via ingestion from contaminated prey. The uptake, accumulation and removal of contaminants by striped bass can be modeled following the approach of Thormann (1981). As mentioned above, this approach

utilizes a general mass balance where the change in contaminant concentration in a fish is equal to the amount of the contaminant absorbed from the water plus the contaminant concentration absorbed from the food minus the contaminant concentration eliminated by the body. The approach of Thormann (1981) is described below with several minor modifications.

(16) $$\frac{dv_{k,i}}{dt} = \eta_{uk,i}c + \alpha_{k,k-1,i}C_{k,k-1,i}v_{k-1} - v_k \mathrm{N}_{k,i} - v_k G_{k,i}$$

where, *k*= trophic level (e.g. *k* prey's upon *k-1*), *i*= striped bass life-stage, G_k= growth rate (d^{-1}), v_k= chemical concentration in the k^{th} trophic level, μg/g_k, η_{uk}= uptake sorption rate for the k^{th} organism, L/d-g_k, c= concentration of dissolved form of the chemical in water, μg/L, $C_{k,k-1}$= realized weight specific consumption of organism *k* by *k*-1, g_{k-1}/g_k, d^{-1}. α_k= desorption or excretion rate for the k^{th} organism, $\alpha_{k,k-1}$= assimilation efficiency, μg chemical absorbed/μg chemical ingested, t= time, d.

The growth rate (G) and weight specific realized consumption (C_r) are defined in similar fashion as the bioenergetic approach described in the above section. Additionally, Equation 1 is evaluated for every individual l at each life-stage *i*. For simplicity, the subscripts *i* and l have been omitted from the remainder of equations in this section.

We will not be modeling multiple trophic levels at this time and will only be considering trophic levels *k*=1 (i.e. striped bass) and *k*=0 (i.e striped bass prey), with v_0 inferred from data and not explicitly modeled. Therefore, the tropic level subscript notation has been dropped in most instances. This results in the formulation of Equation 16 as:

(17) $$\frac{dv_1}{dt} = \eta_{u1}c + \alpha C_r v_0 - v_1 \mathrm{N} + v_1 G$$

Where v_1 is the chemical concentration in the striped bass (μg/g_{bass}) and v_0 is the chemical concentration in the prey (μg/g_{prey}).

2.3.1 DOSE TERM EXPANSIONS

The terms in Equation 17 can be expanded, allowing for a better understanding of how to interpret and obtain each parameter as outlined below.

Uptake Sorption Rate: Environmental Sources

The uptake sorption rate (η_{u1}) of the contaminant by striped bass from the environment refers to how efficiently the fish (in each life-stage) uptakes a contaminant per unit time. This term implies that it is factorable into:

$$\eta_{u1,i} = \left[\frac{M_{cont\ uptook}}{M_{cont}\cdot}\right] \times \left[\frac{L}{t \times M_{fish}}\right]$$

Or, $\eta_{u1,i}$ = "contaminant efficiency uptake rate" (the massic mass of contaminant adsorbed per mass of contaminant ingested) multiplied by the "specific respiration rate" (volume of water through fish per time, per mass of fish).

UPTAKE SORPTION RATE: DIETARY SOURCES

The uptake sorption rate from dietary sources ($\alpha C_r v_0$) term from Equation 2.2 relates to the contaminant uptake by striped bass from their food sources at each life-stage. This term is more challenging and complex to model as the realized consumption term (C_r) varies temporally and spatially. Additionally, depending upon the type of prey, the concentration of the contaminant (v_0) varies (i.e. smaller prey such as plankton would typically have a lower contaminant burden then a small fish). Therefore, precise evaluation of these terms would require field data on the spatial and temporal distribution of striped bass prey *in addition to* the contaminant burden in these prey sources. Such data does not exist in the large-scale format needed to be beneficial to the model so some simplifications are necessary, which are described here subsequently.

For purposes of our IBM, I focus here upon PCB's but the approach should be applicable to other contaminants. Since contaminant distributions of suspended and sediment PCB's exist throughout the Bay-Delta we then want to relate this known dataset to the PCB burden found in striped bass prey. If we know the diet of a striped bass in a given age class from gut analysis (e.g. 50% prey 1, 50% prey 2) and we know the relationship between environmental PCB concentrations and the concentrations found in that prey (i.e. a biomagnifaction factor), we obtain the following relationship:

(18) $$\alpha C_r v_0 = \alpha \sum_{j=1}^{n} C_j v_{0j}$$

where, C_j is the weight specific consumption rate of prey type *j* ($g_{prey}*g_{bass}^{-1}*d^{-1}$) by striped bass and v_{0j} is the PCB concentration in prey type *j* (□g/g_{prey})

and,

(19) $$v_{0j} = k_j c_s$$

where, k_j is the specific uptake sorption for prey type *j* is L/g_{prey} or $g_{sediment}/g_{prey}$ and c_s is the sediment (or environmental) concentration of PCB's [□g/L (or □g/$g_{sediment}$)].

Combining Equations 2.3 and 2.4, yields:

(20) $$\alpha C_r v_0 = \alpha \left(\sum_{j=1}^{n} C_j k_j \right) c_s$$

The consumption by striped bass of prey *j* is identical to the consumption term in the previous bioenergetics section. The specific uptake sorption rate is assumed a constant whose value will be obtained from literature sources. Lastly, the assimilation efficiency is assumed a striped bass life stage dependant constant.

CONTAMINANT LOSS THROUGH EXCRETION

Returning now to Equation 2.2, the $v_1 K$ term implies the rate coefficient K_i can carry (as a constant of proportionality) any difference between excretion massic mass concentration of contaminant and body massic mass concentration of contaminant.

CONTAMINANT DILUTION THROUGH GROWTH

The final term in Equation 17 ($v_1 G$) relates to the dilution of the contaminant concentration in the fish due to growth. Specific growth rate, whether normalized per mass of fish or not, is not a constant and depends upon factors such as the age and size of the fish. The specific growth rate as formulated in Equation 17 is equivalent to the specific growth rate calculated under the bioenergetics approach and as such, for each model time step the specific growth rate is applied to Equation 17.

2.3.2 IMPLEMENTATION OF CONTAMINANT UPTAKE EQUATION

Several of the above terms in Equation 17 are treated as constants with their values obtained from laboratory experiments on striped bass. The values for constants currently obtained include, K_1= 0.0054 +/- 0.0008 h^{-1} (Pizza 1983) and □$_{10}$= 0.9 (Thomann 1981).

Both values of c and c_s change on a spatial and temporal scale. PCB contaminant data from multiple measurement sites (obtained from IEP and SFEI) throughout the Bay-Delta obtained from these sites can be interpolated over the spatial grid of the IBM, thereby providing the sources of data for these parameters.

2.3.3 LOSS/CHANGE IN CONTAMINANTS DUE TO SPAWNING

Only lipophilic (fat soluble) contaminants are cleared by striped bass females each year they spawn. Metals such as mercury accumulate through life and are not metabolized or excreted to any great degree. Males and young females (prior to 1st spawning) continue to accumulate all contaminants and don't clear/reset each year.

2.4 STRIPED BASS INDIVIDUAL BASED MODEL

To combine the outputs of the bioenergetics and dose accumulation models described above, we have developed an individual based model of the juvenile and adult life-stages of striped bass. At each time-step, the model considers six main sequential processes; (i) movement, (ii) growth, (iii) age, (iv) contaminant uptake, (v) mortality, and (vi) fecundity. The principal motivation for developing an IBM of the striped bass population in the Bay-Delta is to *explicitly* capture spatial heterogeneity in processes affecting fecundity and mortality at all life-stages.

2.4.1 MOVEMENT

The program uses a stochastic Monte-Carlo approach to simulate the movement of individual fish using the random-walk, run and tumble model. At every iteration in our model, the fish randomly 'chooses' a step length, turning angle, swimming time and a swimming velocity, all consistent with the random walk algorithm and given probability density functions. The probability density functions considered in the model are:

- Run time: a gamma distribution is adapted to model run-time. To bias the run and tumble model in the future, the parameters specifying the gamma distribution is considered to be a function of food and habitat gradient.
- Turn angle: A uniform distribution is used to generate random numbers representing turn angles.
- Velocity: A normal distribution of the mean and variance in fish size is used to generate velocities.

The selection of the step length, turning angle, swimming time and a swimming velocity can be biased whereby a fish may actively seek a more productive habitat or area of greater food availability.

2.4.2 GROWTH

Individual fish growth is determined by the bioenergetics model described in the above section.

2.4.3 AGE

The age of each individual fish is simply incorporated by the cumulative model time-steps. The age of the fish can be used to determine spawning potential where young female striped bass, less than 5 years on average, will have very low fecundity rates as they do not typically reach sexual maturity until age 5.

2.4.4 DOSE ACCUMULATION

Individual striped bass accumulation of contaminants (i.e. PCB's) is determined by the contaminant accumulation model described in the above section.

2.4.5 MORTALITY

Mortality is modeled as a Bernoulli process with its probability is described as a function of contaminant dose accumulated, age, and size. At every time-step a random number between zero and one is produced according to the uniform probability density function and its value is compared to the probability of the fish dying during one time-step, *P(Dose,Age).∆t*. If the value is smaller, the fish is considered dead and is removed from the system.

2.4.6 FECUNDITY

The California Department of Fish and Game's Central Valley Bay Delta Branch Sport Fish Unit performed an examination of adult striped bass fecundity (defined herein as total number of eggs produced per fecund female) during May 2005 (DFG 2005). A total of 27 striped bass over a four day period were collected from the fyke traps and three where collected from the gill net. Upon capture, each fish's fork length was recorded to the nearest cm, the right and left ovaries were removed and several scales were collected. Ages were assigned to each fish based upon growth increments from the collected scales. Total fecundity for each fish was assigned by multiplying the average number of eggs/gram of 2-3 subsamples from each left ovary by the total mass of both ovaries (DFG 2005). The collected field data allows for fecundity prediction based upon the fish's age, fork length or age and fork length. To determine which independent variable was the best predictor of fecundity several different regression models were tested and the best-fit regression model for fecundity proved to be an age versus fork length regression.

It is worthwhile to mention that the IBM model described above has been formulated in a generic fashion to allow for expansion of additional biological processes and to apply a similar formulation to other fish species. There are numerous species specific parameters that are utilized by this model, but the basic framework applies to all POD species.

3.0 THREADFIN SHAD SPECIFIC FISH MODELS

3.1 THREADFIN SHAD BIOENERGETICS MODEL

Vatland et al. (2008) developed a bioenergetics framework to model threadfin shad and striped bass dynamics in Lake Powell, Utah/Arizona. Specifically, they utilized 20 years of historical data on temperature, diet, growth, and abundance of these fish, collected similar data on a finer scale in 2003–2004, and used components of this data set to develop specific conversions between coarser historic data and present data (Vatland et al. 2008). Their aim was to determine consumption of threadfin shad by striped bass and determine the how this prey/predator interaction influences the population of both species. The model resulted in an apparent explanation of 53% of the variation in condition of striped bass (Vatland et al. 2008). The modeling approach of followed by Vatland et al. would likely be applicable to the threadfin shad in the Bay-Delta with minor modifications. Other general bioenergetic modeling approaches, such as that developed in (Hanson et al. 1997) would also be applicable to the Bay-Delta system, assuming the necessary species-specific parameters are available. Siebring (2002) has developed a bioenergetics model for gizzard shad based upon the general bioengergetics model developed by Hanson et al. (1997). While not identical species, parameters used to develop the gizzard shad model may aid in developing a bioenergetics model for threadfin shad.

4.0 LONGFIN SMELT SPECIFIC FISH MODELS

4.1 LONGFIN SMELT BIOENERGETICS MODEL

Lantry and Stewart (1993) developed a bioenergetics model on rainbow smelt in the Great Lakes area following a similar approach as Hanson et al. (1997). Diet composition was estimated on a seasonal basis from gut analysis and published diets of smelt throughout the Great Lakes area. For each lake, Lantry and Stewart (1993) used specific temperature regimes, growth rates, mortality rates, and abundance estimates. There would be several problems in applying these parameters to longfin smelt in the Bay-Delta, the first and foremost being that these are two different species. Additionally, it is likely that new diet evaluation would be needed to reflect the different species dynamics and prey availabilities. This model (and associated parameters) would be useful as guide for the needed data and could possibly used as a first pass approximation into longfin smelt population dynamics.

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APPENDIX IV

REVIEWER COMMENTS AND ANNOTATIONS

TABLE OF CONTENTS

INTRODUCTION

To guide the development of this report, an expert panel was convened to review and comment on the approach, development, and conclusions drawn. The four members of the Expert Panel included Dr. Susan Anderson – independent consultant (lead), Dr. Jeffrey Miller – AquaScience, Inc., Dr. Debra Denton – US EPA Region IX, and Dr. Lisa Thompson – UC Davis Cooperative Fisheries Extension Specialist. In addition, formal comments were provided by three staff from the Central Valley Regional Water Quality Control Board; Dr. Christopher Foe, Mr. Jerrold Bruns, and Ms. Stephanie Fong. Dr. Anderson worked with Dr. Johnson in the early stages of report writing, and again in the final stages of completion. As a result, two sets of comments were provided by Dr. Anderson. The remaining reviewers provided comments on the final draft only.

Comments provided by the reviewers are often accompanied by a page and paragraph number. The revisions made to the report changed the length and consequently, the comments no longer refer to the page number listed. In some of the responses to comments, the current page number of the modified text is included, in other responses it is not.

The annotation of comments was done by providing a response to each comment provided by the reviewers. All responses were made by the senior author. No attempt was made to group comments with similar themes from the different reviewers. In a few instances, the reviewers made conflicting requests for changes in the document. In these instances, a decision was made as to which reviewer's comments were followed. The decision was generally based on the reviewer's expertise. Each response is provided in italics immediately following the comment.

Several reviewers also provided copies of the report with a series corrections embedded in the track changes mode or provided as written notes on hard copies of the report. The vast majority of these corrections are minor addressing issues such as grammar (e.g. verb-tense agreement) and spelling, or indicating that table numbers in the text did not refer to the correct table, or that figure legends needed to be expanded, etc. These minor comments were then accompanied by the more formal review which repeated some comments in the margin of the report. In other instances reviewers did not include the comments embedded in the text in the formal review. None of these minor comments are addressed in the annotated response to comments in this appendix. To do so would require scanning the hard copies with comments into electronic form to attach to this appendix, and also embedding the track changes comments into this appendix. It was determined that the corrections were minor, they did not address the organization, development of the arguments, or the conclusions drawn in the report, and would not improve understanding of the report. The minor comments were not included in this appendix.

The reviewers were given a very short time to read and review the document, and in some instances there were a very few spelling errors or abbreviations used in the reviews that could be difficult to understand. Although the reviewer's comments are unaltered with respect to content and tone, in the reviewer's comments simple misspellings were corrected and some abbreviations expanded. These changes do not in any way alter the meaning or the tone of the comment and in no way changed the response provided.

COMMENTS OF SUSAN ANDERSON

POD CONTAMINANTS REPORT COMMENTS: INITIAL DRAFT

Susan L. Anderson 8/28/09

FOCUS OF REVIEW AND KEY CONCERNS

This review was focused on the following key questions:

- Does the structure of the document make sense relative to the goals?
- Were the data sources clearly identified and was the data search complete?
- Were criteria for including or excluding data clearly identified?
- Was the approach to data analysis sound?
- Were data summarized effectively?
- Did the conclusions follow from the findings?
- What is the most constructive suite of initial revisions given tight resources?

My synthetic conclusions to each of these points are provided below in Section 1, followed by a lengthy section comprised of more specific comments (Section 2). The latter are presented in the order they appear in the report.

Overall my impression is that so much effort went into creating the database, that an insufficient budget was available for analysis. Since my charge is not to comment on funding *per se*, I have tried to simply suggest the most constructive, realistic, and specific comments possible for this first draft. It is a great accomplishment to have created the database and completed this initial summary.

SECTION 1: SYNTHETIC CONCLUSIONS ON KEY CONCERNS

Report Structure

I am suggesting some simple revisions that might make the document more readable. Pleaser refer to the attached diagram that I have hand-drawn and scanned in. I propose you include something like this as a figure. Here is a summary of what I suggest:

- First 2 pages would be one section called “Goal and Approach
- Section 2 would be entitled “Background” and begin with POD decline and go through the bulleted summary on p.15.
- Section 3 would begin where you start to discuss hypotheses and the section could be called “structure of hypotheses”
- Section 4 would be “methods of dataset development and data analysis” and datasets for chemistry, toxicity, and histopathology should be presented followed by a description of database development.
- Section 5 would be “data analysis” and also would have three sections: chemistry, toxicity, histopathology

- Section 6 would be conclusions
- The modeling section is not tied to the report in any way and can be submitted as an appendix

Please consult the diagram for more details.

