



SAN FRANCISCO ESTUARY INSTITUTE

REGIONAL MONITORING PROGRAM FOR WATER QUALITY  
IN THE SAN FRANCISCO ESTUARY

# **Workshop Report: The Effects of Polycyclic Aromatic Hydrocarbons (PAH) in San Francisco Bay Sediments**

Prepared by

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Regional Monitoring Program for Water Quality in the San Francisco Estuary

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San Francisco Estuary Institute, Oakland, CA

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## 1. Introduction

The purpose of the PAH workshop was to reach a common understanding of the state of knowledge regarding the concentration of PAH in sediments and potential effects to estuarine/marine fishes. This is a prelude to broader discussion that will occur at a follow up meeting. For that purpose, we invited scientists who address these issues nationally and locally to summarize the status of what we know about sources, fate, and effects of PAH and management strategies for their assessment and control. Finally, we convened a panel of the same experts to address specific questions posed by the management and user community and suggest ways in which the existing assessment of PAH could be improved.

The issue of PAH contamination arises in the regulatory framework in two ways:

- Regulation of PAH discharges to the Bay from point and non-point sources.
- Regulation of the dredging and discharge of sediments that contain high levels of PAHs.

Historically, PAHs have been evaluated by comparisons to USEPA water quality standards that were adopted as part of California's toxics rule. State sediment quality objectives (SQOs) are still in the process of being developed so there has been less clear guidance on determining what concentrations of PAH in sediments have the potential to cause ecological damage.

We expect that this workshop will be the first step in a longer process of reaching consensus among the management agencies for assessing the ecological risk associated with PAH contamination in sediments. The wide variety of stakeholders who participated in this first meeting will ensure that the consensus reached on how to proceed will be robust.

Included in this report are the series of presentations that were given at the workshop. The aim of the presentation series was to stimulate discussion on the regulatory concerns and state of the science of PAH in sediments and their potential for effects on estuarine/marine fishes. Questions or comments regarding the specific contents of the presentations should be directly addressed to the individual authors, whose contact information is also included in this report.

In his presentation, Dave Mount (USEPA) discussed Equilibrium partitioning theory (EqP) and how it can be used to predict the toxicity of non-ionic organic chemicals in sediments. EqP is grounded in three basic assumptions: 1) that the chemical activity, or chemical "pressure", exerted by a sediment contaminant is proportional to its concentration in interstitial water; 2) that the response of benthic organisms to sediment contamination can be predicted based on water column toxicity data, as indexed by chemical concentrations in interstitial water; and 3) that the concentration of chemical in interstitial water can be reliably predicted using partition coefficients, such as the organic carbon partition coefficient,  $K_{oc}$ .

Todd Bridges (USACE) discussed the USACE national perspective on PAH policy and research. He described the general framework for conducting dredged material evaluations using weight of evidence approaches in sediment assessment. He further discussed the use of sediment quality guidelines in assessing PAH and their potential for impact on biota.

Daniel Oros (SFEI) discussed results from Regional Monitoring Program for Water Quality (RMP) monitoring of PAH in the San Francisco Bay sediments over the period 1993-2001. Temporal trend analysis suggests that total PAH concentrations in sediments remained constant over the period 1993-2001. Source analysis show that PAH are derived primarily from combustion of fossil fuels/petroleum and biomass, with minor amounts of PAH derived from direct petroleum input. He found that the total PAH sediment quality threshold of 1000 ppb suggested by NOAA to protect estuarine bottom dwelling fish against adverse health effects (Johnson et al., 2002) was frequently exceeded (19 of 26 stations or 73%) over the sampling period.

Lyndal Johnson (NOAA Fisheries) discussed how NMFS researchers have linked PAH exposure with DNA damage, cancer and related liver lesions, reproductive impairment, and reduced growth in bottom dwelling fish in Puget Sound. Effects thresholds were estimated through segmented regression of site-specific sediment PAH concentrations and associated DNA damage and disease prevalence in English sole. Both effects were minimal at sediment PAH concentrations below 1000 ppb, however, at levels above 1000 ppb, the risk of contaminant-related injury to English sole increased, with substantial proportions of animals showing effects at concentrations above ~5000 ppb.

Tom Gries (WA Dept. of Ecology) discussed the risk posed to benthic communities, human health, fish and wildlife from exposure to sediment PAH. He presented the results of a case study from the Puget Sound region showing that a reduction in exposure to sediment PAHs caused a reduction in biological effects in fishes.

Fred Hetzel (SFB-RWQCB) presented on the early work by the Water Board to help evaluate disposal options for dredged sediments. He presented on the background concentrations of PAHs. There are known areas around the San Francisco Bay where elevated PAH concentrations are found in deeper sediments due to historical activities including contamination from coal gasification processes.

The expert panelists agreed that sediment testing using chemical and biological methods outlined in the USEPA/USACE national testing manuals allow scientists at the management agencies to assess the risk of sediments contaminated with PAH. The panelists also suggested ways in which managers could assess the overall status of the Bay in regards to these issues through the monitoring of the Bay conducted by the RMP. These suggestions are timely as the RMP's Exposure and Effects Pilot Studies workgroup is currently developing a workplan for 2007-2008.

In the near future, there will be a follow-up to this meeting that will specifically allow for more discussion by any and all interested parties about what the information presented about PAHs may or should mean for management of dredged material in the Bay Area. One concern that will be addressed is whether any programmatic changes to the

existing indicator-based dredged material testing program will need to be made. This workshop should be seen as a first step in that process.

## 2. Discussion

Immediately following the series of oral presentations a panel discussion was convened and several questions, which are shown below, were posed to the science panel by the regulatory managers and other attendees. Several responses to the questions are included here.

1. Is there evidence of biological effects (lesions) in fish from the San Francisco Bay? How do we determine if a problem exists?
2. How can individual effects be related to population level effects?
3. Are the testing procedures that the Dredged Material Management Office (DMMO) using protective of fish species of concern to NOAA?
4. Shouldn't we be incorporating tests other than acute benthic toxicity tests to assess effects to fish? Is there an analytical test available that would assess impacts?
5. Should PAH concentrations be reported on an organic carbon (OC) basis?
6. Is there an easy method to determine PAH bioavailability?
7. Do fish use the areas that are dredged? What happens at the dredge site? What happens after dredged material is disposed?
8. Are different long-term monitoring measurements necessary?
9. What statistical techniques should be used to define "ambient"? Is there a way to correlate clean up goals with "ambient" levels?
10. Should we consider maintenance dredging to have different potential for impacts than deepening projects?

## **Responses to Questions**

### **Question 1. Is there evidence of biological effects (lesions) in fish from the San Francisco Bay? How do we determine if a problem exists?**

There is no good white paper summarizing this topic. Dr. Johnson suggested that it might be possible for her lab to at least summarize the work done by the Seattle Fisheries Center. Below we briefly summarize several studies that have presented evidence of biological effects in fish from the San Francisco Bay. PAH contamination in San Francisco Bay sediments has been previously correlated with adverse impacts on fish and invertebrates. Stehr et al. (1997) previously reported that low molecular weight PAH (LPAH, 2-3 rings) and high molecular weight PAH (HPAH, 4-5 rings) in San Francisco Bay sediments were associated with an increased risk in starry flounder for developing liver lesions, such as specific degeneration/necrosis and hydropic vacuolation. Hydropic vacuolation of biliary epithelial cells and hepatocytes was the most prevalent liver lesion found in starry flounder. Stehr et al. (1997) also reported that LPAH and HPAH in sediment were associated with an increased risk in white croaker (*Genyonemus lineatus*) for developing liver necrosis, while LPAH in sediments were associated with an increased risk of developing specific degeneration/necrosis. In addition, Spies et al. (1988) previously reported low prevalence of liver lesions including hepatic neoplasms and foci of cellular alterations in starry flounder from the San Francisco Bay. Based on the relatively small dataset, histological biological indicators of toxicant exposure appear to be prevalent in two San Francisco Bay bottom-dwelling fish species, starry flounder and white croaker. The data suggest that PAH contamination in San Francisco Bay sediments could be a principal factor causing fish liver disease and reproductive impairment, and potential effects on growth.

Johnson et al. (2002) suggested a sediment quality threshold of 1000 ppb or ng/g dry wt for sediment total PAH concentrations to protect estuarine fish against health effects that included selected degenerative liver lesions, spawning inhibition, and reduced egg viability. This threshold is based on effects evident in English sole (*Pleuronectes vetulus*) in Puget Sound. English sole is also a common fish species in the San Francisco Bay. The 1000 ppb threshold is proposed by Johnson et al. (2002) as the lowest concentration where effects in English sole begin to be observed. At concentrations >1000 ppb, there appears to be a substantial increase in the risk of liver disease and reproductive impairment, and potential effects on growth. Johnson et al. (2002), based on their English sole data and model, further pointed out that at PAH concentrations of 5000 ppb the levels of hepatic DNA adducts would be approximately 10-fold the levels found in English sole from uncontaminated reference sites, about 30% of the fish population was predicted to have some form of liver disease, and the number of fish failing to spawn was predicted to increase from about 12% to over 35%. At 10000 ppb, DNA adducts levels in English sole would have increased 12-13 fold, 50% of the fish would be expected to have liver disease, nearly 30% of the females would show inhibition of gonad growth, and over 40% would show inhibition of spawning. The total

PAH threshold concentrations (ppb) for observed DNA damage, liver lesions, and reproductive abnormalities are shown below.

More definitive studies are needed to determine the extent of potential damage/harm done to fish and if the effects that are observed in the Bay are specifically due to PAH exposure alone and not to exposure from other contaminants or mixtures of contaminants. PAH occur as a complex mixture that includes parent, alkylated PAH, and heterocyclic PAH compounds.

**Question 2: How can individual effects be related to population level effects?**

Extrapolating from tumor incidence or other histopathological changes in fish to an overall effect on the health of the population is difficult. For instance, the NMFS Milford laboratory did an extensive study of winter flounder along a gradient of contamination in urban harbors from Boston to New York. While they were able to document many histopathological impacts in the more contaminated harbors, the success of fertilization, and hatching, and juvenile, and larval growth was not significantly different in the most contaminated areas as compared to the least contaminated. The usual approach to make such an estimate is to develop a population model of the fish and evaluate sources of mortality at different life stages. The best example of this approach is an ongoing effort by Swee Teh, Bill Bennett and others at UC Davis. Such efforts require extensive information on all the sources of mortality for the fish under investigation.

**Question 3: Are the testing procedures that the Dredged Material Management Office (DMMO) using protective of fish species of concern to NOAA?**

The DMMO uses the Inland Testing Manual (ITM) as its major guidance document. Several panelists agreed that the procedures described in the ITM (all four tiers including risk assessment) are sufficient to address questions regarding risk, toxicity, and thresholds setting for protecting aquatic species. The ITM is considered a good starting point in that it provides the framework needed to make such assessments. The panelists didn't feel sufficiently up-to-speed on DMMO procedures to make specific judgments. It was suggested that managers develop a tiered framework for evaluating dredging programs so that the process for determining suitability of different dredging sites and procedures would be transparent. The panelists also suggested that simple measurements, such as sediment organic carbon content and bioaccumulation potential would be useful where more complex decisions were required.

**Question 4: Should we be incorporating tests other than acute benthic toxicity tests to assess effects to fish? Is there an analytical test available that would assess impacts?**

Bioaccumulation tests are not conducted routinely. The question was brought up as to whether bioaccumulation tests should be conducted more often and using resident fish species such as green sturgeon. Because additional bioaccumulation testing would lead to higher costs to the dredging community, it was suggested that they could be tiered based on the results of sediment chemistry measurements. It was suggested that

an alternative method for measuring bioaccumulation of PAH in fish could be done, which included using solid phase microextraction (SPME) devices (see Question 6 for more details).

Another possibility is to use a combination of direct toxicity testing and a comparison of chemical concentrations to existing water quality standards—see the Mount and Bridges presentations for further details.

**Question 5: Should PAH concentrations be reported on a organic carbon (OC) basis?**

Total PAH (all compounds on EPA Method 610 list) and total organic carbon (OC) are routine sediment measurements for dredged material that is to be disposed. Don Mount's presentation showed that the OC content of sediments can be used to normalize the concentrations of PAH and other similarly hydrophobic compounds that readily adsorb to the surface of organic. Thus, sediments with high OC generally have higher contaminant concentrations associated with them than low OC containing sediments. This simple and inexpensive evaluation on OC content can be used to normalize the contaminant concentration and improve the ability to predict whether the sediments will display toxicity.

Organic carbon (OC), in its various forms, is the main controlling variable for bioavailability.

**Question 6: Is there an easy method to determine PAH bioavailability?**

Commercially available passive sampling devices have been used extensively for assessing the bioavailability of chemicals in water and sediments. Two types of passive sampling devices include the semi-permeable membrane device (SPMD) and the solid phase microextraction (SPME) fiber. SPMDs are membranes composed of low density polyethylene (LDPE) tubing that is filled with a known amount (by weight) of triolein, which is a neutral lipid. SPMDs mimic the function of bipolar lipid membranes in the uptake of lipophilic contaminants. On the other hand, SPME fibers are useful for sampling volatile and non-volatile hydrophobic organic compounds without the use of extraction and concentration procedures prior to gas chromatographic (GC) analysis. The fiber is inserted directly into the GC and the adsorbed chemicals are released following the temperature controlled program settings of the GC analysis.

Particularly problematic in determining the bioavailability of PAHs is the presence of soot carbon. From about 1850-1950, coal was used as the principal source of energy in the Bay Area. The legacy of coal use in the Bay Area is evident in deeper sediments that contain coal and coal soot, which have high levels of PAH. Sediments that contain soot particles, which are also formed from combustion of refined fossil fuels (e.g., diesel and fuel oils), and vegetation (biomass burning), can bind or occlude PAH, thus making them less available to partition with the OC in sediments. At this time there is no agreed upon standard method for measuring the amount of soot carbon in sediments. In general, soot carbon is expected to reduce the bioavailability of PAHs and other

lipophilic contaminants, but there have not been extensive studies on the effects of soot carbon on fish histopathology.

It was further discussed at some length during the PAH Workshop that the Koc for various forms of carbon can vary by several orders of magnitude. The key issue is that not all carbon is created equal with respect to controlling bioavailability. The point was that an assessment of PAHs with regard to fish must consider the influence of bioavailability.

**Question 7: Do fish use the areas that are dredged? What happens at the dredge site? What happens after dredged material is disposed?**

The major concern here is to determine what happens to fish as a result of dredging activities. SFEI pointed out that this same concern was addressed in one of their recent studies: Dredging Impacts on Food-Web Bioaccumulation of DDTs in San Francisco Bay, CA” ([http://www.sfei.org/rmp/reports/418\\_RMP\\_dredgingImpacts\\_final.pdf](http://www.sfei.org/rmp/reports/418_RMP_dredgingImpacts_final.pdf).; Oram and Melwan, 2006). However, this study was based on fish exposure to contaminants associated with suspended sediments in the water column and not bedded sediments. Studies still need to be conducted that will specifically address the questions posed.