RESPONSE: ORGANIZATIONAL CHANGES MADE AS REQUESTED.

A second key point about the report structure is that the decision to use Koch's Postulates as an "organizing principle" is understandable but it results in a rather "wordy" approach to what is really a straightforward toxicologic analysis of exposure and effects. I would make a serious effort to edit down the words and present more in tables, bulleted lists or simple figures. I would also edit the text heavily for redundancy. This will be easier to do after the structure and analyses are cleaned up. Also, I would focus on enhancing the toxicologic interpretation which is weak. I make some suggestions below. Also please see below important suggestions about the section on "necessary vs. sufficient" and on consistent analysis of structural comparisons.

RESPONSE: KOCH'S POSTULATES HAVE BEEN REMOVED FROM THE FINAL DRAFT AS AN ORGANIZING PRINCIPLE.

Data Sources

Overall, the report is inconsistent in its description of data sources and criteria for using them. The data sources used for toxicity testing are clearly presented in the "analysis section". However, summary text is needed in the proposed "methods section"

RESPONSE: A SLIGHTLY EXPANDED EXPLANATION OF DATA SOURCES HAS BEEN INCLUDED IN THE METHODS SECTION. A MUCH EXPANDED VERSION OF DATA SOURCES IS NOW INCLUDED AS APPENDIX I, APPENDIX II, AND APPENDIX III.

The chemistry data are obviously numerous but to my knowledge the datasets are not identified. Criteria for including or excluding any data are not laid out. This would be an important revision. It was not possible to assess whether this data search was complete given the lack of detailed information.

RESPONSE: THE CRITERIA FOR INCLUSION IN THE ANALYSIS ARE NOW INCLUDED AS BULLET POINTS ON PAGES 13 AND 14.

The histopathology datasets should have been clearly identified in a Table if they could not be included in the database for some reason. The rationale for a chemical by chemical review for other findings is unclear, even distracting. In my opinion, this section could be treated like the others with key data summarized in tabular form and an analysis of conclusions.

RESPONSE: THE HISTOPATHOLOGY SECTION HAS BEEN REORGANIZED AND THE CHEMICAL BY CHEMICAL APPROACH HAS BEEN ELIMINATED. A MORE INCLUSIVE ORGANIZATIONAL STRUCTURE IS NOW IN PLACE.

Criteria for Data Inclusion/Exclusion

A simple table or bulleted list of criteria should be included in each of the sections: chemistry, toxicity testing, histopathology.

RESPONSE: THE CHEMISTRY DATA SETS ARE PROVIDED IN APPENDIX I, THE TOXICITY DATA SETS ARE PROVIDED IN APPENDIX II WITH THE RESULTS FROM DIFFERENT PROGRAMS PROVIDED IN SEPARATE TABLES. THERE ARE NO HISTOPATHOLOGY DATA AVAILABLE. ALL HISTOPATHOLOGY DATA ARE TABULATED IN THE ORIGINAL REPORTS USED IN THIS SYNTHESIS.

Data Analysis Approach

I outline below some important concerns about the data analysis. For the chemistry section, the selection of the somewhat arbitrary approach of using poorly documented data on Cerios and Hyalella needs more justification. The exclusive use of acute values also should be justified. In a regulatory setting, acute values are used to designate a maximum instantaneous exposure. Please see the discussion below for copper for more details.

RESPONSE: THE USE OF THE CERIODAPHNIA AND HYALELLA AS THE FRAMEWORK FOR THE ANALYSIS WAS ABANDONED IN FAVOR OF THE CUMULATIVE FREQUENCY DISTRIBUTION APPROACH. THE FOCUS ON ACUTE VALUES IS BASED ON THE REGIONAL BOARD'S COMPILATION OF WATER QUALITY GOALS WHICH PROVIDES THE CONCENTRATION THAT TRIGGERS REGULATORY ACTION IN SOME REGIONAL BOARD PROGRAMS. BECAUSE AN APPROPRIATE ACUTE OR CHRONIC VALUE WAS IMPOSSIBLE TO ESTABLISH DUE TO A LACK OF TOXICITY DATA RELEVANT TO THE POD SPECIES, THE WQGS WERE DETERMINED TO BE THE MOST APPROPRIATE VALUES TO SET AS BENCHMARKS FOR COMPARISON. TABLE 4 WAS ADDED TO PROVIDE THE EPA'S OPP AQUATIC LIFE BENCHMARK TOXICITY VALUES FOR NUMEROUS CHEMICALS AND TABLE 6 WAS ADDED TO PROVIDE TOXICITY BENCHMARKS FOR SOME SPECIES IN THE DELTA, BUT NO POD SPECIES WERE INCLUDED.

For the toxicity testing section, it was unclear how the sediment TUs were derived. I couldn't analyze this information further without understanding what was done.

RESPONSE: THE SEDIMENT TUS WERE ELIMINATED FROM THE FINAL DRAFT.

The analysis was interesting in its attempts to address the key structural comparisons such as focus on: winter/spring, early life stages, delta habitat, and 00-02 vs. 03-08. If the Koch's postulate approach is to ultimately succeed it will require tabular comparisons or bulleted summaries relevant to these points in each section (chemistry and toxicity anyways). This is where synthesis and improved toxicologic interpretation could be very meaningful. For example, time course and seasonal comparisons could be achieved with summary figures, spatial summaries/synthesis could be presented on figures or tables and toxicity benchmark values for fish early life stages and fish prey could be gleaned from the literature for a few example chemicals. For example, the copper criteria document would be a useful source of information.

RESPONSE: ALTHOUGH THE INFORMATION ON KOCH'S POSTULATES AS AN ORGANIZING PRINCIPLE WAS ELIMINATED, BULLETED SUMMARIES FOR EACH SECTION WERE DEVELOPED. TABLES OF

AVAILABLE TOXICITY BENCHMARKS WERE PLACED INTO THE TEXT. THE COPPER CRITERIA DOCUMENT WAS USED AS A SOURCE FOR THE COPPER SECTION WHICH WAS REVISED COMPLETELY.

The text should be edited to improve toxicologic interpretation. For example, there is a large emphasis on numbers of chemical detections or frequency of toxicity but spotty emphasis on chemical concentrations and levels of toxicity.

RESPONSE: THE TEXT WAS REVISED TO ADDRESS THE CONCENTRATIONS OF THE CHEMICALS DISCUSSED. THERE IS ALMOST NO INFORMATION AVAILABLE ON THE TOXICITY OF CHEMICALS TO THE POD SPECIES AND THIS SECTION REMAINS A DATA GAP IN OUR OVERALL KNOWLEDGE OF THE EFFECTS OF CHEMICALS ON FISH IN THE DELTA.

Effectiveness of Data Summary

As stated above, there is not enough synthesis of data. The toxicity section should show summaries of key findings in tabular/graphical form, comparisons among years, seasons or locales etc. Tables 1 and 2 in the chemistry section should include the range of chemical concentrations observed compared to toxic levels or half life. This might also be used to discuss chemical fate in your rationale for use of only acute values. Then summary figures could also be presented to draw conclusions about the structural comparisons as stated above. These things are always easier to do once an initial draft is in place! Also, please consider whether a section on the significance of toxic resuspended sediments can be added. Inge could do this.

RESPONSE: TABLE 5 WAS ADDED TO FOCUS ON THE WQG FOR SEVERAL SPECIFIC CHEMICALS AND INCLUDED A COLUMN REPORTING HALF-LIFE FOR THOSE CHEMICALS WHEN AVAILABLE. AN EXPANDED SYNTHESIS SECTION WAS ADDED TO THE REPORT THAT INCLUDED A SUMMARY OF AVAILABLE DATA AND THE RESULTS OF THE HYPOTHESES/QUESTION. NO ADDITIONAL VERBIAGE WAS ADDED TO THE SECTION ON THE RESUSPENSION OF SEDIMENT.

Conclusions

It seems to me the conclusions do not follow in a structural way from the findings. I may be too quick to judge but it seems they essentially say that there is not enough data and then provide key points for future study. I think there should be a point by point summary of the most significant toxicity and chemistry findings in an accessible format. I think the existing data show that there is a significant potential for contaminants to be involved in maintaining the decline and that there are adequate data to be used in assessing the issue in a more targeted manner. The data you show certainly cannot be used to take toxics OFF the table so I think that means they are on the table. I think you should not get bogged down in the words around the Koch's postulates and "necessary and sufficient" etc. Just summarize the key data systematically. Build on the Koch's approach by referring to the structural comparisons as feasible. When this is all reworked I would be happy to help you with a serious edit of the conclusions.

RESPONSE: A SET OF SPECIFIC CONCLUSIONS WAS ADDED TO THE END OF THE CHEMISTRY, TOXICITY, AND HISTOPATHOLOGY SECTIONS. AS INDICATED ABOVE, THE KOCH'S POSTULATES APPROACH WAS ABANDONED AS WAS THE EXPLANATION OF NECESSARY AND SUFFICIENT CONDITIONS.

SECTION 2: SPECIFIC COMMENTS FOR EACH SECTION

Approach

To accomplish their goal, the authors implement Koch's postulates to integrate basic toxicologic assessment of chemical exposure and biological effects with the natural history of the POD species. The first time I read the document, I liked the approach. However, when I reread it, I noted some complications and possible inconsistencies. Many of my comments relate to an attempt to be constructive in tightening up the broad approach. In many respects, a traditional toxicologic analysis of exposure and effects data (without an additional overarching framework) would have been more straightforward, but the use of Koch's postulates does provide a bridge to the ecological framework of the POD decline. The PI deserves credit for attempting an integrative approach. It simply should be tightened up and the toxicologic interpretations given more depth. It should be used to structure the report and hypotheses but not to develop decision criteria for supporting conclusions. For example the "necessary vs sufficient" section just doesn't fly. The straightforward toxicology findings get lost.

One initial question is how can Koch's postulates be rephrased to address toxicity to prey species, which is discussed in some depth in the report?

RESPONSE: KOCH'S POSTULATES ARE NO LONGER USED TO STRUCTURE THE ANALYSES AND THE DISCUSSION OF NECESSARY AND SUFFICIENT IS NOT INCLUDED IN THE FINAL REPORT.

POD decline

Summarize which 3 species pass the criteria for step decline. Is it LS that is eliminated?

The last paragraph on effects on prey should be edited down and clearly reference the broader discussion of this topic which follows a few pages later. When you initiate a discussion of the topic here, one wants you to go further but then finds it is covered later.

RESPONSE: A MORE DETAILED DISCUSSION OF THE POPULATION DYNAMICS OF THE POD SPECIES IS INCLUDED IN A SECTION TITLED POS AND NON-POD SPECIES POPULATION DYNAMICS ON PAGES 7-10. THE THREADFIN SHAD IS THE SPECIES THAT IS EXPERIENCING A SLIGHT INCREASE IN ABUNDANCE.

POD species biology

The introduction to this section does not set up the key points that are raised and should exactly echo the key points in the bulleted summary of this section. The bulleted summary is fairly strong.

RESPONSE: A BULLETED SECTION IN THE SECTION LIFE HISTORIES WAS ADDED TO ESTABLISH THE KEY POINTS OF THE SECTION.

Life cycles, 3rd paragraph needs strong topic sentence emphasizing focus on early life stages.

RESPONSE: THE SECTION WAS REORGANIZED AND THE THIRD PARAGRAPH IS NOW THE FIRST PARAGRAPH OF A SECTION TITLED "POPULATION GROWTH RATES (λ)". THE TOPIC SENTENCE NOW EMPHASIZES THE JUVENILE STAGE AS THE CRITICAL STAGE CAUSING POPULATION DECLINES.

Your statement that the focus on spring makes sense is weak. This is a critical (and logical) conclusion. Please strengthen.

I am unclear throughout the report whether you believe that early life stages are also the important stage for SB. Can you please clarify and make consistent throughout.

RESPONSE: THERE IS NOW A SECTION TITLED "HABITAT AND TIMING" THAT CLEARLY ESTABLISHES WHY THE OVERLAP OF THE POD SPECIES IN TIME AND SPACE IS CRITICAL. IT ALSO STATES THAT THE ONLY OVERLAP IN SPACE AND TIME IS THE SPRING WHEN ALL SPECIES SPAWN IN THE DELTA. TABLE 1 HAS BEEN INSERTED TO SUMMARIZE THE LIFE HISTORY CHARACTERISTICS OF THE POD SPECIES INCLUDING WHEN THEY WOULD BE SUSCEPTIBLE TO ACUTE TOXICITY. STRIPED BASS ARE INCLUDED AND THERE IS REFERENCE TO THE POTENTIAL EFFECTS ON JUVENILE STRIPED BASS.

State clearly that focus on the winter/spring is a major pillar of the analysis.

RESPONSE: STATED, SEE EXPLANATION ABOVE.

POD vs non POD

Paragraph 1- make numbered sections flow in the same order as the report.

RESPONSE: THE REPORT HAS BEEN REORGANIZED TO ELIMINATE THE NUMBERING ORGANIZATIONAL STYLE.

Paragraph 2- Differential sensitivity is toxicology dogma. There is never ONE most sensitive species to all compounds. What is important is whether the species of interest are sensitive to the compounds of concern. Any relevant data on sensitivity of POD species to chemicals listed as relevant should be summarized.

RESPONSE: THERE ARE NO DATA ON THE SENSITIVITY OF THE POD SPECIES TO ANY CONTAMINANTS. RECENT WORK ON THE SENSITIVITY OF DELTA SMELT TO CONSTITUENTS LIKE AMMONIA BY INGE WERNER WERE RELEASED AFTER COMPLETION OF THIS REPORT.

Paragraph 3-4- Why not include a table summarizing prey for POD and non-POD with a comparison of toxic levels for the 3 most relevant compounds in your analysis. Okay, maybe that's unrealistic but think about a way to take this a bit further. You last sentence should be deleted.

RESPONSE: THE ENTIRE SECTION HAS BEEN REORGANIZED AND THERE IS NOW A DISCUSSION OF THE DIETS OF POD AND NON-POD SPECIES IN THE SECTION TITLED "POD AND NON-POD SPECIES POPULATION DYNAMICS" IN SUFFICIENT DETAIL TO DOCUMENT DIFFERENCES IN DIET. UNFORTUNATELY, THERE ARE INSUFFICIENT DATA AVAILABLE TO COMPARE THE TOXICITY OF VARIOUS CONSTITUENTS TO THE PREY ITEMS OF THE DIFFERENT POD AND NON-POD SPECIES. THE LAST SENTENCE WAS DELETED IN THE REORGANIZATION OF THE SECTION.

Topsmelt discussion paragraphs should be a stand alone subsection with appropriate subheading.

RESPONSE: THERE IS NO DISCUSSION OF TOPSMELT IN THE REPORT. IT IS ASSUMED THIS COMMENT REFERS TO THREADFIN SHAD. THERE IS A SECTION TITLED "POD POPULATION INCREASES – THREADFIN SHAD PHENOMENON."

The bulleted summary is very good. Please add one more bullet (in second place) stating that you think a focus on winter/spring is important.

RESPONSE: BULLET POINT ADDED.

As mentioned above, a new section should begin here entitled something like "Structure of Hypotheses". The hypothesis statement should be edited to include "sufficient to cause and/or maintain…."

RESPONSE: NEW SECTION ADDED AND THE WORDING WAS INCORPORATED INTO THE HYPOTHESES.

The final three pages of this section are troublesome. I don't think the discussion of necessary and sufficient works. Perhaps it could be deleted but more discussion may be needed.

RESPONSE: THE DISCUSSION OF NECESSARY AND SUFFICIENT CONDITIONS WAS DELETED FROM THE FINAL DRAFT.

Historical Perspective

Starting at this point add a new 1st order subheading called "Analysis" (see diagram).

Then history section should be moved out, maybe to the background section?

RESPONSE: A NEW SECTION TITLED "ANALYSIS AND METHODS" HAS BEEN ADDED TO THE REPORT. A SECTION TITLED "TIME PERIOD COMPARISON" WAS ADDED AS A SUBSECTION TO THE SECTION "STRUCTURE OF HYPOTHESES". SEVERAL OTHER ASPECTS OF THE HISTORICAL ANALYSES WERE PLACED IN THE APPROPRIATE SECTION, E.G. THE HISTORICAL INFORMATION ON WATER CHEMISTRY WAS PLACED INTO THE SECTION "WATER CHEMISTRY DATA." SEE NEXT COMMENT FOR MORE DETAILS.

The history section is useful, and I understand it is meant to be brief. However, it could be tightened up. My perception is that it is either poorly referenced or completely dependent on the Fox and Archibald (1997) reference. I am not familiar with the article. Is it solid? Also, this section emphasizes percentages of samples showing detections of chemicals and toxicity, but it is the concentrations of chemicals and levels of toxicity that are most relevant in many respects. Consider adding more references and a table with representative data.

RESPONSE: THE HISTORICAL DATA ARE NOW PLACED IN THE APPROPRIATE SECTION; HISTORICAL WATER CHEMISTRY DATA WITH THE WATER CHEMISTRY SECTION, ETC. UNFORTUNATELY THERE ARE VERY FEW AVAILABLE HISTORICAL DATA FROM THE PRE-POD PERIOD AND THE ANALYSIS RELIES HEAVILY ON THE FOX AND ARCHIBALD (1997) REFERENCE AND THEIR GATHERING OF AVAILABLE DATA. THE BUDGET DID NOT ALLOW TIME TO ACQUIRE THE PRIMARY LITERATURE AND REPORTS REFERENCED BY FOX AND ARCHIBALD NOR DID IT ALLOW FOR A SEARCH FOR ADDITIONAL DATA AND REPORTS. CONSEQUENTLY, THE DISCUSSION OF THE HISTORICAL DATA RELIES HEAVILY ON THAT REFERENCE. THE INTERPRETATION OF FOX AND ARCHIBALD (1997) WAS NOT USED IN THE ANALYSIS; ONLY THE DATA

FROM TABLES IN THE REPORT WERE USED. SOME OF THOSE DATA WERE SUMMARIZED IN TABLES IN THE CONTAMINANTS SYNTHESIS REPORT E.G. TABLES 2-4.

Water Chemistry Datasets and Database Development

These two subsections are essentially the "Methods Section" of the report. There was an enormous amount of work done to assemble the data! The data gathering and database development alone could easily justify much of this budget, at least as far as I understand the context here. At any rate, as I discuss above, I think the "Methods" should be more specific and detailed. First, I think there should be four subsections: 1) Chemistry datasets, 2) toxicity datasets, 3) histopathology and 4) database development. Regarding water chemistry, can you be more specific, in a sentence or two, what constitutes "sufficient data"? Also, I must be missing something but I really don't see a list of data sources or reference to such an appendix. It just says there are 37 projects. Nor do I really see a list of criteria for including or excluding data. It is critical to add/clarify this. Toxicity testing datasets are defined throughout the text but a brief section here would summarize this for the reader. A bulleted list or table and brief text would suffice.

RESPONSE: THE SUBSECTIONS HAVE BEEN PLACED INTO THE FINAL DRAFT. THE ORGANIZATION IS THAT "DATABASE DEVELOPMENT" IS THE FIRST SUBSECTION FOLLOWED BY THE OTHER THREE SUBSECTIONS. WITHIN THE SUBSECTION "WATER CHEMISTRY DATA" THERE ARE BULLET POINTS THAT IDENTIFY WHAT ARE CONSIDERED SUFFICIENT DATA FOR INCLUSION IN THE ANALYSIS. THE LIST OF DATA SOURCES IS NOW INCLUDED IN APPENDIX I. THE TOXICITY DATASETS USED ARE SUMMARIZED IN APPENDIX II, AND ANY TOXICITY DATA GENERATED BY A LABORATORY FOLLOWING STANDARD EPA PROTOCOLS WAS CONSIDERED OF SUFFICIENT QUALITY FOR INCLUSION IN THE ANALYSIS. ESSENTIALLY, ALL TOXICITY DATA IDENTIFIED WAS USED IN THE ANALYSIS.

The histopathology section later in the report is weak. Why are there no lists of datasets available and a straightforward analysis? It would be great if datasets (very few) were identified and loaded into this database if format allows. Otherwise, a table with all references could be provided as an appendix. Otherwise, there is no real analysis of histopathology (more below).

RESPONSE: NO RAW HISTOPATHOLOGY DATA EXIST. ONLY A FEW HISTOPATHOLOGY STUDIES HAVE BEEN PERFORMED AND THE DATA ARE MAINTAINED BY THE INDIVIDUAL INVESTIGATORS WHO GENERATED IT. THERE IS NO STANDARD FORMAT FOR THESE DATA AND CURRENTLY, NO PUBLICALLY AVAILABLE DATABASE STRUCTURE IN WHICH TO PLACE THESE DATA. GENERATING SUCH A DATABASE STRUCTURE WAS BEYOND THE SCOPE OF THIS PROJECT.

Finally, I think the database development should be described in a much more positive light. This was an important accomplishment. Yet, the "methods" begin with a weak statement about limited data. Maybe that needs to be emphasized later, but I would read through this section and think of ways to describe the database development that are not longer but are brief, positive, and specific.

RESPONSE: THE FIRST SENTENCE OF THE SECTION "DATABASE DEVELOPMENT" NOW EMPHASIZES THE ACCOMPLISHMENT OF ASSEMBLING THE DATA FROM NUMEROUS DATA SOURCES.

Analysis of Chemical Data

A lot of interesting information is presented here. Again, it is a great accomplishment to bring this information together. However, I have a few overarching concerns.

I think it would be important to provide more justification for your general approach. First, the use of 1TUa for either Cerios or Hyalella for the insecticides should be justified with appropriate references. Your rationale states that you use this approach for consistency so you can compare across taxa, but this does not seem like the most important point. Rather, I would like to know what action limits are available for each of these compounds and then, based on chemical half life and potential for exposure select acute or chronic values. The approach you have picked is not conservative. However, if the reader had references and some data indicating that for the vast majority of chemicals listed, these were the most sensitive acute values (and that half-lives were certainly less than 4 days), then the approach is more convincing. The approach for herbicides might make sense, but similarly it should be justified.

RESPONSE: THE USE OF THE TU APPROACH WAS ELIMINATED FOR THE FINAL DRAFT. THE APPROACH WAS SHIFTED TO EVALUATING THE RELATIVE NUMBER OF SAMPLES WITH CONCENTRATIONS ABOVE AND BELOW THE WATER QUALITY TRIGGER LIMITS AS DEVELOPED BY THE STATE BOARD AND ALLOWING THE READER TO DETERMINE IF THE RELATIVE FREQUENCY WAS SUFFICIENT TO BE PROBLEMATIC. BECAUSE SOME MIGHT BELIEVE THAT 5% OF THE SAMPLES EXCEEDING THE WQTL IS SUFFICIENT TO INDICATE THAT THE CONTAMINANT OF CONCERN COULD CAUSE THE POD, WHILE ANOTHER MIGHT BELIEVE THAT 5% IS NOT SUFFICIENT, IT IS DIFFICULT TO MAKE JUDGMENTS ABOUT THE IMPORTANCE OF CHEMICALS IN THE POD. WITH ALMOST NO DATA ON THE TOXICITY OF THESE CHEMICALS TO THE POD SPECIES, IT IS VERY DIFFICULT TO DRAW CONCLUSIONS ABOUT THE ROLE OF CHEMICALS IN THE POD. AND GIVEN THE OVERALL LACK OF DATA ON THE EFFECTS OF MULTIPLE TOXICANTS ACTING JOINTLY ON POD SPECIES, OR ANY OTHER SPECIES, IT IS NEARLY IMPOSSIBLE TO DETERMINE THE POTENTIAL EFFECTS OF CHEMICALS ON THE POD SPECIES. HOWEVER, IT IS NOT REASONABLE TO STATE THAT LOW CONCENTRATIONS OF SEVERAL CHEMICALS WOULD BE PROBLEMATIC WHEN THERE ARE NO DATA TO SUBSTANTIATE SUCH A CLAIM.

An additional concern I have is that the assumption that an acute value was a good benchmark and this assumption is not justified. As described above, if Tables 1 and 2 were moved forward and three new columns were added listing range of chemical concentrations observed toxic levels, and chemical half life, then these could be discussed upfront to frame the discussion. If you discuss the half-lives and likely dilution in the Delta (just one or two paragraphs), then use of acute values might be justified but I feel the approach is not well described and certainly not conservative.

RESPONSE: THE ANALYSIS WAS FOCUSED ON VARIOUS WATER QUALITY GOALS WHICH ARE A COMBINATION OF BASIN PLAN OBJECTIVES, WATER QUALITY GOALS, OR WATER QUALITY LIMITS. A DISCUSSION OF THE USE OF THESE VALUES IS INCLUDED ON PAGES 19 AND 20 OF THE SYNTHESIS REPORT. BECAUSE THESE VALUES ARE ASSUMED TO BE PROTECTIVE OF AQUATIC LIFE, THEY ARE PRESUMABLY PROTECTIVE OF THE POD SPECIES ALTHOUGH THE ASSUMPTION HAS NOT BEEN EVALUATED (SEE ABOVE). TABLE 5 WAS ADDED WHICH CONTAINS A COLUMN FOR THE WATER QUALITY GOAL AND THE HALF-LIFE. A DISCUSSION OF THE CONCENTRATION OF SPECIFIC CHEMICALS AND THE POTENTIAL EFFECTS OF THE CHEMICAL ON POD SPECIES IS PLACED INTO THE CONTEXT OF THE

HALF-LIFE. DILUTION WAS NOT CONSIDERED TO BE AN ISSUE BECAUSE THE SAMPLES WERE COLLECTED FROM THE DELTA AFTER DILUTION FROM THE SOURCE.

I would like to use the copper section as an example of why I am concerned about all of the chemistry sections. I think the use of 31ug/l is an error. The 2002 EPA criteria document for copper lists the species mean acute value for copper as 13.99 for C. dubia (Table 3 of the document). The chronic value is lower. According to the figure for dissolved copper, exposure could easily have been chronic as numerous values exist in the 10-20ug/l range over a significant period of time. Why would an acute value be used for this? Were the most authoritative references used? Copper also provides an opportunity to discuss the key structural comparisons. For example toxicity of copper to early life stages of fish could be compared to levels observed in the Delta. I think this would be more relevant than presenting the cumulative frequency distribution from Rogers, 2005. At a minimum, data from the criteria document could be summarized in a small table. Also, are there sufficient data for a winter/spring versus summer/fall comparison? Were there any geographic areas that were frequently high? In essence, you spent a lot of time and effort to develop the key hypotheses so unfortunately you must follow through and really examine them where feasible. I know it is daunting but it is the logical conclusion.

RESPONSE: THE COPPER SECTION WAS REWRITTEN AND THE ANALYSIS WAS PERFORMED USING THE INFORMATION IN THE 2002 EPA COPPER CRITERIA DOCUMENT. THE EFFECT OF THE DISSOLVED CARBON WAS INCORPORATED INTO THE ANALYSIS. THE CUMULATIVE FREQUENCY DISTRIBUTION FROM ROGERS (2005) WAS NOT USED. THE FORMULAS USED IN THE US EPA COPPER CRITERIA DOCUMENT WERE USED TO DETERMINE THE CRITICAL VALUE OF COPPER FOR TOXICOLOGIC EFFECTS IN THE DELTA.

Another thing I am confused about in your general approach is how TUa for sediment are derived. Are there actual data for spiked sediment? Were there references for this or am I just missing something? I couldn't analyze the sediment- related data further without a better understanding. For example, when you say 20 of 62 bifenthrin samples contained measurable concentration/Toxic units, I don't really know what you mean.

RESPONSE: SEDIMENT TUS ARE NO LONGER INCLUDED IN THE REPORT.

Edit the last few pages of this section with more emphasis on chemical levels rather than detections and provide more synthesis as discussed above.

RESPONSE: EDITED AS REQUESTED.

Single species toxicity tests

Most of my thoughts on this section are covered in the comments above on general structure and analysis. I will try to read this again by Monday to make some more specific suggestions but I think it first needs to be edited with more summary of data and use of structural comparisons. I guess my feelings are that the approach is valid but a rewrite/synthesis is needed.

Histopathology

I have commented on this above. The POD species section should come first and include real data tables. When that section is edited with an explicit reference to the structural hypotheses (type and level of

effect, differences among species and life stages, winter/spring vs. summer fall, POD species vs. non POD), then it would be logical to look at the chemical by chemical summary and see what it adds to the overall section as a discussion.

RESPONSE: THE HISTOPATHOLOGY SECTION WAS REWRITTEN ALTHOUGH REAL DATA WERE NOT AVAILABLE TO USE IN THIS REPORT. THE CHEMICAL BY CHEMICAL APPROACH WAS REPLACED BY A DISCUSSION OF THE HISTOPATHOLOGY DATA ON POD SPECIES SPECIFICALLY.

Fish population section

This section has not been linked to the rest of the report. Since there is a lot to do to refine the main report, I think this should be included as an Appendix.

RESPONSE: THE FISH MODELING SECTION IS NOW INCLUDED AS APPENDIX III.

Synthesis and conclusion sections

As stated above, these can be rewritten after there has been more synthesis to emphasize the structural comparisons and to include more accessible data summaries. I can review this more carefully at that time.

Format editing

For the final draft, I assume you will are planning on the following:

Executive summary
Table of contents
Preface/acknowledgments
References
Improved Figure legends
Finalized figures
Consistent identification of abbreviations (e.g. MDL, RL etc)
Consistent use of bold and caps in subheadings (after structural revisions)
Clean up typos, redundancies etc

RESPONSE: ALL ARE INCLUDED IN THE FINAL DRAFT.

POD CONTAMINANTS REPORT- COMMENTS ON SECOND DRAFT

Susan L. Anderson 10/30/09

This document provides comments on the second draft of the POD Contaminants Report produced by Mike Johnson and colleagues at UCD. The author made far-reaching and substantive revisions on the first draft. Much of the report is rewritten, there are all new tables and figures, the chemistry analysis has been revised and toxicity data synthesized more effectively. I am very impressed with the effort put into these revisions.

My comments on the first draft emphasized that the data gathering effort could easily have encompassed the budget for this report. I believe the project was underfunded and that this has caused a challenge for the lead author in particular. Immense efforts have gone into collecting the data, analyzing it and producing this report; and the authors should be congratulated. Nevertheless, there are still a number of rough spots, and I believe one more draft and a short meeting may be needed to finalize the document if the intention is to distribute it widely. For example, there was no Executive Summary provided with the second draft and, for any widely distributed document, this should be a critical part of the review.

A short timeframe was allotted for the review of this second draft. So, I have focused my comments on the major structural and technical issues within the report. I think a meeting with the review committee would help strengthen the document, especially the conclusions. I have listed only a few minor comments and editorial items as time was short but I would be able to provide more editorial comments after my return from Seattle. I was not able to download the appendices and cannot comment on format and completeness.

One overarching concern is whether the datasets are complete, and I am hoping other committee members or agency representatives can answer that question. I understand that the agreed upon focus was to be on monitoring, not research, datasets but I am surprised that there are no data on carbamate insecticides, rice herbicides, mercury (sediment and tissue) and selenium (sediment and tissue). Why were SFEI reports not cited or appropriate for the database? I am sorry as I may just misunderstand the scope.

HYPOTHESES AND INTERNAL CONSISTENCY

This report will have greater impact if key structural elements are consistent. This includes the hypotheses, the summary diagrams, the data analysis, synthesis and conclusions. It also includes the table of contents, Executive summary and other supporting elements. My recommendations in this regard are:

1. The POD species vs. non-Pod species discussion is developed as a major element of the Background section, and in the hypothesis statements. Also toxicity to prey items is discussed. POD vs. non POD is presented in Figure 1 as a structural element of the report, yet it is not developed as an analysis. Yet, it is toxicity to prey items that you actually can address indirectly. For each set of toxicity data, toxicity to *Ceriodaphnia*, *Selenastrum* and *Hyalella* is discussed. In many cases significant toxicity is observed and may indicate the potential for **toxicity to prey items**. I see the potential to add statements within the individual data sections as well as on p.53, p.65, p.67, on fig 12 and in the recommendations. Try to clarify the exact comparisons.

RESPONSE: SEVERAL STATEMENTS ARE MADE IN THE FINAL REPORT INDICATING THAT TOXICITY TO PREY ITEMS IS THE MOST LIKELY EFFECT OF CONTAMINANTS IN THE DELTA AND THE MOST LIKELY POTENTIAL CAUSE OF THE POD.

2. The threadfin shad phenomenon is discussed in the introduction yet, I could not see where it is addressed further in the report. It would be okay to leave this text in the report if you think it is important and then to just state at the end of the section that there were no data to directly address this topic. I would then remove it from other structural elements such as Fig 1. This is an interesting point but somewhat distracting since there are no data to analyze that are relevant.

RESPONSE: THE THREADFIN SHAD PHENOMENON IS ADDRESSED MORE THOROUGHLY IN THE FINAL REPORT.