**Question 8: Are different long-term monitoring measurements necessary?**

It was discussed whether the Regional Monitoring Program (RMP) should regularly assess the health status of fish in the Bay. The RMP’s Exposure and Effects Pilot Studies Working Group has initiated a set of pilot studies to evaluate what kinds of measurements would be most effective. There may be an opportunity to coordinate with ongoing histopathological studies funded by the Interagency Ecological Program as part of their studies of the Pelagic Organism Decline.

In addition, we discussed additional monitoring at dredging sites. The length of time of monitoring following dredging activities was briefly addressed. It was suggested that SPMEs could be used for long-term monitoring of sediments to determine the levels of PAH that are potentially available to bioaccumulate in fish over a given period of deployment (see Question 6 for more details on SPMEs).

It was also questioned as to whether monitoring of PAH in sediments should include measuring for alkylated PAH. As a response, it was mentioned that the San Francisco Bay Regional Monitoring Program (RMP) does include alkylated PAH in their sediment monitoring program. Due to the fact that there are no analytical standards to make an exact measurement of most individual alkylated PAH, any reported concentrations are usually estimated concentrations. The RMP reports only a limited number of individual alkylated PAH compounds, high and low molecular weight PAH, high to low quotient, and total PAH on its database ([http://www.sfei.org/rmp/rmp\\_data\\_access.html](http://www.sfei.org/rmp/rmp_data_access.html)). The total concentrations of the various alkylated PAH groups (e.g., C<sub>1</sub>-phenanthrenes, C<sub>2</sub>-phenanthrenes, C<sub>1</sub>-chrysenes, C<sub>2</sub>-chrysenes, etc.) is what is generally reported.

**Question 9: What statistical techniques should be used to define “ambient”? Is there a way to correlate clean-up goals with “ambient” levels?**

One way to determine the concerns associated with a certain contaminant level is to determine whether the level of contaminant is different from “ambient” levels. Determining ambient levels still begs the question as to whether ambient concentrations are problematic—ambient concentrations in New York Harbor have a different meaning than ambient concentrations in Tomales Bay. Nonetheless, NOAA’s National Status and Trends Office has used the concept of “ambient” as defined by the 85<sup>th</sup> percentile to distinguish the dirtiest sites from other sites.

As discussed above, ambient concentrations will be more effective if they take into account the normalization for organic carbon. Ambient concentrations in sandy sites will be much different from ambient concentrations in muddy sites.

**Question 10: Should we consider maintenance dredging to have different potential for impacts than deepening projects?**

The presumption of this question is that maintenance dredging probably only removes the active surface sediments that have been moving around the Bay and that the effects should be less since dredging is simply moving these surface sediments from one spot to the next. The panelists rejected this presumption because the reality of maintenance dredging can include deeper cuts, slumps of bank material, etc. They recommended basing management decisions on the specific chemical and biological test data collected.

**Related Websites**

Dredge Material Management Office - San Francisco District  
<http://www.spn.usace.army.mil/conops/dmmo.htm>

Environmental Protection Agency - Region 9  
<http://www.epa.gov/region9/>

San Francisco Bay Regional Water Quality Control Board  
<http://www.waterboards.ca.gov/sanfranciscobay/>

California State Lands Commission  
<http://www.slc.ca.gov/>

Bay Conservation and Development Commission  
<http://www.bcdc.ca.gov/>

California Dept. of Fish and Game  
<http://www.dfg.ca.gov/>

National Marine Fisheries Service  
<http://www.nmfs.noaa.gov/index.html>

Dredging Impacts of Food-Web Bioaccumulation of DDTs in San Francisco Bay, CA.  
SFEI Technical Report 418, April 2006  
[http://www.sfei.org/rmp/reports/418\\_RMP\\_dredgingImpacts\\_final.pdf](http://www.sfei.org/rmp/reports/418_RMP_dredgingImpacts_final.pdf)

Testing Guidelines for Dredged Material Disposal at San Francisco Bay Sites. US Army  
Corps of Engineers, Public Notice 93-2, 1 February 1993.  
<http://www.spn.usace.army.mil/regulatory/93-2.pdf>

### 3. Data Gaps and Uncertainties

Several data gaps and uncertainties that were identified from the discussion are listed below. These data gaps were synthesized directly from the questions that were posed to the science panel and from subsequent discussion or from presentations that were made at the workshop. More data gaps will likely be identified in the near future and these should be prioritized as needed. Some of these issues can be resolved by scientific studies: others need for a policy determination to be made.

#### Data Gaps and Uncertainties:

##### We need

1. Studies that link biological and population effects to PAH exposure.
2. California Sediment Quality Objectives based on organic carbon.
3. Indicators of environmental and biological recovery implemented into monitoring programs.
4. Definition of Ambient or reference levels of PAH in sediments.
5. Assessment of fish usage of dredging sites before and after dredging operations to understand their exposure.
6. Ways to address cumulative effects of multiple contaminants.
7. Ways to address PAH mixtures.
8. A standardized methodology for PAH measurement (e.g., 34 PAH, alkylated PAH).
9. A determination of which toxicological endpoints are most relevant.
10. Ways to determine the geographic extent of biological impacts of a PAH hot spot.

## Appendix 1. Workshop Agenda

### WORKSHOP ON EFFECTS OF POLYCYCLIC AROMATIC HYDROCARBONS IN SAN FRANCISCO BAY SEDIMENTS

*USEPA, 75 Hawthorne St., San Francisco, July 20, 2006, 9:00 am-4:30 pm*

Meeting Purpose: To reach a common understanding of the state of knowledge regarding the concentration of PAH in sediments and potential effects to estuarine/marine fishes.

9:00 Welcome and Introductions

9:05 General Overview of Issue (Mike Connor, SFEI and Moderator)

9:20 Presentations: National Overview of State of Knowledge

- Dave Mount (USEPA): EPA national perspective on PAH policy and research
- Todd Bridges (USACE): Corps national perspective on PAH policy and research

10:20 Break

10:30 Presentations: Focus on the West Coast and San Francisco Bay- Research

- Daniel Oros (SFEI): A 10-Year Retrospective on PAH Monitoring in San Francisco Bay
- Lyndal Johnson (NOAA Fisheries): Relationship between Sediment PAH Concentration and Adverse Effects to Estuarine Fish

12:00 Break for Lunch

1:00 Presentations: Focus on the West Coast and San Francisco Bay - Policy

- Tom Gries (WA Dept Ecology): Management of PAH in Washington State
- Fred Hetzel (SFRWQCB): Determination of Ambient PAH Sediment Concentrations in San Francisco Bay

2:30 Open Discussion

What are the information gaps? What assumptions are we making? Are we using best available information? What are the next steps?

4:30 End

## Appendix 2. PAH Workshop Participants

Name	Affiliation	Email
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## Appendix 3. Participant Comments on the PAH Workshop

SFEI asked for comments from the participants immediately following the PAH Workshop:

SFEI Request: *If you have information or brief comments on any (but not limited to) of the topics discussed at the workshop or on the questions listed below, we would appreciate hearing from you. Please send any comments by July 27, 2006. All comments will be kept anonymous.*

*What did you like/dislike about the PAH Workshop?*

*What would you do to improve the workshop format?*

*What issues/items do you suggest be included in the next PAH Workshop?*

*Would you attend a science/policy workshop if it were held in Oakland?*

All responses that follow here are kept anonymous:

Respondent 1: Yes, a very good workshop. Some of us are not so steeped in the topic, so some of the basic chemistry would be nice and a list of acronyms.