3. Time period comparisons. There are three types of time period comparisons: winter/spring vs. summer fall, step decline period vs. post-step decline (01-02 vs. 03-08) and "past history by Archibald and Cox vs. current history presented in this report. The time period issue is obviously difficult to deal with and greatly improved over the first draft. However, some further suggestions include:

- Add one table at the beginning of the toxicity section and one table at the beginning of the chemistry section that shows which datasets permit seasonal and pre and post decline comparisons. Text below the table could include a description of the geographic ranges and other dataset descriptors.

RESPONSE: NO TABLES WERE INCLUDED AS NO DATASETS PERMIT PRE AND POST DECLINE COMPARISONS. IN ALL INSTANCES, NO PROGRAM OF MONITORING WAS IN PLACE FROM BEFORE THE DECLINE UNTIL ANY POINT IN TIME AFTER THE DECLINE WITH THE EXCEPTION OF NPDES COMPLIANCE MONITORING. THESE PROGRAMS ARE EXCEPTIONALLY LIMITED IN GEOGRAPHIC SCOPE, AND THE DATA AVAILABLE LACK SUFFICIENT QUALITY CONTROL DATA TO BE OF ANY USE.

- Look through each data description and a brief statement formalizing your conclusions for each comparison. These exist in many places but are missing in others. For example on p. 41 it looks to me that some statement could be made about seasonal comparison. On p. 43 you develop seasonal comparisons and maybe these could be summarized in one sentence saying that "Overall data suggest higher toxicity in the winter period for all species tested. On p. 44, the C. dubia data are not presented quantitatively so specific numbers for a seasonal comparison could be provided. Just track through each dataset in the toxicity section and make sure that opportunities for comparisons are taken and that the finding is summarized. This is also true for pages 22 and 24 of chemistry sections.

RESPONSE: ALL OPPORTUNITIES FOR SEASONAL COMPARISONS HAVE BEEN EXPLOITED AS POSSIBLE. TABLES WITH SEASONAL COMPARISONS HAVE BEEN ADDED TO THE REPORT.

- Synthesis and conclusions sections should succinctly state whether a seasonal pattern was observed and for what species, noting the strongest datasets in this regard. The same is true for pre-decline vs post-decline. Much of this information is nearly complete but

should be consistent through all the elements of the report. Paragraphs with strong topic sentences in each synthesis section are essential. Then, recommendations should state whether you still feel that a focus on the winter/spring is justified.

RESPONSE: THE SYNTHESIS AND CONCLUSIONS INCLUDE STATEMENTS AS TO THE SEASONAL PATTERNS AND PRE- VS. POST-DECLINE COMPARISONS.

- The Fox and Archibald report is obviously a key reference. I do not know the report, but if you think it is a solid critical review (wasn't much of the old Striped Bass data controversial/widely criticized?) it certainly adds a lot to this report to mention it. Yet, I would be more cautious about it. Comparisons to this document should not have the same weight as comparisons made from your own data synthesis.

RESPONSE: THE FOX AND ARCHIBALD REPORT IS A KEY TO UNDERSTANDING THE RELATIVE AMOUNT OF TOXICITY AND THE CONCENTRATIONS OF SEVERAL KEY CHEMICALS IN THE DECADES PRECEDING THE POD. WHILE THIS REVIEW IS VIEWED AS IMPORTANT, THE CONCLUSIONS ARE BASED ON THE CURRENTLY AVAILABLE DATA AS WELL. THE FOX AND ARCHIBALD REVIEW IS BUT ONE ASPECT OF THE EVIDENCE LEADING TO THE CONCLUSIONS IN THE REPORT.

- Figure 12 (not listed in list of figs) actually is confusing. It should be revised so that it exactly mirrors your hypotheses. These are : POD vs. non POD, POD fish vs. prey items, winter/spring vs. summer fall, 00-02 vs. 03-08. Then you can either conclude by chemical, toxicity test species, histopath. Also, the judgments made seem arbitrary. In contrast Table 10 provides criteria for the conclusions reached. In my opinion, Table 10 was a good idea and refocuses the reader on the intent of the document.

RESPONSE: FIGURE 12 WAS COMPLETELY REVISED AND THE INFORMATION INCLUDED IN FIGURE 12 IS NOW INCLUDED AS FIGURE 11.

INTRO/APPROACH

Intro- 1st para ..change trophic chain to trophic web

Approach- how does this report document exposure pathways? Maybe you just mean exposure scenarios. I think references to exposure and dilution are still weak throughout the report. If Jeff Miller does not comment on this, I can try to help edit and firm this up. Examples include adding reference to published literature such as Kuivila and Foe toxicity studies that couple toxicity and chemistry and I believe some of Inge's papers do this quite well. On p. 7 the discussion of exposure of different species should acknowledge more about what is known re dilution which would be huge. Also, last line p.12 seems naïve, changes in dilution and bioavailability could make all the difference.

RESPONSE: THIS REPORT DOES NOT DOCUMENT EXPOSURE PATHWAYS. THERE ARE NO CONCURRENT STUDIES THAT CO-LOCATE CONTAMINANTS AND POD SPECIES. INSTEAD, THE ANALYSIS DISCUSSES THE POTENTIAL FOR EXPOSURE BY EVALUATING THE CONCENTRATIONS OF VARIOUS CONTAMINANTS IN THE DELTA FROM JANUARY TO JUNE WHEN ALL POD SPECIES ARE KNOWN TO BE PRESENT. SO LITTLE IS KNOWN ABOUT SOME OF THE POD SPECIES THAT EGGS HAVE NEVER BEEN FOUND. CONSEQUENTLY, IT IS NOT POSSIBLE TO DETERMINE ACTUAL EXPOSURE. ALTHOUGH DILUTION IS ASSUMED TO BE LARGE,

VERY LITTLE IS KNOWN ABOUT DILUTION. AS MENTIONED IN A LATER RESPONSE, IT WAS DETERMINED THAT NOT ASSUMING A LARGE DILUTION WAS THE MOST CONSERVATIVE APPROACH. IF A LINK BETWEEN CONTAMINANTS AND THE POD CAN NOT BE ESTABLISHED WITH THE MOST CONSERVATIVE APPROACH, IT IS UNLIKELY TO EXIST.

BACKGROUND

Is there any data from the review under way on POD vs non pod species sensitivity?? It would be great to have a small table here.

RESPONSE: NO DATA ARE AVAILABLE ON SENSITIVITY OF THE POD SPECIES TO CONTAMINANTS.

Also see comments on report structure above

HYPOTHESES

See comments above. It is important to be sure there is consistency between hypotheses and all structural elements or the report. Also make it clear if there are NO data to address one of the hypotheses (e.g. POD vs. non POD?).

In figure 2. You identify the dark line in the legend. What is the lighter line? There are two figure 2s. The other one is in the chemistry section.

RESPONSE: THE FIGURE CAPTION FULLY IDENTIFIES THE LINES IN THE FIGURE. THE FIGURE NUMBERS ARE CORRECTED IN THE FINAL REPORT.

DATABASE DEVELOPMENT

3rd bullet…missing words? Not possible…to interpret….for the constituent

RESPONSE: THE MISSING WORDS WERE ADDED TO THE BULLET POINT.

WATER CHEMISTRY

Historical section- Table 2 and 4 indicate units (ug/L?). Text seems very sparse relative to tables. Please elaborate a bit. Could provide some comparison to toxic levels.

RESPONSE: UNITS WERE ADDED TO THE TABLE. TEXT WAS EXPANDED AND TWO COLUMNS WERE ADDED TO TABLE 2 PROVIDING THE US EPA OPP AQUATIC LIFE BENCHMARK VALUES FOR PLANTS AND ANIMALS.

Current- How did you get from 38 chemicals to only 10?

RESPONSE: THE RATIONALE FOR REDUCING THE NUMBER OF CHEMICALS IS NOW PROVIDED IN THE TEXT ON PAGES 18 AND 19. IN ADDITION, THE RATIONALE FOR THEN EXPANDING THE NUMBER OF CHEMICALS IS PROVIDED IN THE TEXT ON PAGE 34.

Good that you revised the chemistry section. Please continue to refine. I believe D. Denton will suggest that the terms used for Water Quality Objectives were not correct and that she is providing specific revisions.

NO RESPONSE NECESSARY.

Add Table XX which is listed in text but not given.

RESPONSE: ALL TABLES WERE ADDED TO THE FINAL REPORT.

Some discussions of half life (e.g. p.21) seem too general. Half lives under the conditions in the delta are narrower than those listed as broad ranges given in report. Do you want me to call K. Kuivila to discuss?

NO RESPONSE NECESSARY.

Also, specific timing of pesticide application for chlorpyrifos and diazinon is well characterized and could be discussed. Again, K. Kuivila is a good reference person.

RESPONSE: THE FINAL REPORT CONTAINS INFORMATION ABOUT THE CONCENTRATION OF CHLORPYRIFOS AND DIAZINON DURING THE JANUARY TO JUNE PERIOD WHICH CORRESPONDS WITH DORMANT SPRAY APPLICATIONS AND EARLY SEASON APPLICATIONS TO NUMEROUS COMMODITIES SUCH AS ALFALFA AND CORN. HOWEVER, NO SPECIFIC DISCUSSION OF THE APPLICATIONS OF ANY PESTICIDES IS INCLUDED IN THE DISCUSSION BECAUSE THE FOCUS IS THE CONCENTRATION OF CHEMICALS IN SURFACE WATERS.

Fig 9- are these sediment values? What does the “unknown” mean?

RESPONSE: FIGURE 9 FROM THE DRAFT HAS BEEN REMOVED FROM THE FINAL REPORT.

List actual values for lambda-cyhalothrin and cypermethrin

RESPONSE: FIGURE 9 FROM THE DRAFT HAS BEEN REMOVED FROM THE FINAL REPORT.

Please refer to comments about hypotheses and consistency above

NO RESPONSE NECESSARY.

TOXICITY

I believe D. Denton is commenting on the Background section. Please follow her guidance in revising discussion about utility of toxicity tests. They are of course used for regulatory decisions and there is a large and old literature about how many species must be tested to capture the sensitivity of 95% of species in a community. Yet, of course, the tests should not be the final word on a specific ecological scenario such as the POD.

NO RESPONSE NECESSARY.

p.38 transit time not known but also dilution not known

RESPONSE: DILUTION IS ADDRESSED LATER IN THE PARAGRAPH AND THERE IS THE STATEMENT THAT DILUTION IS UNKNOWN.

p.39 why is the meaning of the first category unknown?

RESPONSE: IN THE LITERATURE AND SUPPORTING DOCUMENTATION FOR THE DATA, NO EXPLANATION WAS PROVIDED FOR THE FIRST CATEGORY. THE CATEGORY DESIGNATION WAS IN THE COMMENTS SECTION OF THE DATABASE AND NO EXPLANATION WAS PROVIDED. THOSE STATEMENTS ARE NOW ON PAGE 45 OF THE FINAL REPORT AND THE EXPLANATION IS INCLUDED IN THE FINAL REPORT.

p.41 3rd para.. Sentence starting with Consequently should be the topic sentence of this paragraph

RESPONSE: CHANGE MADE AS REQUESTED (PAGE 47).

p.43 2nd para..Why is the Selenastrum data not summarized in a table?. Please add numbers where the ww% is listed in the text.

RESPONSE: THE PERCENTAGE HAS BEEN ADDED TO THE TEXT. TABLES OF TOXICITY RESULTS FOR EACH INDIVIDUAL PROGRAM ARE PROVIDED IN APPENDIX II. IT WAS DETERMINED THAT RATHER THAN PLACING ALL TEST RESULTS IN TABLES IN THE BODY OF THE REPORT, ONLY SUMMARIES WOULD BE PROVIDED AS TEXT.

p. 44 4th para on SWAMP add specific data values

RESPONSE: SPECIFIC DATA WITH RESPECT TO THE NUMBER OF TESTS AND THE NUMBER OF TOXIC SAMPLES HAVE BEEN PLACED IN THE FINAL REPORT.

HISTOPATHOLOGY

While I have not read the recent publication by Ostrach in PNAS, I would like to see it and speak with colleagues. However, on principle, it seems to me that a published paper such as this should have more weight and emphasis than it has received. If it provides data indicating a roll of contaminants in the Striped Bass decline, then this should be underscored and not lost in the report. For example it should come first in the histopathology synthesis section and given greater weight than unpublished work by Teh.

RESPONSE: AFTER A BRIEF INTRODUCTION, THE DISCUSSION OF THE OSTRACH ET AL PAPER FROM PNAS IS PLACED FIRST IN THE SECTION AS REQUESTED.

Other detailed comments on this section include:

2nd line add POD to statement on no data on fish

RESPONSE: CHANGE MADE AS REQUESTED.

2nd para delete 3rd sentence starting with “Research to…” redundant

RESPONSE: CHANGE MADE AS REQUESTED.

2nd para end add reference to Whitehead et al. (Whitehead, A., K.M. Kuivila, J.L. Orlando, S. Kotolvestev, and S.L. Anderson. 2004. Genotoxicity in native fish associated with agricultural runoff events. Environmental Toxicology and Chemistry 23 (12): 2868-2877.)

RESPONSE: REFERENCE ADDED AS REQUESTED.

3rd para- please fill in missing numbers indicated by x, y, z etc

RESPONSE: INFORMATION ADDED AS REQUESTED.

4th para second line consistency not consistence

RESPONSE: CHANGE MADE AS REQUESTED.

Section on Teh datasets. 4th paragraph – add some numbers

RESPONSE: NUMBERS WERE ADDED TO THE TWO PARAGRAPHS THAT DISCUSS THE TEH RESULTS. THESE INCLUDE THE NUMBER OF FISH AND PERCENTAGES.

Summary statement: This is good as it emphasizes the striped bass info.

NO RESPONSE NECESSARY.

CHEMISTRY AND TOXICITY SYNTHESIS

As described above, these sections should be rewritten or edited to emphasize the key comparisons described above. I find the sediment toxicity alarming. I think the Striped Bass histopathology data seems critical. I think there is enough data on fish, invertebrates and Selenastrum toxicity that the potential for toxic effects on residents is a cause for concern. I think some of the statements on p.67 2nd paragraph in particular may be too casual. I think you are safest to stick with a crisp summary of your own data and comparisons and conclude that there is sufficient data to keep the toxicity hypothesis alive and warrant further study. Also, when you refer to biomarkers on p.67 you can mention the Whitehead paper (ref above). I suggest the following text "One study conducted within the POD years (2000-01) documented DNA damage in Sacramento sucker and extensive genotoxicity in San Joaquin River water. The results followed a time course associated with agricultural runoff events in both years and effects were repeated in the field and the laboratory"

RESPONSE: THE CHEMISTRY AND TOXICITY SECTIONS OF THE SYNTHESIS WERE REVISED SUBSTANTIALLY FOR THE FINAL REPORT. THE REFERENCE WAS NOT ADDED TO THE TEXT.

RECOMMENDATIONS

As always, recommendations come at the end of a long and arduous writing effort and they are always hard to do. It is tempting to make many detailed recommendations but I believe you have to be very careful to keep this document focused. The recommendations need some work and some focus. They should not be extraordinarily long but they should be more specific, more strategic and very carefully edited.

I think a really helpful thing would be to make the conclusions of the document resonate with the conclusions of the "POD Biomarker Taskforce" that I chaired. I will send you the document in a separate email in case you don't have it. Obviously that document focuses on biomarkers alone but an attempt was made to provide a strategic way forward that was prioritized. There are Tier 1 (fairly specific) and Tier 2 (general) recommendations for future monitoring. Resource and design limitations are mentioned in general terms. This may be beyond your scope but you could provide priorities and note key stumbling

blocks. For example, to develop resident species tests, organism availability and hatchery limitations must be addressed. In addition, in situ studies are a good idea but may be more appropriate for a Tier 2 investigation. In addition, numerous study design issues must be addressed to simply conduct more intensive toxicity testing that is coupled with better chemistry and TIE. In general, the list provided in #1 of the recommendations has several good points.

RESPONSE: THERE WERE CONFLICTING OPINIONS AMONG THE REVIEWERS ABOUT THE WAY THE RECOMMENDATIONS SHOULD BE STRUCTURED. THE SUGGESTIONS ABOVE WERE NOT INCORPORATED INTO THE FINAL REPORT.

Below, I provide a few quick specific comments below:

Toxicity tests are critical here and the issue is good sampling design, chemistry and appropriate conclusions from the data.

NO RESPONSE NECESSARY.

Resident species tests are a great idea. For which species can you get adequate supply, in which seasons? How long to develop protocols? Also integration with biomarkers is a good idea for the resident species.

RESPONSE: THERE WAS DISAGREEMENT AMONG REVIEWERS AS TO THE UTILITY OF RESIDENT SPECIES TESTS. THE TEXT WAS EDITED TO REFLECT THE OPINIONS OF THE TWO TOXICITY TEST EXPERTS ON THE PANEL WHICH CAUTIONED AGAINST LARGE SCALE USE OF RESIDENT SPECIES FOR TOXICITY TESTS.