Respondent 2: Thank you so much for putting this workshop on. I really appreciate your work and the work of the folks who presented. I would attend a workshop in Oakland, but would prefer it be near public transportation. 10 am would work better for me, but I would make 9 am if necessary. For the next workshop I think it would be good to go into more detail about SF Bay and PAH issues here. Map out how we might span the data gaps and develop a process that addresses this issue more carefully for dredging. I would have liked to have seen the data for the margins of the Bay where most dredging happens. Ok, that's it for me – great first meeting.

Respondent 3: *What did you like/dislike about the PAH Workshop?* I liked your effort to bring in outside experts to address the science aspects of the issue (e.g., Mount, Bridges, and Johnson), their presence brought a greater understanding of the issues. The meeting space was not optimal in terms of the room layout, security hassle, and background noise. I did not think the panel discussion was particularly effective. Much of the discussion was related to the specifics of dredging studies in S.F. Bay, which I do not think was the best use of the expert's time in this meeting. This type of discussion may have been the goal of the meeting, but I don't think it was a very effective format to bring about resolution of the issues.

*What would you do to improve the workshop format?*

A clearer statement of the workshop's objectives, including items to resolve, would be helpful.

*What issues/items do you suggest be included in the next PAH Workshop?*

You need to determine what you hope to accomplish with this series of workshops. I suggest a focus on determining the effects of PAH on marine life in San Francisco Bay. The goal would be to describe the results of studies that addressed the generic topics raised at the first meeting: What is the bioavailability of PAH in various SF Bay locations? How do measured effects on fish and invertebrates compare to results in Puget Sound or other areas used to establish PAH sediment quality guidelines. What are the research/data analysis priorities to better understand PAH impacts in SF Bay? Do the results indicate whether existing sediment assessment methods used or proposed for the bay are protective of PAH effects on fish? If these questions cannot be adequately answered using existing data, then a workshop priority would be to identify needed research that should be undertaken (e.g., special studies by the DMMO, SFEI, or other agencies).

*Would you attend a science/policy workshop if it were held in Oakland? Yes.*

Respondent 4: *What would you do to improve the workshop format?*

Several of us in the audience were uncertain as to the impetus for this first workshop. The format worked well. Mike Connor does an excellent job as moderator.

*What issues/items do you suggest be included in the next PAH Workshop?*

Bioaccumulation of PAH and public health concerns; application of sediment guidelines for PAH (pitfalls and proper use); further discussion of normalization to carbon (why do it, is it always appropriate to do it, elaboration of comments concerning type of carbon); and determining sources from ratios.

*Would you attend a science/policy workshop if it were held in Oakland? Yes!*

Respondent 5: Thank for your invitation. It was a great workshop. I believe that "biomonitoring of fish" is a better approach than toxicity testing. Setting guidelines based on the sediment toxicity is old and bad science. The presence of a high concentration of a chemical does not always relate to the same result seen in field. We have a huge MIXTURES problem in the ecosystem. The best thing to do is to study "ecosystem health" and who best to tell you that the ecosystem is healthy but the aquatic organism within the ecosystem. For example, the only way to know when there is an outbreak of bird flu is when you hear people die in a certain location. Combining our expertise in tissue chemical analysis and biomarker and endocrine disruptor work is by far the most appropriate approach that we could use for determining effects.

*Would you attend a science/policy workshop if it were held in Oakland? Yes*

Respondent 6: *What did you like/dislike about the PAH Workshop?*

I liked the openness of the panel discussion. Mike is a good facilitator in that he knows how to encourage participation without being intimidating and he can anticipate questions or concerns that audience members have that they may be reluctant to verbalize.

I can't think of anything I disliked other than the cramped space of the meeting room itself. I had a hard time seeing all portions of the screen during the presentations and had to keep bobbing and weaving in my chair to see around the heads of people in front of me. I noticed other people doing the same thing. I could have sat more toward the front of the room, but there still would have been other people who couldn't see well. Next time, if you could get a larger room and spread the chairs out more and/or project the presentations up higher, perhaps that would help.

*What would you do to improve the workshop format?*

I had read Lyndal's paper before the workshop, but I didn't have any background information from the other presenters. Dave Mount's presentation was especially packed with technical information and he went fast, so it would have been helpful to have some background on the equilibrium partitioning sediment benchmarks (ESBs) beforehand. I didn't know EPA's ORD had actually issued ESBs for several chemicals, including PAH. I guess providing background information isn't essential as long as the presentations are made available afterwards, but if the presenters have something canned that they can provide beforehand, it might help those of us who have time to read it follow their presentations better.

*What issues/items do you suggest be included in the next PAH Workshop?*

I'd really like to focus on whether the current dredged material testing framework used by the DMMO adequately addresses NOAA's concerns regarding impacts to fish.

Specifically:

1. Will measuring bioavailability the way we normally do (28 day bioaccumulation test with two invertebrate species, usually a clam and a polychaete worm) adequately assess the potential for adverse impact to fish species?
2. Is a 28-day lab exposure long enough to reach steady-state tissue concentrations for all the PAH of interest? It appears from the Corps/EPA inland testing manual that 28 days may not be long enough for those PAH with Log Kow in the range of 5.5 to 8.5. This covers several HPAH on the priority pollutant list. I believe the RMP uses a 90-day lab exposure in its bioaccumulation testing program. Is there some way we could come to a consensus on the appropriate exposure period?
3. Is the major exposure route to fish from dredged sediment through the diet, and if so, can we just compare invertebrate tissue levels to some dietary threshold for species of concern (or surrogate species)?
4. Are there other exposure pathways that might be significant (direct dermal contact with sediment for example) that wouldn't be characterized by bioaccumulation testing with invertebrates? How would we test for effects to fish from these other pathways?
5. We currently only require 16 of the priority pollutant PAH to be measured in sediment during the pre-dredge characterization process. Should we be requiring alkylated PAH to be measured also? Are we potentially underestimating total PAH by a factor of 2 or more by not measuring alkylated PAH?
6. We currently require total organic carbon (TOC) to be measured in all pre-dredge sediment characterizations, but we don't require reporting the form of the organic

carbon (OC). How important is this? Could it help us rule out the need to do further bioavailability testing up front (high OC in certain forms means insignificant bioavailability)? How expensive is differentiating the forms of OC? Is it a standard test that commercial labs can easily do?

*Would you attend a science/policy workshop if it were held in Oakland?*

Sure. It helps if it's either BART accessible or there's plenty of parking.

Respondent 7: Good workshop. The AM session flushed out the main issues. The PM session deteriorated into a bit of a free-for-all which I didn't find particularly informative nor productive. I was especially heartened to see the issue of bioavailability come up had I known or anticipated that I would have come prepared with a presentation. Issues for next PAH workshop should include dealing with (variable) bioavailability across sediments (of course), measurements, and modeling.

Respondent 8: As an HES specialist responsible for obtaining permit approvals for annual dredging, the workshop was very useful for hearing the input of the various agencies and subject matter experts. Although my chemistry background was too weak to fully appreciate all of the speakers, I was able to capture some highlights and share with my organization.

I appreciate the efforts to involve stakeholders in understanding this issue and having dialogue, and look forward to any future forums on the issue.