In situ is a good idea for selected, well characterized situations. Consider qualifying your discussion.

SEE RESPONSE ABOVE.

Sublethal endpoints and “condition indicators” are addressed in the biomarker report. Perhaps some of those ideas would resonate.

Note to edit for considerable redundancy. Chronic is repeated twice as is in situ

RESPONSE: EDITS MADE AS REQUESTED.

I question recommendation 2 for this report. In my mind, priority should stick to Tier 1 and Tier 2 toxicity studies. This is just an opinion.

RESPONSE: RECOMMENDATION 2 WAS RETAINED FOR THE FINAL REPORT BASED ON COMMENTS FROM ANOTHER REVIEWER BUT EXPANDED SLIGHTLY TO EXPLAIN THE NEED FOR SUCH A RECOMMENDATION.

Research needs: should be edited so highest priorities and those clearly related to this report are given. Resolving the effects of toxic suspended sediments seems key (listed last). Following up on the striped bass data seems key. It might be wise to list some research steps that are presented in the biomarker document as well. Be careful that anything listed is truly a priority.

RESPONSE: THE RECOMMENDATIONS WERE NOT ALTERED FOR THE FINAL REPORT BUT WERE EDITED FOR CLARITY AND BREVITY.

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY

REGION IX

75 Hawthorne Street

San Francisco, CA 94105

October 30, 2009

To: Dr. Michael L. Johnson
Center for Watershed Sciences
University of California, Davis
One Shields Ave
Davis, CA 95616

Thank you for the opportunity to provide a review and comments for the report on "Evaluation of Chemical, Toxicological, and Histopathologic Data To Determine Their Role in the Pelagic Organism of Decline (POD)". Acknowledged that this was quite an undertaking to partake, therefore I appreciate the data collection and synthesis that you have conducted and applaud your work. I appreciated the opportunity to provide informal comments on the draft in regards to background language for the toxicity section and chemical benchmark approach, and technical and editorial comments for the toxicity section. Since my review time was limited for this report, I focused my review on the toxicity testing portion and report recommendations. With the limited time, I started with the assumption that the complex data synthesis of the individual toxicity testing programs summaries of the individual programs reviewed are both accurate and complete, as I am confident in the expert data interpretation of Dr. Inge Werner and the expert review by Dr. Susan Anderson (on previous draft).

Overall:

- A map is critical for this document which includes the boundary line for the area within the Delta, locations of the POD sampling sites (e.g., Site 902, 915 etc), and the NPDES permitting locations for which data was evaluated.

RESPONSE: A MAP WAS ADDED TO THE BODY OF THE REPORT, AND SEVERAL MAPS WERE PLACED IN THE APPENDIX OF TOXICITY RESULTS.

- An acronym list to assist reader.

RESPONSE: LIST ADDED AS REQUESTED.

For the toxicity testing section, I provided some excerpts from Denton et al., 2007, as background information regarding the purposes of conducting and evaluating toxicity testing with the USEPA standard test species. Some of that discussion includes:

The primary advantage of using the toxicity testing approach is that this tool can be used to assess toxic effects (acute and chronic) of all the chemicals in aqueous samples of effluent, receiving water, or stormwater. This allows the effect of the aqueous mixture to be evaluated, rather than the toxic responses to individual chemicals. Some advantages of WET testing include the toxicity of effluent or ambient water is measured directly for the species tested; the aggregate toxicity of all constituents in a complex effluent is measured; and ecological impacts can be predicted before they are occur. Toxicity tests can be used to assess ambient waterbodies (i.e., receiving water) making these tools effective in the assessment of small and large watersheds (de Vlaming et al., 2000). This has been demonstrated by the State of California which has successfully used an ambient toxicity testing approach to identify and regulate frequently occurring toxic chemicals. This approach includes pinpointing critical sampling locations for collecting the ambient waters to be assessed using acute and chronic toxicity tests. If toxicity is detected, then additional samples are collected to determine the spatial and temporal toxicity patterns. Subsequently, EPA's Toxicity Identification Evaluation (TIE) procedures are used to identify the causative toxicant(s). The goal of the TIE is to identify the chemical(s) causing toxicity in an aqueous sample. This ambient toxicity testing approach has led to the 303(d) listing of chemicals beyond the 126 priority pollutants commonly tested, such as listing the pesticide diazinon, which is not a priority pollutant (SWRCB 2003). In addition, the approach of toxicity testing in conjunction with TIE analysis may be used to determine chemical interactions. These interactions can be additive, synergistic, or antagonistic. Lydy et al., (2004) provides a synthesis review of challenges in regulating pesticide mixtures and pesticide toxicity to aquatic organisms. Toxicity tests using standard WET organisms and performed on ambient water samples are considered surrogate exposures for environmental realism. Exposing these test species *in situ* can increase the environmental relevance. The test organisms used for *in situ* toxicity testing range from the same organisms used in WET toxicity testing to a wide array of other test organisms (like the POD species).

RESPONSE: MUCH OF THE TEXT WAS ADDED TO THE INTRODUCTION OF THE TOXICITY SECTION.

Recommendations in the Report:

- I suggest that our existing baseline and regulatory based programs like NPDES, ILRP, etc, need to continue evaluating for the presence of relevant chemicals of concern including the emerging chemicals, current use pesticides such as pyrethroids with methods with MDL at toxicological levels of concern and conduct the USEPA test methods (USEPA 1995, 2002a, 2002b, 2002c) to evaluate for both acute and chronic endpoints in a complete fashion. In a complete fashion, I mean that these programs need to test at a frequency that will capture the exposure of adverse effects, conduct and follow proper QA/QC procedures (USEPA 2000 and test methods – USEPA 1995, 2002a, 2002b, 2002c) respond to toxic responses with TIE promptly as the program defines, report in a standardized format (Denton et al., 2007). Beyond these baseline and regulatory programs, we need to develop models to inform us spatially and temporally (see last bullet) in the watershed we should be testing, apply additional sublethal endpoints such as swimming performance, and biomarkers, along with *in situ* exposures as needed in pertinent locations.

RESPONSE: NO RESPONSE NECESSARY.

- Additionally, for these baseline programs and developing a comprehensive monitoring program, conducting proper toxicity test reviews are an important part of an overall quality assurance program. I and my colleagues have found in many program reviews, that both program managers and the entity gathering the data that critical elements of report preparation and review needed a more thorough review (see chapter on Report preparation and test review, USEPA 2002b and USEPA 2000). The report preparation and test review chapter states, "Test review should be conducted on each test by both the testing laboratory and the regulatory authority." It is necessary to ensure that all test results are reported accurately. The components of test review include: 1) review of sample handling and collection, 2) review of test acceptability criteria, 3) review of test conditions, 4) review of concentration-response relationships, 5) review of reference toxicant tests, and 6) review of test variability (i.e., examination of PMSD values).

RESPONSE: THE RECOMMENDATION IS INCORPORATED INTO FIRST MAJOR RECOMMENDATION IN THE FINAL REPORT.

- Since ambient and stormwater samples are tested at 100% receiving or stormwater and a control, the comprehensive program should consider implementing the data analysis approach, which utilizes a bioequivalence approach ("NPDES Test of Significant Toxicity Implementation Document, an Additional Whole Effluent Toxicity Statistical for Analyzing Acute and Chronic Test Data" contact me for more information). This approach provides a consistent (not having different thresholds for determining what is toxic), establishes a beta error, thereby test power is controlled, and provides a streamlined data analysis approach for these programs.

RESPONSE: THE RECOMMENDATION IS INCORPORATED INTO FIRST MAJOR RECOMMENDATION IN THE FINAL REPORT.

- To expand upon the recommendation *in-situ* testing as an application to be used to augment the existing ambient toxicity testing program is the following. *In situ* water column toxicity tests can integrate toxicity over time, and could probably be used more sparingly, at least temporally. This is because the utilization of *in-situ* involves more sampling logistics such as location without interference with loss of organisms and apparatus, ease of sampling staff for frequent observations, and personnel safety issues, etc. In fact, (EPA ORD – Adam Biales, EPA Region 9 – Denton, DWR – Rich Breuer & Dan Riordan, and UCD – Inge Werner) has employed flow-through exposure tanks at two permanent DWR locations, Hood of Sacramento River and Vernalis at San Joaquin River. These locations are optimal sites to consider for additional future *in-situ* and flow-through exposures for both fish and invertebrates with the standard endpoints and additional biomarker tools and sublethal endpoints such as swimming performance because the need exists to examine appropriate time-scales of exposure.

RESPONSE: MANY OF THE SUGGESTIONS ABOVE WERE INCORPORATED INTO THE RECOMMENDATIONS PROVIDED AT THE END OF THE REPORT.

- I concur with the effectiveness of augmenting the traditional assessment tools, such as ambient toxicity testing and typical endpoints such as survival, reduced growth and reproduction with additional sublethal endpoints such as swimming performance. Swimming, as a measure of performance in fishes (Smith 1990; Health 1998; Werner and Oros 2005) is a key factor in linking an organism's phenotypic character (e.g., genetic makeup, anatomy) with its use of environmental resources (e.g., food, oxygen, nesting sites) for the overall reproductive output and survival of the individual and population (Wainwright 1994).

NO RESPONSE NECESSARY.

- I concur that learning and using templates from programs like SWAMP, it is a great goal to generate and provide high quality data that is comparable and accessible. The current requirements necessary to be considered SWAMP-compatible are detailed in the links found at www.swrcb.ca.gov/swamp. In order to develop a comprehensive watershed monitoring program, there are recommended steps critical in the development and implementation of these environmental studies (see figure 1 and SWAMP and http://www.waterboards.ca.gov/water_issues/programs/swamp/).

NO RESPONSE NECESSARY.

- To further elaborate on the bullet on biomarkers, Anderson et al., (2007) report on biomarkers and Health (1998) provide provides an excellent overview of fish physiological measurements and outlines measurements critical to successful assessment and integration of the impact of multiple stresses (e.g., chemicals, physical and/or chemical condition limitations) on aquatic ecosystems. Effects of environmental stress can be evaluated at several levels of biological organization, from molecular processes up to growth and reproduction that impact overall population size and community interactions. Some physiological endpoints commonly tested include hematological and immunological (e.g., hematocrit, plasma cortisol concentrations), assessments of liver and gill structure and function (e.g., mixed function oxidases enzyme induction), energetics (e.g., swimming performance, feeding and growth rates), and behavioral and nervous system function (e.g., temperature tolerance, swimming performance, altered predator-prey interactions).

RESPONSE: THE RECOMMENDATION ABOVE IS INCORPORATED INTO FIRST MAJOR RECOMMENDATION IN THE FINAL REPORT. A SIMILAR RECOMMENDATION EXISTED IN THE DRAFT REPORT.

- On the conclusion, every attempt should be made to use resident species important to the Delta ecosystem as toxicity test organisms, I have the following comments. First, see my bullet numbers 1, 2, 3, 4 as to further implement and use the standard USEPA test methods well to asses for the potential of toxicity within our existing programs and the use of these standard test species as testing tools for watershed assessment. I suggest augmenting the standard species testing with the rainbow trout (USEPA 2002a; Miller 2009) to further protect for the POD or other potentially sensitive species. A recent paper by Raimondo et al., (2008) concludes, "Comparison of relative sensitivity of narrow fish taxonomic groups showed that standard test fish species were generally less sensitive than salmonids and listed fish." This indicates that rainbow trout might be a protective test species, in general, where as others may not. This is the

case for fish; however, *Ceriodaphnia dubia* is also known to be very sensitive. For which in the Dwyer et al., (2005) paper concludes that if a combination of fathead minnow and *C. dubia* were tested, listed fishes were protected approximately 95% of the time. So, it prudent to conduct multi-species with both fathead minnow and *C. dubia* and additional sites should be tested with rainbow trout. I will provide further information, as I have asked EPA ORD, Dr. Mace Barron and colleagues to conduct some queries next week to examine whether POD fish (if sufficient data is available) to comment on their relative sensitivity (i.e., conducting some species sensitivity distributions [SSDs] and examine whether the POD species fall on the sensitive or insensitive portion of the SSD).

RESPONSE: THE LANGUAGE ON THE USE OF RESIDENT SPECIES FOR TOXICITY TESTING WAS EDITED AND THE INFORMATION CONTAINED IN THE ABOVE COMMENT WAS INCORPORATED INTO THE RECOMMENDATION IN THE FINAL REPORT.

- For ambient sampling, knowledge of land use, pesticide application patterns and timing, and system hydrology is required to select sample site locations and timing. For both stormwater and ambient samples, sites that demonstrate adverse effects, timely collection of additional site samples is essential to establish the frequency, magnitude, and duration of the toxicity at the site. During sample collection, it is critical to confirm and record the site location using GPS coordinates, note site characteristics, measure basic water chemistry (temperature, dissolved oxygen, conductivity), and estimate flow velocity and volume. The latter information may be challenging to obtain but is critical for estimating toxicant loading.

RESPONSE: SAMPLE SITE LOCATION IS BEYOND THE PURVIEW OF THIS REPORT.

- Storm drains need to be evaluated in the permitting programs for industrial and municipal facilities as these drains often discharge into these watercourses often contribute significantly to elevating pollutant concentrations during wet weather, especially following extended periods of dry weather over which pollutants accumulate (USGS 1998; Denton 2001). Contaminants will usually move into the receiving water as the storm hydrograph increases. Depending on the purpose of the study, multiple samples can be collected and tested throughout the runoff event to assess short-term effects and contaminant loading. Miller et al., (2005) present results of flow-through toxicity studies for studying stormwater in an urban creek using *C. dubia*, as a model to be considered. A note about the exiting chemistry analysis may not have identified contaminants like pyrethroids for several reasons being, these compounds have been increasing over the recent several years, the method detection levels (MDL) need to be developed at toxicological levels of concern (for water column < 1 ng/L), and enhanced collecting and sampling protocols to deal with more hydrophobic compounds like pyrethroids were needed and not available during the POD step decline period. Therefore, moving forward we need to assess what chemicals need methods to be developed with toxicological relevant MDL, along with proper sampling protocols (TDC annual UP3 reports 2007 and 2008). The initial efforts for examination of adsorption to testing containers was by Wheelock et al., 2005, which highlighted that we maybe under representing full toxicity potential. Of recent there exists guidance on collecting water or sediment samples for which pyrethroids may be of interest, see Hladik et al., (2009). This document discusses the preferred container material, container size, holding conditions and sample-handling to minimize pesticide losses. Note, the is report analyses where conducted where the full potential for chemical detection may have been under represented because of recent improved analytical procedures with lower MDLs (e.g., pyrethroids) and improved sampling and collecting procedures for hydrophobic compounds. Additionally, chemicals maybe missed because of insufficient monitoring frequency and missed sampling locations. This could be improved with real time monitoring and/or modeling efforts to identify where and when to sample (see last bullet).

RESPONSE: SAMPLE SITE LOCATION AND METHODOLOGY ARE BEYOND THE PURVIEW OF THIS REPORT.

- Sampling programs and plans need to specify that if toxicity is detected, how and when will the site sample water be re-sampled and retested using a dilution series to determine the duration, frequency and magnitude of the toxicity. Additionally, these programs need to require toxic samples to be immediately subjected to TIE procedures to identify the toxic chemical(s). To ensure successful TIEs, close communication between the toxicity and chemistry laboratories is essential. (See Wagner and Denton letter to CVRWQCB on April 2, 2009 for more details on details for the ILRP toxicity program elements). We found in our program review of the toxicity components of the ILRP that the coalitions needed to address toxic samples effectively. We note, in our letter, that "When repeated toxicity occurs at a site (e.g., > 1 month), the subsequent toxicity identification evaluations should be specific to the suspected class of toxicants and potentially include Phase II or III manipulations. The toxicity laboratory needs the chemical analysis reports or laboratory to provide the results to assist with the best choice of TIE manipulations. The laboratories should include a broader range of manipulations to minimize

inconclusive results. It is prudent to utilize additional tests to delineate/confirm the role of a particular class in the ambient sample, especially when multiple toxicants are present (Norberg-King et al., 2005, USEPA 1991a, USEPA 1991b, 1992, 1993a, 1993b)." This all goes to the point that we need to fully implement the baseline programs like NPDES both for continuous effluent discharges, and storm events, ILRP to capture the potential toxicity. Areas of improvement include: follow up promptly to toxic samples with TIEs, proper QA/QC, test review steps must be followed, consider implementing the alternative statistical approach (see bullet #3) for effluent, ambient and stormwater toxicity testing, testing at a frequency that will capture the potential toxic events (majors at least monthly), and develop a standardized formatting consistent among the programs.