## Appendix 4. Technical Presentations

### ***Dave Mount***

US Environmental Protection Agency, Office of Research and Development, National Health and Environmental Effects Research Laboratory, Mid-Continent Ecology Division, Duluth, MN

### **Assessing Risks from PAHs in Sediment**

**Abstract:** Equilibrium partitioning theory (EqP) can be used to predict the toxicity of non-ionic organic chemicals, like PAHs, in sediments. EqP is grounded in three basic assumptions: 1) that the chemical activity, or chemical “pressure”, exerted by a sediment contaminant is proportional to its concentration in interstitial water; 2) that the response of benthic organisms to sediment contamination can be predicted based water column toxicity data, as indexed by chemical concentrations in interstitial water; and 3) that the concentration of chemical in interstitial water can be reliably predicted using partition coefficients, such as the organic carbon partition coefficient,  $K_{oc}$ . These relationships have been well demonstrated for a variety of non-ionic organic chemicals. PAHs exist as hundreds of different chemical structures, only a fraction of which are typically measured. This introduces two additional challenges: 1) the need to predict the toxicity of PAHs that have not been previously tested for toxicity; and 2) the need for a mixture effects model to account for the interactive toxicity of PAH mixtures. The USEPA Office of Research and Development has addressed these issues in the development of an EqP-based Sediment Benchmark (ESB) for PAH mixtures, which is available at [www.epa.gov/nheerl/publications](http://www.epa.gov/nheerl/publications). Assessment of field sediments contaminated with PAHs has indicated that there are three primary factors that introduce substantial variation into the potency of PAH contamination in sediment. The first is the influence of organic carbon partitioning, which can be accounted for by measuring organic carbon and normalizing PAH concentrations to organic carbon content. A second factor is altered bioavailability. Certain substances sometimes found in sediment can reduce the bioavailability of PAHs beyond the effect of organic carbon partitioning alone; these substances include coal, soot, tire rubber, and highly weathered asphalt among others. The third factor pertains to the source of the PAHs. While most PAHs are quantified by measuring primarily unsubstituted PAH structures – so-called “priority pollutant PAHs” – there are many more PAHs that may be present in field mixtures, depending on the source of the PAHs (e.g., petroleum versus coal tar). Taken together, these factors can create a 30,000-fold range in the dry wt concentration of priority pollutant PAHs in sediment that can be expected to cause toxicity. Accurate assessment of the ecological risks associated with PAHs should recognize these issues either explicitly or via uncertainty analyses.

*This abstract does not necessarily reflect USEPA policy.*

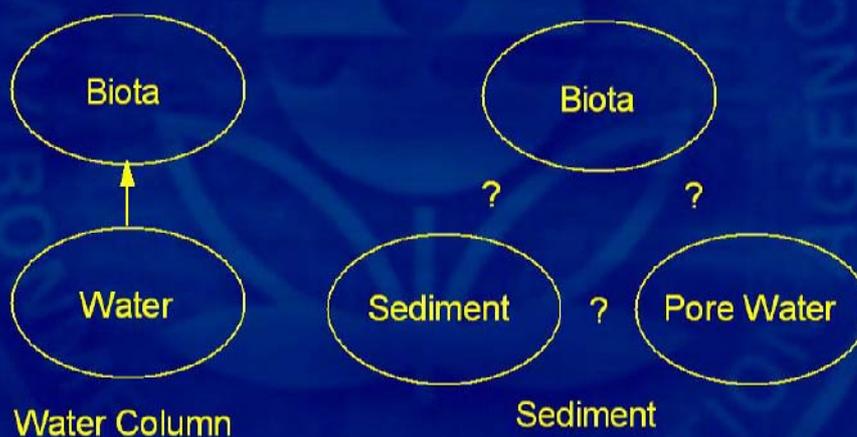
# Assessing Risks From PAHs in Sediment

David R. Mount

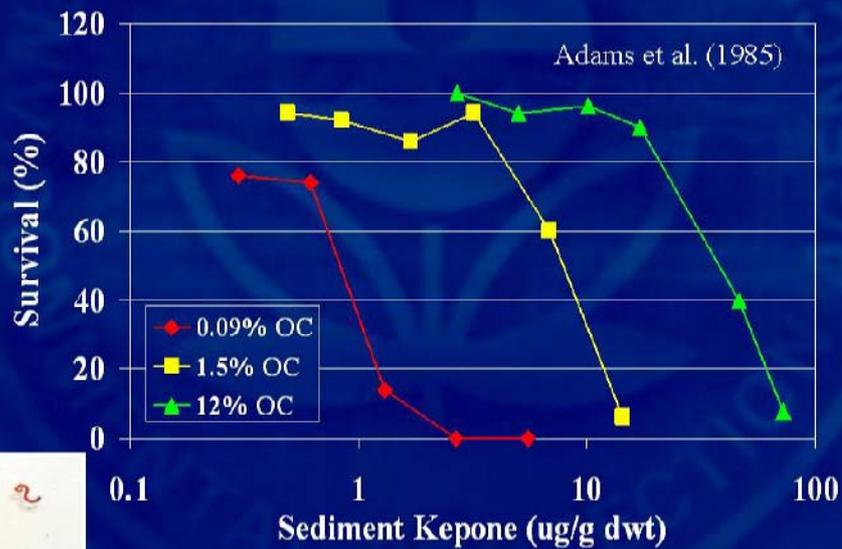
U.S. Environmental Protection Agency  
Office of Research and Development  
Mid-Continent Ecology Division  
Duluth, MN

Views expressed herein are those of the author and do not necessarily represent those of the USEPA.

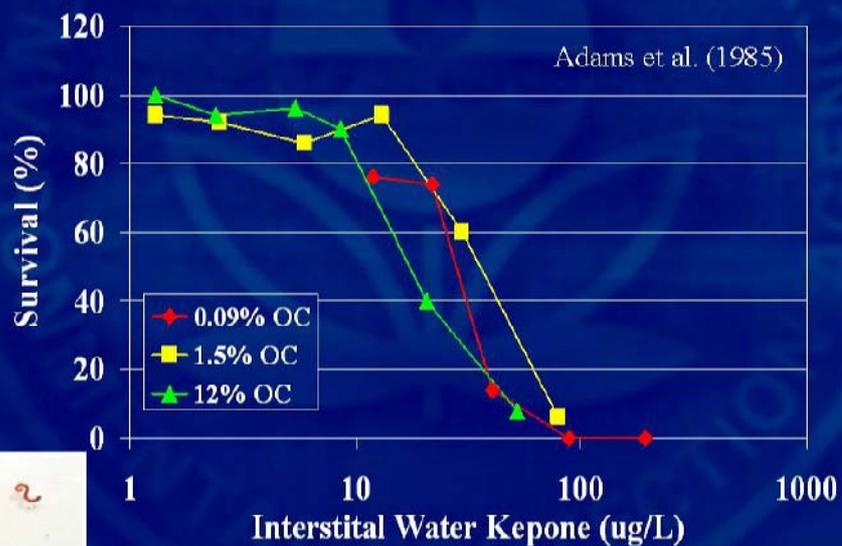
## Conceptual Models of Chemical Exposure



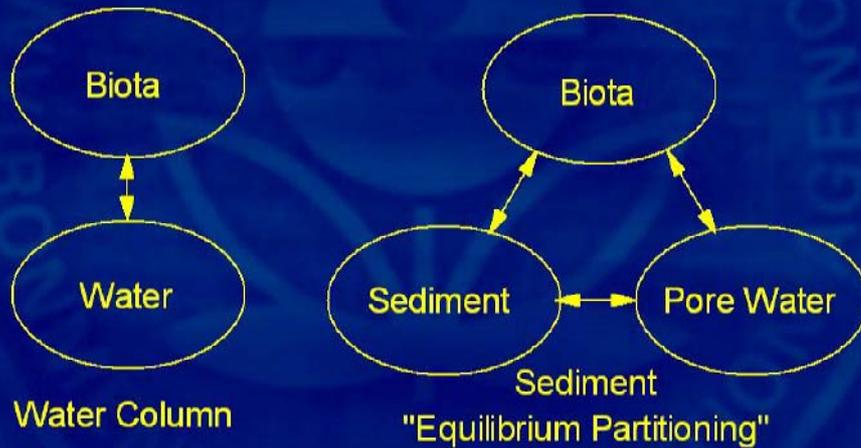
## Response of Midge Larvae to Kepone in Sediment -- Dry Weight Normalization



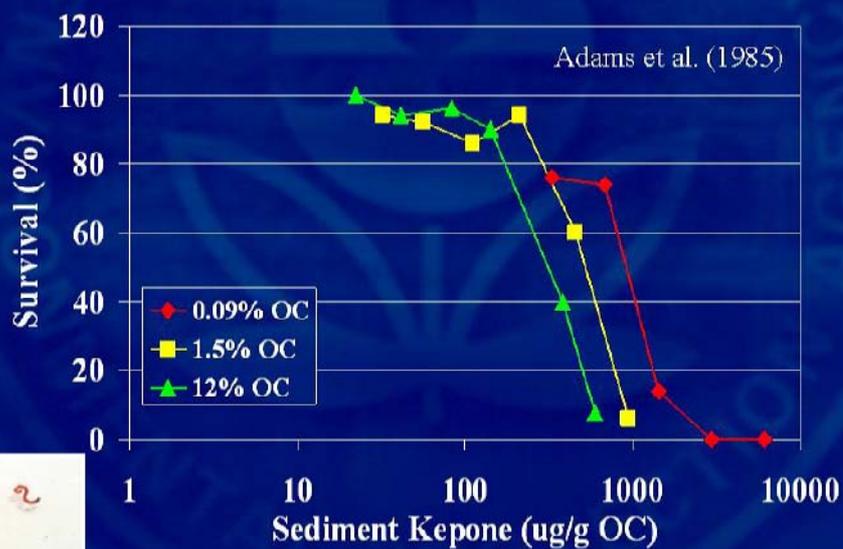
## Response of Midge Larvae to Kepone in Sediment -- Interstitial Water Normalization



## Conceptual Models of Chemical Exposure



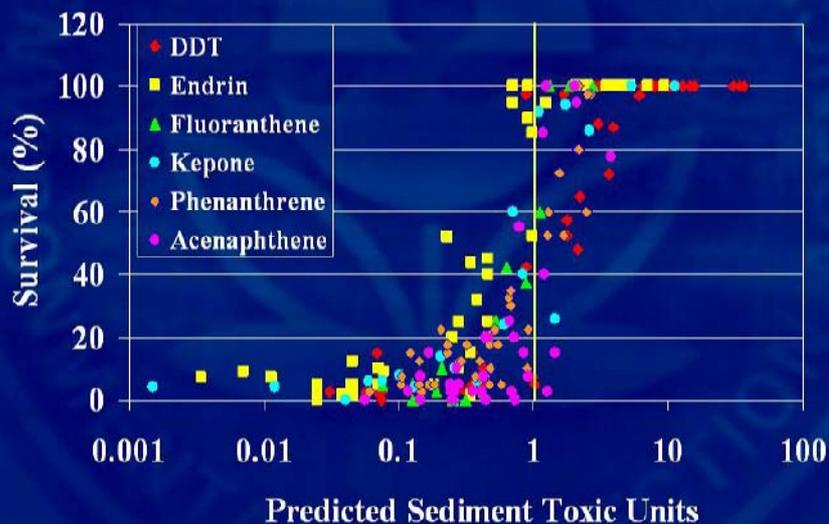
## Response of Midge Larvae to Kepone in Sediment -- Organic Carbon Normalization



## Establishing Biological Effect Concentrations in Sediment

- Basic Partitioning:  $C_{\text{SOC}} = C_{\text{IW}} * K_{\text{OC}}$
- Biological Effect:  $C_{\text{SOC}} = C_{\text{effect}} * K_{\text{OC}}$
- Sediment "Criteria":  $C_{\text{SOC}} = C_{\text{AWQC}} * K_{\text{OC}}$
- Equilibrium Partitioning Sediment Benchmarks (ESB)

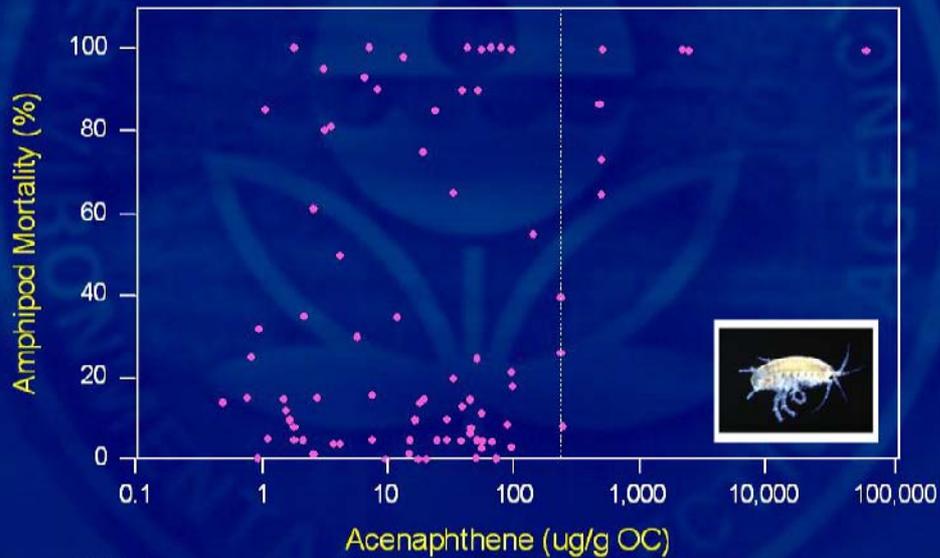
## EqP Effectively Predicts Toxicity in Spiked Sediments



USEPA 2004

## Mortality of Amphipods in PAH-Contaminated Sediments

Organic Carbon-Normalized Acenaphthene Concentration



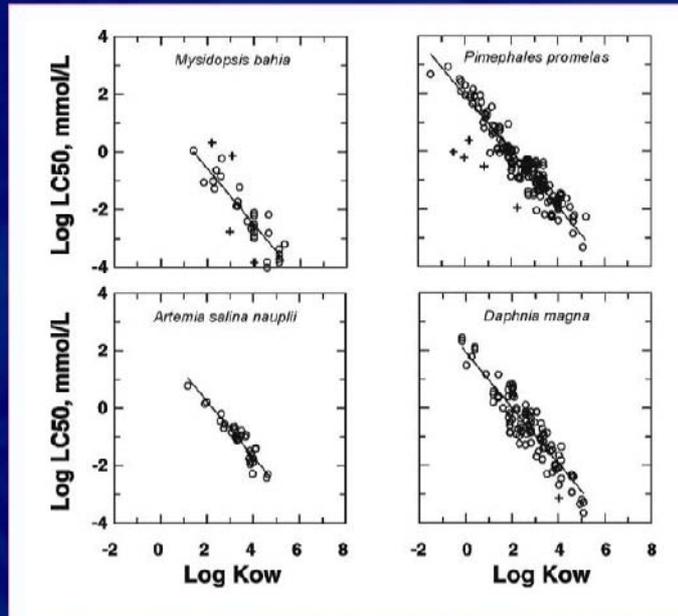
Swartz (1999)