RESPONSE: THIS RECOMMENDATION WAS NOT INCORPORATED INTO THE FINAL REPORT. IT WAS FELT THAT THIS RECOMMENDATION WAS TOO DETAILED FOR INCLUSION IN THE SYNTHESIS REPORT.

- It is paramount for the comprehensive monitoring program that chemical models need to be developed to assess spatial and temporal chemical loadings to the watershed, to thereby evaluate the feasibility and effectiveness of monitoring programs and mitigation measures for effective implementation. Model development should be used hand-in-hand with monitoring data to better evaluate where and when to monitor within a watershed. Models can be used to identify source areas, waterbody reaches of highest risk, optimize where and when to focus monitoring efforts, and where to target BMP research projects and mitigation measures. Models have the ability to forecast changing trends in land use, pesticide use, and climate. In addition, models can be used to evaluate the feasibility and effectiveness of mitigation measures prior to their implementation. In combination, these tools provide risk assessors with a "weight-of-evidence" approach for regulatory decision-making especially since there exits a large array of contaminants, along with the geographical distance, provides a challenge of both resources and management to an individual program. Therefore, efforts are needed to tailor monitoring and assessment efforts under the listed programs, as well as provide POD investigators with needed information about pesticide peak loadings (as a parameter to start modeling initially), to assist those researchers trying to determine if contaminants were contributing to the decline of pelagic organisms in the Delta. For example, development of a model approach to assess both spatial and temporal pesticide loadings to the Delta is under initial development through a CALFED study (William et al., 2008; Dasgupta et al., 2008). Once the foundation of this model for pesticides is in place, with the land use, weather, hydrology GIS informational layers, it would create the foundation for future funding other constituents can be modeled, like sediment, nutrients, metals, etc. Identified are some aspects of model outputs of value to watershed managers:

    - Provide further knowledge of the fate and transport of the modeled chemicals (e.g., copper, organophosphates) and emerging pesticides in the Sacramento River, San Joaquin River, Bay-Delta Estuary, and headwater tributaries;
    - Match results to the location of sensitive species critical habitats;
    - Evaluate implications of future chemical use trends and changes in climatic conditions;
    - Identify and rank areas of highest risk and chemical source areas contributing to those risks;

- Aid in developing plans to improve ecosystem quality and water quality by strategic placement of best management practices and hydrologic operations;
- Support current and future monitoring programs (recommendations on strategic locations and sampling frequency);
- Link results to life cycle models currently underdevelopment for striped bass and delta smelt, as well as existing models for (salmonids); and
- Provide a data-link to support other water quality models and population models.

RESPONSE: THE RECOMMENDATION FOR THE MODELING COMPONENT WAS RETAINED FOR THE FINAL REPORT. HOWEVER, THE RECOMMENDATION WAS LEFT GENERAL WITHOUT THE DETAIL SPECIFIED ABOVE. THE READER IS REFERRED TO THE COMMENTS ABOVE FOR DETAILS OF DESIRED MODEL ELEMENTS.

If you have questions regarding my comments and suggestions, please contact me at

(916) 341-5520.

Sincerely,

Debra Denton, PhD
Environmental Scientist
USEPA Region 9

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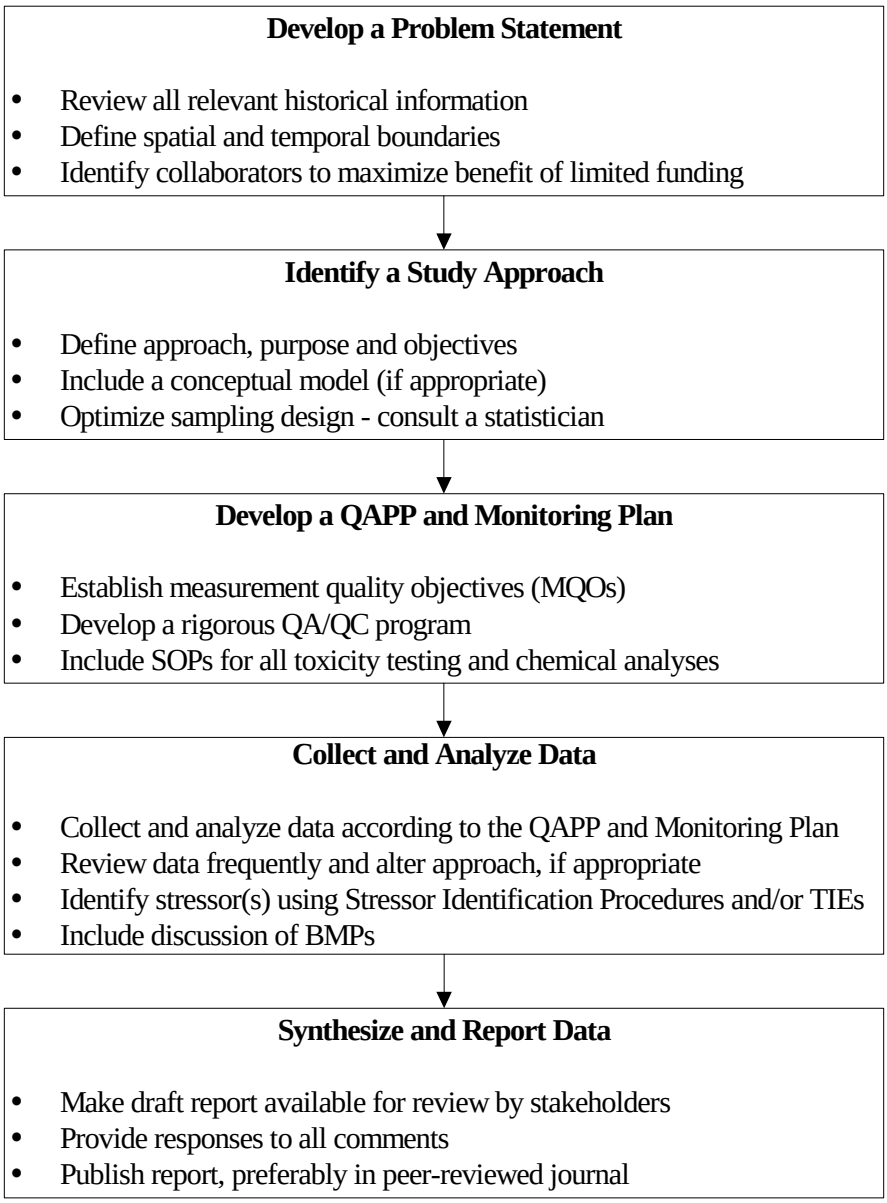
Figure 8. Recommended Steps in Development and Implementation of Environmental Monitoring Studies.



COMMENTS OF JEFFREY MILLER

To: Mike Johnson
University of California, Davis
One Shields Ave
Davis, CA 95616
From: Jeff Miller
AQUA-Science
17 Arboretum Drive
Davis, CA 95616

Date: October 30, 2009 (via email)

I appreciate the opportunity to review the Document " Evaluation of Chemical, Toxicological, and Histopathologic Data to Determine Their Role in the Pelagic Organism Decline" Draft Final. It was agreed that my review will be confined to the Toxicology and related sections of the document, although I did read the Background section which I think readers will find well written and informative, particularly the "Habitat and Timing" section and associated Summary.

I have previously provided you with an annotated version of the document that covers minor editorial changes. More detailed suggestions follow.

GENERAL COMMENTS ON TOXICITY-RELATED SECTIONS

- Overall, the Toxicology section of the document appears to be inclusive of most of the published monitoring data that I am aware of with relatively minor exceptions. I have provided you with a paper that discusses a 3-year rainbow trout embyrotoxicity study from the Sacramento River watershed that should be mentioned in the document. Also, it appears that Bailey et al 2000 which contains information on OP concentrations in the Sacramento River watershed during pre-POD years was not included.

RESPONSE: NO RESPONSE NECESSARY

- A map of the legal Delta indicating sampling sites should be included.

RESPONSE: MAPS INCLUDED AS REQUESTED.

- The reference section appears to be incomplete compared to the text.

RESPONSE: REFERENCE SECTION WAS COMPLETED IN THE FINAL REPORT.

- The Appendices were not available for review.

RESPONSE: APPENDICES WERE INCLUDED IN THE FINAL REPORT.

SPECIFIC COMMENTS

Toxicity Data Sets

p.35 The fish kills mentioned in the late 1960s-early 1970s were caused by the rice herbicides, Ordram and Molinate. DPR-mandated increased holding times of discharge water from rice fields after herbicide application resolved this problem.

RESPONSE: THESE CHEMICALS WERE NOT ADDED TO THE LIST ON PAGE 38 (OF THE FINAL REPORT) BECAUSE THEY WERE NOT INCLUDED IN THE LIST OF CHEMICALS DETERMINED BY CDFG TO BE RESPONSIBLE FOR THE FISH KILLS.

p. 38. Until demonstrated to the contrary, there is no reason to believe that non-standard test organisms are any less 'sensitive' than standard test organisms (see comments on Recommendations).

RESPONSE: THE PARAGRAPH WITH THE STATEMENT WAS REMOVED FROM THE FINAL VERSION AND THE STATEMENT IS NO LONGER PART OF THE FINAL REPORT.

p. 40-41. The summary tables are very useful for assessing both temporal and quantitative aspects of toxicity. There are "??" for the *C. dubia* and *H. azteca* tests for 2003.

RESPONSE: THE ?? HAVE BEEN REPLACED BY NUMBERS IN THE FINAL REPORT.

p. 42. Indicate what % of toxic samples had corresponding TIEs.

RESPONSE: THE PERCENTAGE OF TOXIC SAMPLES WAS NOT ADDED TO THE FINAL REPORT

pp. 45-48. A tabular summary of these data (similar to Tables7-9, including the effect on toxicity with PBO) would be very helpful here.

RESPONSE: TIME CONSTRAINTS ON COMPLETING THE REPORT PREVENTED THE DEVELOPMENT OF THE TABLES REQUESTED.

p. 47,50-51. The "loss" of toxicity appears to be a common problem with TIEs (I note in particular, the toxicity demonstrated in the mainstem Sacramento River for a period of 3 months in 2006 for which no cause was identified). The inability to identify causes of fugitive toxicity appears to be systemic problem and should be addressed in a programmatic way (see comments in Recommendations).

RESPONSE: COMMENT ADDRESSED IN THE RECOMMENDATIONS OF THE FINAL REPORT.

p. 51. I'm puzzled by the inability to access NPDES toxicity testing compliance reports. I believe they are required to be submitted to the Regional Boards and stored in a publicly accessible archive.

RESPONSE: THE NPDES REPORTS WERE NOT INACCESSIBLE, BUT THEY WERE VERY DIFFICULT TO OBTAIN. MOST OF THE REPORTS ARE AVAILABLE IN PAPER COPY ONLY AND ARE LOCATED IN RECORDS MAINTAINED AT THE REGIONAL BOARD OFFICES. THE TIME AND FUNDING CONSTRAINTS ON THE PROJECT PREVENTED ALLOCATING SUFFICIENT TIME TO PERFORM A RECORDS SEARCH NECESSARY TO

OBTAIN THE COMPLIANCE REPORTS AND CONVERT THE INFORMATION TO ELECTRONIC FORM. THE ELECTRONIC DATA AVAILABLE WAS USED IN THIS ANALYSIS.

p. 66. The Figure on this page and the accompanying text should be revised for clarity.

RESPONSE: THE FIGURE WAS REVISED AS REQUESTED.

RECOMMENDATIONS

(Note there are several Recommendations that are redundant - these comments respond to the first time they occur in the document)

p. 72 (bullet 2). Due to the difficulty and expense inherent in resident species testing including protocol development and validation, test organism supply and other significant factors, the need to use of resident species for routine monitoring tests should be based on differential sensitivity compared to routine test organisms and systems. First, comparative studies should be conducted to validate the need for this prodigious effort.

RESPONSE: THE SECTION/RECOMMENDATIONS CONCERNING THE USE OF RESIDENT SPECIES IN TOXICITY TESTS WAS REVISED TO REFLECT THE COMMENTS OF DRS. DENTON AND MILLER AS REQUESTED.

p.72 (bullet 3). Before routine chronic testing is implemented, problems associated with acute testing including the inability to identify 'fugitive' causes of toxicity should be fully resolved. Based on research by Wheelock et al, it is apparent that highly hydrophobic chemicals e.g. pyrethroids, are rapidly and extensively lost within 24 hrs from currently used test systems. Therefore, the toxicity of these types of materials is almost certainly underestimated. Currently used test systems should be evaluated and optimized for retention of toxicants prior to implementation of indigenous organism testing or chronic toxicity testing.

RESPONSE: SEE RESPONSE IMMEDIATELY ABOVE.

p. 72 (bullet 5). Judicious use of in situ testing may be able to eliminate some of the problems discussed above. However, due to cost and effort, their use should be carefully validated prior to widespread implementation. including methods for capturing toxic pulses for TIE evaluation There is extensive literature on this subject including the use of real-time activity monitoring (Ed Smith, Bodega Marine Lab) which could be used in conjunction with satellite up-linking and remote transmission to facilitate this testing method.

RESPONSE: SEE RESPONSE ABOVE.

p. 73 (bullet1) On the contrary, we have found that properly applied and interpreted standard USEPA Phase I (solid phase extraction - SPE, elution and add-back methods, PBO addition), Phase II (SPE concentration and HPLC fraction) and Phase III (toxicity accounting) TIE procedures have been very useful in identification of causes of ambient toxicity including pyrethroids. Some new methods that have been developed but have not been widely adopted, possibly for cost considerations. Clearly, it is unacceptable for the causes of obvious toxicity not to be identified in so many cases mentioned in this document. Likely, the principal causes of failure to indentify causes of toxicity in TIEs is the wide disparity in the TIE

capability and experience of the various testing entities involved in environmental monitoring. We have previously suggested that a Regional Center for TIE Support be developed and funded to assist in identification of unknown ambient toxicants. A group of TIE experts assembled by SETAC unanimously agreed with this recommendation (see Footnote 1). Until causes of toxicity can be routinely identified and sourced, it is questionable that expanding monitoring will lead to an improvement in water quality.

RESPONSE: A RECOMMENDATION FOR A REGIONAL CENTER FOR TIE SUPPORT HAS BEEN INCLUDED IN THE FINAL REPORT.

p.76 (bullet2). USGS and other monitoring entities have confirmed that multiple potentially toxic chemicals are frequently present in ambient samples. The interaction of multiple toxicants present in ambient waters has likely been underestimated (see note 2). However, a greater understanding of contaminant mixtures toxicity can be derived from the application of successful TIE analysis of toxic samples, facilitated by a center of TIE expertise as described above.

RESPONSE: A RECOMMENDATION INCORPORATING THE INFORMATION ON MULTIPLE TOXICANTS PROVIDED ABOVE HAS BEEN INCORPORATED INTO THE FINAL REPORT.

FOOTNOTES

1. " Success in identification of the chemical causes of toxicity would be substantially enhanced through the generation and continued funding of several regional analytical centers primarily devoted to the support of TIE investigations. In these centers, highly experienced analytical chemists with access to state-of-the-art equipment would work closely with the TIE practitioners on samples for which competently conducted TIEs have failed to identify the causes of toxicity. Clearly, considerable procedural development will be required in this effort. Analytical approaches used in the chemical identification processes along with spectral information could be made available via internet and through frequent presentations, workshops, and publications Although resolving issues of securing funding (for both creation and continued support), personnel, site selection, and logistics would likely be challenging, development of such analytical centers should be considered a research priority." SETAC 2005.

2. Identification of the cause(s) of toxicity using the TIE process is complicated by the simultaneous occurrence of multiple pesticides and their degradates in ambient samples. In the National Water Quality Assessment (NAWQA) ambient monitoring studies, mixtures of pesticides were detected in more than 90% of the samples analyzed, with three or more pesticides detected more than 70% of the time (USGS, 2006). Yet, most research has evaluated the effects of pesticides as if they occurred alone. The assessment of the causes of ambient toxicity is further complicated by the presence of pesticide degradates resulting from biotic and abiotic transformation of parent pesticides in the environment. Many of the degradates are more persistent in the environment than the parent compounds, and many are more mobile, as well (Boxall et al, 2004). In most cases, there is a paucity of toxicological information on pesticide degradates. However, a recent review of pesticide degradates for which some toxicity information is available, reported that 39% of the degradates had similar toxicity as the parent chemical while 20% were more than three times as toxic and 10% were more than 10 times as toxic (Sinclair and Boxall, 2003). Similar patterns are apparent for eight pesticides frequently detected by NAWQA with 23% of the degradates being more toxic to fish and 21% being more toxic to daphnids than the parent chemical (USGS, 2006). It is clear from these reports that more toxicity information is needed on major

pesticide degradates and that ambient monitoring programs should include analyses of at least the most toxic of these chemicals.