## Issues for Developing PAH ESB

- There are over hundreds of PAH structures
- PAHs always occur as mixtures
- Needs for ESB development
  - Way to predict toxicity of all PAHs
  - How to integrate toxicity of mixtures
- PAH ESB available at:  
[www.epa.gov/nheerl/publications](http://www.epa.gov/nheerl/publications)

## Narcosis Toxicity Related to Kow

DiToro et al. 2000



## Toxicity of PAH Mixtures

- Chemicals with a common mode of action generally show additive toxicity
- Contribution of each chemical to the mixture is represented by its “Toxic Units”
- Toxic units are the ratio of the concentration to the concentration of that single chemical that would cause effects

## Derivation of PAH Mixture ESB

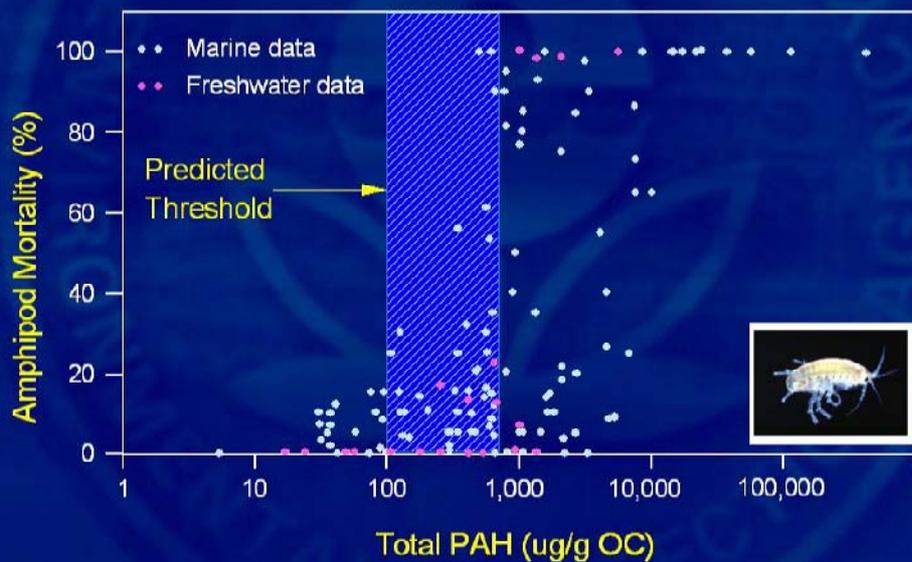
For each PAH:



Across all PAH:

$$\sum_{i=1}^n \frac{\text{Sediment [PAH ]}_i}{\text{ESB [PAH ]}_i}$$

## EqP Predicts Toxicity Threshold in Field Sediments

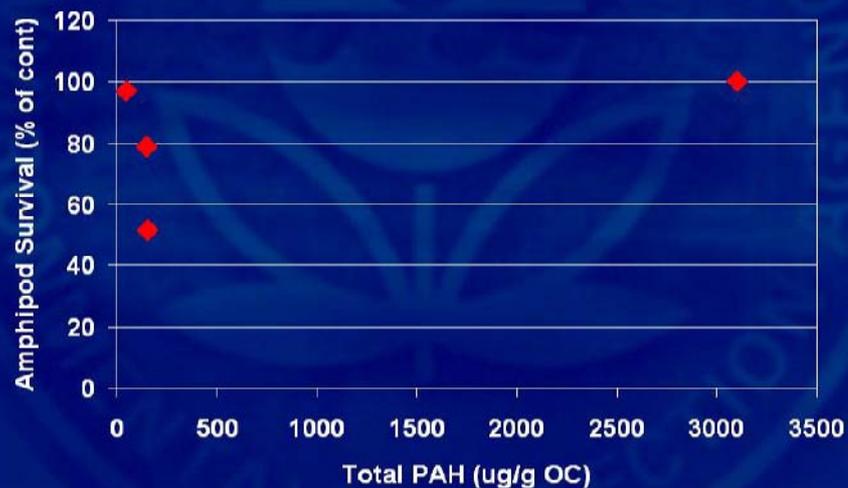


## Assessment Scenario: Harbor Site

- PAH contamination in harbor setting
- PAH analysis for “Priority Pollutant” PAHs
- Toxicity testing with amphipods (*Hyalella*)

## Total PAH v. Toxicity -- Harbor Sites

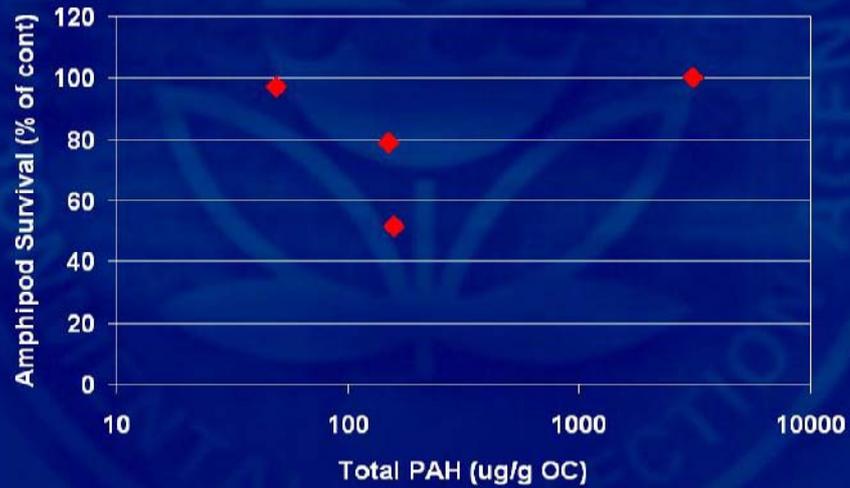
Sum of 7 unsubstituted PAH



Data adapted from Ankley et al. 1994; West et al. 2001

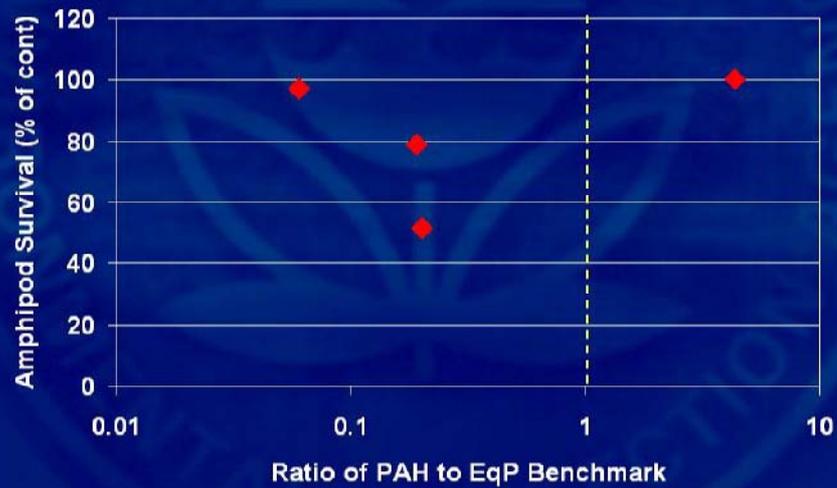
## Total PAH v. Toxicity -- Harbor Sites