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COMMENTS OF LISA THOMPSON

INTRODUCTION, BACKGROUND, STRUCTURE OF HYPOTHESIS

I reviewed pages 1 – 13.

You may want to define "step decline" the first time it appears. At first I thought it was a typo and that you meant "steep decline".

RESPONSE: THE TERM WAS NOT DEFINED THE FIRST TIME IT APPEARS. IT WAS FELT THAT THE READERS OF THE REPORT WOULD BE FAMILIAR WITH THE TERM.

Change "can not" to "cannot" (underlined in blue in several spots in the document – should be able to do a find-replace search.

RESPONSE: CHANGE MADE AS REQUESTED.

p. 8, line 10 – choose one version or the other:

stages for POD and non-POD species are – appear to be different (Grimaldo 2004). Bennett

RESPONSE: CHANGE MADE AS REQUESTED.

p. 13, Fig. 2 – Can you define the other two lines through the data points? (the dashed line, and the non-bold line). Also, did MacNally et al. offer any comment about the shifts in abundance that occurred around 1980? All 4 species show a decline after either being stable (DS) or a slight increase. The timing of the decline is out of synch, but I'm wondering if the weather could have played a part (e.g., and El Nino or La Nina) in affecting spawning or rearing success over a period between 1977-1982, perhaps combined with changes in intraspecific competition for food as one species declined first, leaving more food for the offspring of another species the following year. All speculation on my part, of course, but inter-annual changes in lake zooplankton occur in sockeye lakes in Canada, where a strong year-class of sockeye suppresses the zooplankton for the following year class.

RESPONSE: THE FIGURE WAS REMOVED FROM THE FINAL REPORT.

CONCLUSIONS, RECOMMENDATIONS

I reviewed pages 63-74.

p. 61, Summary, line 10 – "CDFA" should be "CDFG"

RESPONSE: CHANGE MADE AS REQUESTED.

p. 61, Summary, line 12 – "silversides" should be "silverside"

RESPONSE: CHANGE MADE AS REQUESTED.

p. 64, Table 12, first box – sentence doesn't make sense: "Does demography [suggest] that a single cause is responsible for the POD?"

RESPONSE: THE FIGURE HAS BEEN MODIFIED FOR THE FINAL REPORT.

p. 64, Table 12, second box, second blue bubble – remove "t" from "Deltat"

RESPONSE: CHANGE MADE AS REQUESTED.

p. 65, first bullet point, line 3 – change "I.e." to all lower case

RESPONSE: CHANGE MADE AS REQUESTED.

p. 65, first bullet point, line 5 – change word placement to avoid potential confusion about "sufficient" vs. "in-sufficient":

POD species are exposed to water with chemicals in sufficient concentration to cause

POD species are exposed to water with chemical concentrations sufficient to cause

RESPONSE: CHANGE MADE AS REQUESTED.

p. 70–71 – two bullet points highlighted in pink appear to have some overlap (see Word file)

RESPONSE: THE HIGHLIGHTED POINTS REFER TO TWO RECOMMENDATIONS ABOUT THE USE OF STANDARD TOXICITY TEST SPECIES AND RESIDENT SPECIES IN TOXICITY TESTING OF DELTA WATERS. THE TWO RECOMMENDATIONS HAVE BEEN MODIFIED SUBSTANTIALLY IN RESPONSE TO COMMENTS FROM OTHER REVIEWERS. THERE IS NO LONGER OVERLAP.

p. 70–71 – two bullet points highlighted in green appear to have some overlap

RESPONSE: THE HIGHLIGHTED POINTS REFER TO TWO RECOMMENDATIONS ABOUT THE USE OF IN SITU STUDIES. THE TWO BULLET POINTS HAVE BEEN COMBINED INTO A SINGLE RECOMMENDATION.

p. 70–72 – three bullet points highlighted in yellow appear to have some overlap

RESPONSE: THE THREE POINTS REFER TO TOXICITY TESTING WITH CERIODAPHNIA AND HYALELLA. TWO OF THE POINTS WERE COMBINED INTO A SINGLE RECOMMENDATION FOR THE FINAL REPORT. THE OTHER BULLET POINT WAS DETERMINED NOT TO BE OVERLAPPING AND WAS RETAINED.

p. 71–72 – two bullet points highlighted in blue appear to have some overlap

RESPONSE: THE HIGHLIGHTED POINTS REFER TO TWO RECOMMENDATIONS ABOUT THE USE OF CHRONIC ENDPOINTS IN TOXICITY TESTING. THE TWO POINTS HAVE BEEN COMBINED INTO A SINGLE RECOMMENDATION.

p. 73, item #5, 2nd bullet point. Change:

The significance of small Delta water bodies for the survival and rearing for POD fish

To

The significance of small Delta water bodies for the survival and rearing of POD fish

RESPONSE: CHANGE NOT MADE.

APPENDIX III

I reviewed pages 110-133. Please see edits in track changes.

RESPONSE: EDITS IN THE TRACK CHANGES MODE MADE AS REQUESTED.

Was Ecopath/EcoSim (http://www.ecopath.org/) considered as a way to track the abundances of the different POD species? It would allow the modeling of the population dynamics of all the POD species (and non-POD species simultaneously. I'm not sure whether the developers ever got EcoSpace running, but that would also allow spatial modeling over time. I think that the chronic and acute effects of contaminants could be modeled as declines in survival or fecundity rate (enter a fishing mortality rate equivalent to the assumed rates caused by toxicants).

RESPONSE: THE USE OF A MODEL TO TRACK ABUNDANCES WAS BEYOND THE SCOPE OF THIS PROJECT. ALTHOUGH USEFUL, MODELING THE EFFECTS OF CONTAMINANTS ON THE POD SPECIES WILL NECESSARILY BE DONE AT SOME OTHER TIME.

COMMENTS OF CENTRAL VALLEY REGIONAL WATER QUALITY CONTROL STAFF

CENTRAL VALLEY REGIONAL WATER BOARD STAFF COMMENTS ON MIKE JOHNSON'S "EVALUATION OF CHEMICAL, TOXICOLOGICAL, AND HISTOPATHOLOGIC DATA TO DETERMINE THEIR ROLE IN THE PELAGIC ORGANISM DECLINE"

GENERAL COMMENTS:

Overall, the report represents a lot of thoughtful hard work and is a valuable contribution. It should provoke much thought and discussion and help the Water Boards move toward a more robust monitoring and analysis plan. These comments are in addition to specific formatting, typographical, and grammatical suggestions submitted separately.

SPECIFIC COMMENTS:

Page 1 – The contract had the twin objectives of evaluating (1) whether chemical concentrations/toxicity in the delta constituted a violation of the narrative objective, "*no toxics in toxic amounts*" (2) whether contaminants could be contributing to the POD. An assessment of whether there continue to be violations of the narrative objective is extremely valuable. Ultimately, we will never know to what extent contaminants caused the POD but developing and implementing a better program to detect and control contamination will be valuable in protecting resources in the future. To do that we need a report we can point to as evidence of ongoing problems and the need for follow up work.

RESPONSE: THE CUMULATIVE FREQUENCY DISTRIBUTIONS FOR CHEMICALS IN THE DELTA WATERS SHOULD BE SUFFICIENT TO EVALUATE VIOLATIONS OF THE NARRATIVE OBJECTIVE. IT WAS DETERMINED THAT THIS REPORT SHOULD NOT MAKE JUDGMENTS ABOUT VIOLATIONS AS THAT IS THE PURVIEW OF THE REGIONAL BOARD.

Page 5 – Please include some discussion of the food habits of each of the POD species with an emphasis on diet during the larval/juvenile period. In particular, is there some overlap in diet and is it likely that toxics might be reducing prey abundance at key times and places in the Delta? For example, if most of the POD species are constrained to open water while sunfish and bass are more littoral in their distribution and silversides are everywhere, could there have been a collapse of open water zooplankton that resulted in a collapse of their predators? What are the primary prey items of each species?

RESPONSE: THE DIETS OF EACH SPECIES ARE PROVIDED IN AS MUCH DETAIL AS IS KNOWN. RELATIVELY LITTLE IS KNOWN ABOUT THE SPECIFIC DIETS OF THE POD SPECIES IN THE LARVAL/JUVENILE STAGES. DISCUSSIONS OF THE TROPHIC CASCADE ARE NOW IN THE REPORT ALTHOUGH NOT IN GREAT DETAIL. THE CONCLUSION THAT CONTAMINANTS COULD BE RESPONSIBLE FOR EFFECTS ON PREY OF POD SPECIES IS MADE.

Page 6, Table 1 – Please include additional text explaining how the periods of indirect toxicity, particularly June – December for TS and SB, were determined.

RESPONSE: THE COLUMN IN TABLE 1 THAT INCLUDED THE PERIOD OF INDIRECT TOXICITY WAS REMOVED FROM THE FINAL REPORT.

Page 7, paragraph 2 – The assumption that larval and juvenile stages are the most susceptible is a fairly large assumption, especially when we don't know which contaminant(s) may be most problematic. There should be some discussion of the appropriateness of this assumption, or reference to studies that support this.

RESPONSE: THE STATEMENT ABOUT LARVAL AND JUVENILE SUSCEPTIBILITY WAS REMOVED FROM THE FINAL REPORT.

Page 8, paragraph 1 – Would the fact that sunfish and bass have a more diverse prey group make them less vulnerable to decreases in one or two species of their prey? The heading of this section was differences between POD and non-POD prey toxicity, which I took to mean pelagic declining and non-declining species, but this seems to be moving to an open water versus pelagic discussion. I think contrasting the pelagic makes more sense here, and discussing the open water versus pelagic for the species that go into open water belongs in another paragraph.

RESPONSE: THE EFFECT OF THE DIVERSITY OF THE PREY BASE ON THE VULNERABILITY OF THE SPECIES TO THE EFFECTS OF CONTAMINANTS ON THEIR PREY BASE IS NOT KNOWN. THE DEFINITION OF PELAGIC IS OPEN OCEANS OR SEAS RATHER THAN LIVING CLOSE TO LAND. AS SUCH, THE DESCRIPTION OF THE DECLINE IN ABUNDANCE OF THE FOUR SPECIES AS THE PELAGIC ORGANISM DECLINE MAY NOT BE COMPLETELY ACCURATE AS NOT ALL OF THE SPECIES USE THE OPEN OCEAN. HOWEVER, AS A GENERAL DESCRIPTOR, PELAGIC IS OFTEN MEANT AS LIVING IN OPEN WATER RATHER THAN BENTHIC (BOTTOM) OR LITTORAL (NEAR THE LAND-WATER INTERFACE) DWELLING. CONSEQUENTLY, PELAGIC AND OPEN WATER ARE SYNONYMOUS IN THE REPORT AND THE COMMENT ABOVE IS NOT POSSIBLE TO ADDRESS. IF THE COMMENT REFERS TO LITTORAL VS. PELAGIC SPECIES, THE PORTION OF THE REPORT IS NOW TWO PARAGRAPHS BUT BOTH INCLUDE DIET OF LITTORAL AND PELAGIC SPECIES. SPECIES THAT ARE LITTORAL AS ADULTS ARE OFTEN PELAGIC AS JUVENILES WHEN THEIR DIET INCLUDES ZOOPLANKTON.

Paragraph 2 – It seems like the decrease of copepods as prey may be a wash among POD species. This should discuss sensitivity and exposure of the non-common prey not simply that they prey on different species.

RESPONSE: ADDITIONAL MATERIAL ON EXPOSURE AND SENSITIVITY WAS ADDED BUT ONLY IN THE GENERAL SENSE. NO SPECIFIC INFORMATION IS AVAILABLE ON THE SENSITIVITY OF NATIVE SPECIES TO THE CHEMICALS FOUND IN THE DELTA.

Page 11 – Bullets 1 and 2 should include language about effects on lower trophic levels. There may be work by Wim Kimmerer that can be referenced in the change point analysis discussion of the last paragraph.

RESPONSE: THE AUTHOR DISAGREES WITH THE COMMENT AND SUGGESTION AND NO LANGUAGE WAS INCLUDED.

Page 12 – The last sentence seems to imply that it doesn't matter where you sample. Is this what you intended? Perhaps additional language specifying conditions that would or would not matter would be pertinent.

RESPONSE: THE STATEMENT WAS REMOVED FROM THE FINAL REPORT.

Page 14, paragraph 2 – Please explain why some hardy copy data was transferred into an electronic format, while others were simply reviewed, and give some perspective on how much data was used versus reviewed for weight of evidence, versus thrown out.

RESPONSE: THE RATIONALE FOR RETAINING DATA IN THE ANALYSES IS NOW EXPLICITLY STATED IN THE BULLET POINTS ON PAGES 13 AND 14. THE VERBIAGE ABOUT TRANSFERRING DATA TO ELECTRONIC FORMAT AND THE WEIGHT OF EVIDENCE WAS REMOVED FROM THE FINAL REPORT BECAUSE ONLY ELECTRONIC DATA THAT MET THE QUALITY CONTROL STANDARDS SET FOR THE PROJECT WERE USED. NONE OF THE PAPER DATA HAD SUFFICIENT QUALITY CONTROL; CONSEQUENTLY THE DATA ON HARD COPY WAS NOT USED IN THE ANALYSIS.

Pages 15 and 16, Tables 2 and 4 – What does percent of median value refer to?

RESPONSE: THE PERCENT OF MEDIAN VALUE COLUMN WAS REMOVED FROM THE TABLE AND REPLACED WITH THE OPP BENCHMARK VALUES FOR PLANTS AND ANIMALS AS REQUESTED BY OTHER REVIEWERS.

Page 20, paragraph 2 – A map of the area (radius of 30 miles) included would be helpful.

RESPONSE: MAP ADDED AS REQUESTED.

Page 21, paragraph 2 – You report that 8.9% of samples exceeded the water quality limit during the POD years. These samples were mostly from upstream places like Orestimba Creek, Del Puerto, etc. There is no comparison between this and the concentrations we observed in the Delta in the early 1990s. The conclusion should be that there are much lower concentrations now and there are not many exceedances in the main Delta channels. The follow-up sentence says that because of the long half-life, you would expect toxicity in the tributaries to be carried into the Delta and that it would remain toxic. This does not account for dilution. Is this a comparison with Water Board and USGS data mentioned in previous sections, or current levels?

RESPONSE: A PARAGRAPH IMMEDIATELY ABOVE STATED THAT THE FATE AND TRANSPORT OF DIAZINON ARE UNKNOWN. ADDITIONAL LANGUAGE WAS ADDED STATING THAT THE TRAVEL TIME FOR PESTICIDES TO THE DELTA FROM MONITORING LOCATIONS USED IN THIS ANALYSIS IS UNKNOWN. HOWEVER, THE COMMENT IS CORRECT IN THAT DILUTION COULD REDUCE THE CONCENTRATIONS SUBSTANTIALLY. THE PARAGRAPH WAS LEFT AS IS BECAUSE IT WAS BELIEVED THAT ASSUMING MINIMAL DILUTION AND MINIMAL BREAKDOWN IS THE MOST CONSERVATIVE APPROACH.

Pages 22-26 – The frequency distributions for diazinon and chlorpyrifos are very interesting and still suggest beneficial use impairments and the potential for instream toxicity. It would be interesting to calculate similar distributions for the period prior to the POD. This is important because it argues against the chemicals being implicated in the collapse. The data could also be used to determine the efficacy of the Water Board's enforcement of the objectives. Diazinon and chlorpyrifos are additive, so what

happens if you repeat the analysis but combine the two? Could you indicate in the legend for the figures the portion of the samples that were collected in the delta proper? The cumulative frequency distribution analysis suggests that a very large proportion of the samples in the Delta during the POD years were at concentrations above the water quality objectives for chlorpyrifos, but most of the samples are in small tributaries to the Delta. There are no samples from the mainstem Delta channels.

RESPONSE: NO RESPONSE NECESSARY FOR MOST OF THE COMMENT. THERE WERE VERY FEW SAMPLES IN WHICH BOTH DIAZINON AND CHLORPYRIFOS WERE MEASURED. IN THOSE FEW SAMPLES, DETECTABLE CONCENTRATIONS OF BOTH WERE ALMOST NEVER FOUND. CONSEQUENTLY, IT WAS NOT POSSIBLE TO DEVELOP CUMULATIVE FREQUENCY DISTRIBUTIONS FOR THE COMBINATION OF THE TWO CHEMICALS.

Pages 23-26 – In the discussion of chlorpyrifos, it would be useful to include the number or percentage of times the objectives were exceeded. You conclude that there was a high incidence of toxicity from chlorpyrifos and that the probability was high that POD species and their food was exposed to toxic levels of chlorpyrifos, but is there any reason to think this was more than before the POD (maybe from use reports)?

RESPONSE: THE PERCENTAGES WERE ADDED TO THE FIGURES AND PLACED IN THE TEXT.

Page 26 – The sediment chlorpyrifos distribution is difficult to evaluate without some sort of biological impairment number, like the water quality objective. Could you review the literature and include one or more sediment toxicity values? Toxicity values for *Hyalella* would be good. Is this biologically available once sequestered in sediment?

RESPONSE: IT WAS UNKNOWN AT THE TIME THE REPORT WAS WRITTEN THAT DON WESTON HAD DEVELOPED TOXICITY VALUES FOR CHLORPYRIFOS IN SEDIMENT. NO OTHER OBJECTIVE WAS FOUND IN THE LITERATURE.

Page 27 – Could you indicate whether these samples were preserved or not; if so, with what; and if not, how lack of preservation might affect the results?

RESPONSE: IT IS UNLIKELY THAT THE SAMPLES WERE PRESERVED ALTHOUGH THERE ARE NO DATA TO DETERMINE EITHER WAY. CONSEQUENTLY, A STATEMENT ABOUT LACK OF PRESERVATION WAS NOT APPROPRIATE. HOWEVER, THIS POINT WAS CONSIDERED TO BE IMPORTANT AND A RECOMMENDATION ABOUT PRESERVATION WAS PLACED INTO THE FINAL REPORT.

Page 32 – There should also be mention that recent studies show that at least one Delta organism (*Eurytemora*) is more sensitive to copper than the EPA criteria.

RESPONSE: THE STUDY FOCUSED ON AVAILABLE DATA AND RELIED ON REPORTS ONLY WHEN NO DATA WERE AVAILABLE. THE EURYTEMORA STUDY WAS NOT YET PUBLISHED AND THEREFORE DETERMINED TO BE INAPPROPRIATE FOR USE IN THIS REPORT.

Page 34 –More could be said about DDT, i.e. there is data that shows decreases. The assumption that contaminants with downward trends could not be responsible for the POD (maybe with the exception of nutrients) would seem reasonable. There are USGS reports that document trends in nitrates, salt, and other parameters that could also be included in this discussion.

RESPONSE: BECAUSE THE SHORT LIFE SPAN OF THE POD SPECIES, EXCEPT THE STRIPED BASS, IT WAS DETERMINED THAT ORGANOCHLORINE COMPOUNDS WERE UNLIKELY TO BE RESPONSIBLE FOR THE DECLINE. THE EFFECTS OF ORGANOCHLORINES WERE ADDRESSED IN REPORTING THE WORK OF OSTRACH ET AL IN THE SECTION ON HISTOPATHOLOGY. CONSEQUENTLY, DISCUSSION OF OC COMPOUNDS SUCH AS DDT WAS KEPT TO A MINIMUM. ALSO, MANY OF THE DDT DATA DID NOT HAVE SUFFICIENT QUALITY CONTROL OR SUFFICIENTLY LOW DETECTION/REPORTING LIMITS FOR INCLUSION IN THE ANALYSIS. OTHER CONSTITUENTS SUCH AS SALT AND NITRATE WERE UNLIKELY TO BE TOXIC TO SPECIES PRESENT IN THE DELTA, AND THE RELATIONSHIP BETWEEN THE CONCENTRATION OF NITRATE AND PESTICIDES SUCH AS BIFENTHRIN OR CHLORPYRIFOS WAS UNKNOWN. CONSEQUENTLY, CONSTITUENTS SUCH AS NITRATE COULD NOT BE USED AS A SURROGATE FOR OTHER COMPOUNDS AND THE TRENDS IN CONSTITUENTS SUCH AS NITRATE WERE CONSIDERED TO BE IRRELEVANT TO THE POD.

End of water chemistry section – It would be useful to include a table that depicts the chemicals, WQGoals/ Objectives, LC50s, RLs, ranges found, n, and percent of samples exceeding the value of interest for available water column and sediment data.

RESPONSE: THERE WAS INSUFFICIENT TIME TO DEVELOP THE TABLE.

Page 36, paragraph 2 – Were the TIE results confirmed by chemical analysis?

RESPONSE: THE TIES WERE PERFORMED BY AQUASCIENCE, INC AND POINTED TO A BIOLOGICAL AGENT AS THE CAUSE OF THE TOXICITY. CHEMICAL ANALYSES OF THE EFFLUENT WERE PERFORMED REGULARLY AS A CONDITION OF THE PERMIT. THE TIE RESULTS DID NOT IMPLICATE A CHEMICAL. THE SIGNIFICANT RESULT IS THAT CHANGING THE SAMPLING APPARATUS TO ONE THAT DID NOT ALLOW THE GROWTH OF THE BACTERIA REMOVED THE TOXICITY ALMOST ENTIRELY.

Page 38 – The statement that toxicity tests can be used in a weight of evidence approach is true, but the tests will still be used by themselves to determine impairment. Even if contaminants are not necessarily the cause of the POD, we are still interested in eliminating toxicity.

RESPONSE: THE CONCLUSIONS OF THIS REPORT ARE SOMEWHAT DIFFICULT TO MAKE BECAUSE OF THE IMPLICATION THAT THE CURRENT CONCENTRATIONS OF CHEMICALS OR THE LEVEL OF TOXICITY OBSERVED IN SAMPLES COLLECTED IN THE DELTA ARE "ACCEPTABLE". THE CONCLUSIONS THAT CONTAMINANTS WERE UNLIKELY TO BE RESPONSIBLE FOR THE POD DECLINE THROUGH DIRECT TOXICITY AND PERHAPS ONLY PLAY A ROLE INDIRECTLY THROUGH TOXICITY TO PREY ITEMS SHOULD NOT BE VIEWED AS AN ENDORSEMENT THAT CURRENT MANAGEMENT OF PESTICIDES IN THE VALLEY IS ACCEPTABLE. THE GOAL FOR ALL INDIVIDUALS THAT APPLY PESTICIDES OR PROVIDE CONTAMINANTS THAT ENTER SURFACE WATERS THROUGH RUNOFF SHOULD BE ZERO DISCHARGE. BECAUSE ESSENTIALLY MEMBER OF SOCIETY CONTRIBUTES CONTAMINANTS TO RUNOFF IN SOME WAY, THE GOAL OF ZERO DISCHARGE SHOULD BE ONE SHARED BY EVERY MEMBER OF SOCIETY.

Page 40, Table 9 – The first line of the heading says that these are results for the San Joaquin River and Delta. The third line says that results are not shown for samples collected outside the Delta. This is inconsistent.

RESPONSE: THE THIRD LINE REFERS TO SAMPLES COLLECTED OUTSIDE THE DELTA + 30 MILES.

Pages 40-43 – The extent of sediment toxicity is very disturbing. Is this a problem for benthic invertebrates or also water column zooplankton? Work by Wim Kimmerer and others have shown that water column zooplankton remain stationary in the estuary by migrating to the bottom and staying there if tidal flows would take them out of their salinity tolerance range. I believe this includes *Pseudodiaptomus* and *Eurytemora*, important POD prey organisms. I wonder whether this twice a day sediment contact and exposure could cause negative impacts.

RESPONSE: AS MENTIONED SEVERAL TIMES IN THE FINAL REPORT, THE ROLE OF SEDIMENT TOXICITY IN THE POD IS UNKNOWN FOR SEVERAL REASONS. 1) THE IMPACT ON DELTA ORGANISMS OF SEDIMENT TOXICITY FROM LOCATIONS OUTSIDE THE DELTA IS UNKNOWN BECAUSE THE RATE AND AMOUNT OF MOVEMENT OF SEDIMENT TO THE DELTA IS UNKNOWN. CLEARLY, SEDIMENT CAN BE MOBILIZED DURING RUNOFF EVENTS AND THE AMOUNT MOBILIZED DEPENDS ON THE FLOWS IN THE TRIBUTARY STREAMS. DEPOSITION IS NOT KNOWN. 2) THE BIOAVAILABILITY OF HIGH KOC SEDIMENT-BOUND PESTICIDES IS UNKNOWN BUT BELIEVED TO BE VERY LOW. THIS ARGUES THAT PELAGIC ORGANISMS WOULD NOT BE EXPOSED TO CONTAMINANTS IN SEDIMENTS TO ANY SIGNIFICANT DEGREE. RECENT WORK SUGGESTS THAT THE CONCENTRATION OF CONTAMINANTS IN THE WATER COLUMN THAT ORIGINATE FROM SEDIMENTS WOULD BE SIMILAR TO CONCENTRATIONS IN THE WATER COLUMN AND WOULD NOT ELEVATE THE CONCENTRATION IN THE WATER COLUMN. THEREFORE, CONTAMINANTS IN THE SEDIMENT ARE UNLIKELY TO CAUSE ELEVATED CONCENTRATIONS IN THE WATER COLUMN, EVEN WITHIN A METER OF THE BOTTOM. 3) THE MOVEMENT OF SEDIMENT-BOUND CONTAMINANTS INTO A PELAGIC FOOD WEB IS DIFFICULT TO DETERMINE. CLEARLY, THERE ARE OC COMPOUNDS FOUND IN PELAGIC FISH SUCH AS STRIPED BASS (SEE OSTRACH ET AL.'S WORK) INDICATING THAT CONSTITUENTS WITH HIGH KOC VALUES CAN ENTER PELAGIC FOOD WEBS. HOWEVER, THE PATHWAY AND SIGNIFICANCE IS NOT UNDERSTOOD.

Page 41 – Fox and Archibald review clearly establishes that there were a substantial percentage of acutely toxic samples in the major rivers in and around the Delta in the April-June period. The table does not show any toxic samples in the Delta during April to June), just the tributaries.

RESPONSE: NO RESPONSE.

Page 43 – The *Ceriodaphnia* toxicity due to ammonia at Lone Tree Creek is quite remarkable, since they are much less sensitive to ammonia than fish. I think this should be mentioned.

RESPONSE: THE CERIODAPHNIA TOXICITY DUE TO AMMONIA WAS MOST LIKELY THE RESULT OF DISCHARGE FROM A DAIRY. CONSEQUENTLY, THIS TOXICITY WAS CONSIDERED IRRELEVANT TO THE POD.

Paragraph 3 – The total number of samples and percent *Selenastrum* toxicity are missing.

RESPONSE: THE NUMBER AND PERCENT VALUES WERE ADDED TO THE FINAL REPORT.

Page 45 – 2007 sediment toxicity results for *Hyalella* in the Delta are reported. If there is any 2008 data, it should be added.

RESPONSE: THOSE DATA WERE UNAVAILABLE FOR INCLUSION IN THE REPORT.

Page 46, paragraph 3 – There was also a study by Teh that should be added to the invertebrate toxicity section. In the *Hyalella* discussion, it doesn't appear that comparisons were made between the sample amended with PBO and the non-amended sample. This would be useful information.

RESPONSE: THE DATA FROM THE TEH STUDY WERE NOT AVAILABLE FOR USE IN THIS REPORT. IT IS UNCLEAR WHAT THE REQUEST IS FOR COMPARISONS BETWEEN THE SAMPLE AMENDED WITH PBO AND THE NON-AMENDED SAMPLE.

Page 50 – SRWP should also have data from Colusa Basin Drain and the American River at Discovery Park that should be included.

RESPONSE: THOSE DATA WERE NOT IN THE DATABASE RECEIVED FROM THE SRWP FOR USE IN THIS REPORT.

Page 51 – The NPDES section discusses effluent with dilution credits accounted for, but I don't see anything about comparisons of receiving waters compared to laboratory controls. Was this data not used in this report?

RESPONSE: IT IS UNCLEAR WHAT IS MEANT BY "COMPARISONS OF RECEIVING WATERS COMPARED TO LABORATORY CONTROLS." ALL TOXICITY TESTS ARE RUN WITH AN INTERNAL CONTROL IN EVERY BATCH OF SAMPLES. ALL DATA REFER TO RECEIVING WATER SAMPLES, NOT EFFLUENT SAMPLES.

Page 58 – The Mercury section should discuss methylmercury as well, or why it wasn't included.

RESPONSE: DUE TO COMMENTS FROM OTHER REVIEWERS, THE DISCUSSION OF INDIVIDUAL CONTAMINANTS WAS REMOVED FROM THE HISTOPATHOLOGY SECTION.

Page 59 – Emerging Pollutants section seems to be missing information from additional studies, many of which were presented at the State of the Estuary Conference this year. If the data itself was not available, you should mention the types of studies and findings.

RESPONSE: SEE RESPONSE IMMEDIATELY ABOVE.

Page 66, Figure 12 – The first column needs a verb. Wording might be "does demography indicate that a single cause (i.e. contaminants) could be responsible for the POD?" The legend needs some more explanation. In addition, three categories might be more appropriate; maybe "yes," "no," and "not enough data." For example, in the third column, "Are contaminants present in the water column in concentrations documented to cause toxicity," you indicate "no" because insufficient data is available for the POD years. I think a more correct response is that there is insufficient data to evaluate the hypothesis. Finally, in the last column you do not mention Dave Ostrach's striped bass results. His work clearly shows impacts to eggs and larvae, but this is likely an ongoing effect.

RESPONSE: FIGURE 12 WAS REVISED COMPLETELY AND THE INFORMATION INCLUDED IN FIGURE 12 OF THE DRAFT REPORT IS NOW INCORPORATED INTO FIGURE 11 OF THE FINAL REPORT.

Page 68 – There should be more information on TMDL monitoring. Was the data inaccessible or non-existent?

RESPONSE: MANY OF THE TMDL MONITORING PROGRAM DATA WERE INCLUDED IN THE ANALYSIS AS THOSE DATA WERE AVAILABLE THROUGH UC DAVIS. HOWEVER, SOME DATA WERE UNAVAILABLE FOR USE.

Page 69, paragraph 2 – Please include that chemical monitoring must also either analyze for pyrethroids immediately or use a keeper solvent. Otherwise, analytical results may be biased low.

RESPONSE: A SENTENCE WAS ADDED TO THE SECTION MAKING THIS STATEMENT AND A RECOMMENDATION HAS BEEN ADDED THAT STATES DCM SHOULD BE ADDED AS A KEEPER SOLVENT.

Paragraph 3 – Please add more on whether or not this could have been due to the switch from OPs to pyrethroids in combination with insufficient reporting limits for pyrethroids in water.

RESPONSE: A SENTENCE WAS ADDED ABOUT THE DCM STORAGE BUT NO ADDITIONAL VERBIAGE WAS ADDED SPECULATING ON THE POSSIBLE REASONS FOR THE LACK OF A SMOKING GUN.

Page 72 – I think I agree with your major conclusion that "contaminants are unlikely to be a major cause of the POD, they can not be eliminated as a possible contributor to the decline." By this I think you mean that contaminant levels are not high enough to cause direct acute toxicity to fish. While you do not directly address fish ration, I am not so sure about the conclusion that contaminants might not be affecting fish through their invertebrate diet. The work by Teh is very limited in scope, and this loops back to how zooplankton populations have faired over the last 20 years. This would also be a valuable addition for addressing the starvation question. Overall, lack of information about the invertebrate food question is a weakness in this report.

RESPONSE: WITH THE DATA AVAILABLE TO REVIEW AND THE MULTIPLE STRESSORS ON ZOOPLANKTON POPULATIONS IN THE DELTA, IT IS VERY DIFFICULT TO TEASE APART THE EFFECTS OF CONTAMINANTS ON POD SPECIES' PREY. LACK OF INFORMATION ABOUT INVERTEBRATES IS A WEAKNESS IN ALL OF THE POD ANALYSES CONDUCTED TO DATE.

Page 74, second recommendation – This is unclear. Please elaborate.

RESPONSE: THE RECOMMENDATION HAS BEEN EXPANDED WITH MORE EXPLANATION.

Page 75 – Who is "the POD team?"

RESPONSE: THE POD TEAM IS THE POD MANAGEMENT TEAM.

Page 76 – As noted in this report, another recommendation is that we should measure pH, hardness, and DOC along with metals. Without the entire suite it is difficult to evaluate the data.

NO RESPONSE NECESSARY.

What do you think about a recommendation to develop another algal test instead of *Selenastrum* for inclusion in the 3 species test series? Maybe something more relevant like a diatom

RESPONSE: A RECOMMENDATION TO DEVELOP ANOTHER ALGAL TEST WAS CONSIDERED TO BE BEYOND THE SCOPE OF THIS REPORT. IN GENERAL, ADDITIONAL TESTS THAT IMPROVE THE ABILITY TO

EVALUATE THE EFFECTS OF CHEMICALS ON BIOTA ARE DESIRABLE AND CONSEQUENTLY, DEVELOPMENT OF SUCH A TEST SHOULD BE CONSIDERED.