Sum of 7 unsubstituted PAH



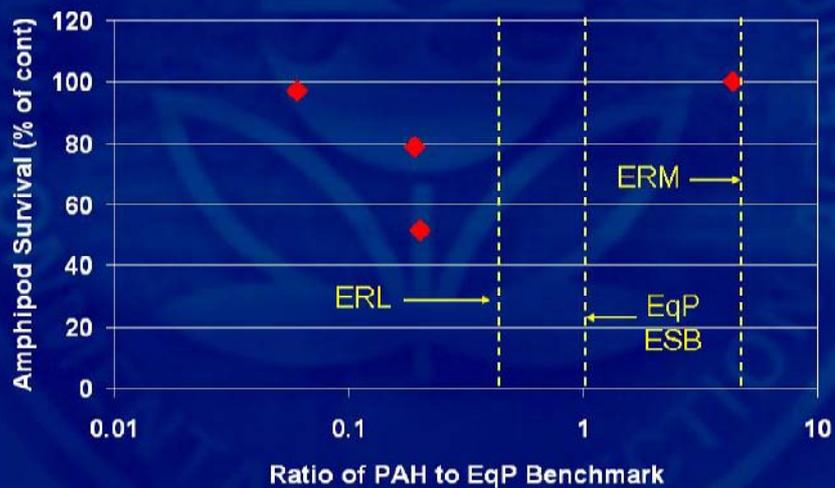
## EqP Guideline v. Toxicity -- Harbor Sites

Sum of 7 unsubstituted PAH



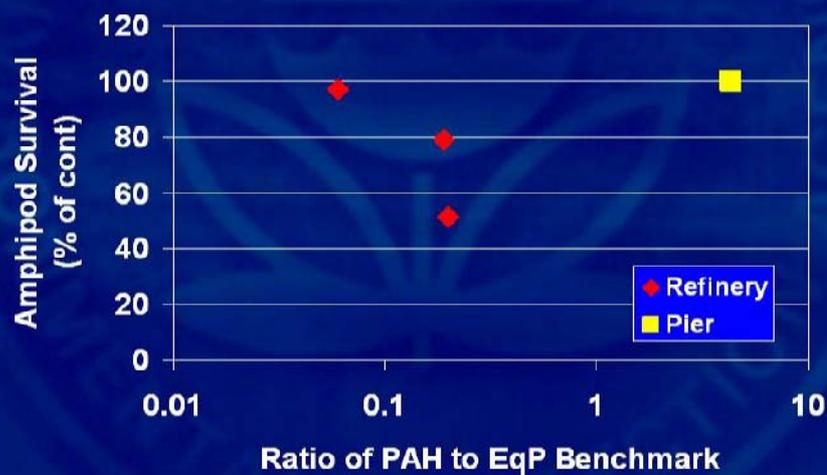
## SQGs v. Toxicity -- Harbor Sites

Sum of 7 unsubstituted PAH



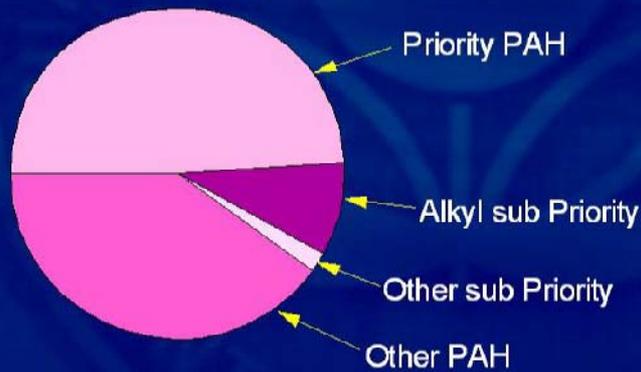
## EqP Guideline v. Toxicity -- Harbor Sites

Sum of 7 unsubstituted PAH



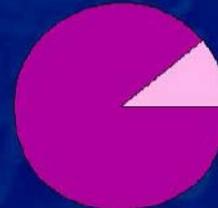
## Relative Composition of PAH Sources

Coke Plant Sediments

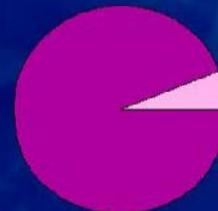


Burkhard et al. 1994

North Slope Crude



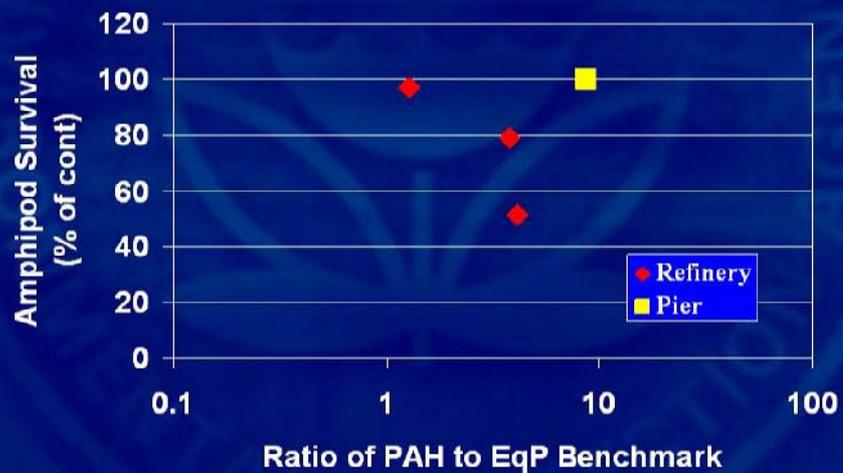
Weathered NS Crude



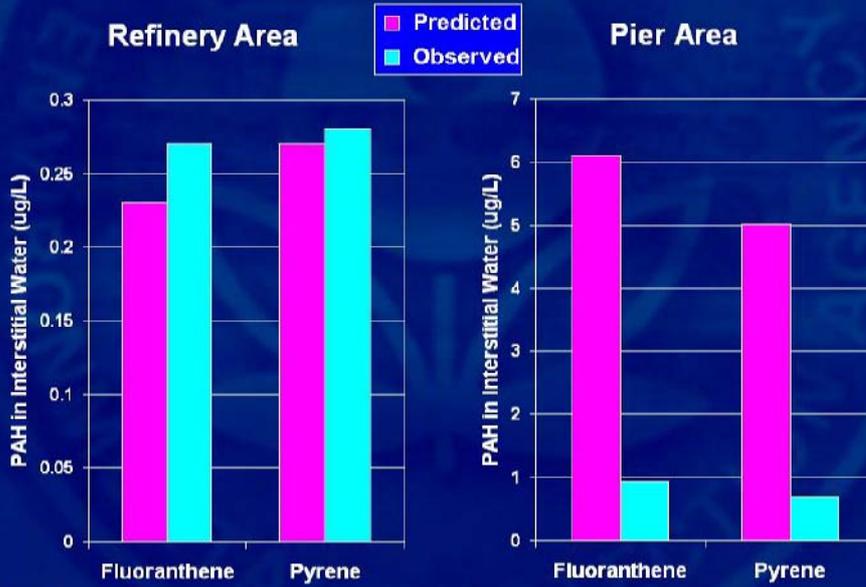
Stubblefield et al. 1995

## EqP Guideline v. Toxicity -- Harbor Sites

Adjustment of 2X for Pier, 20X for Refinery

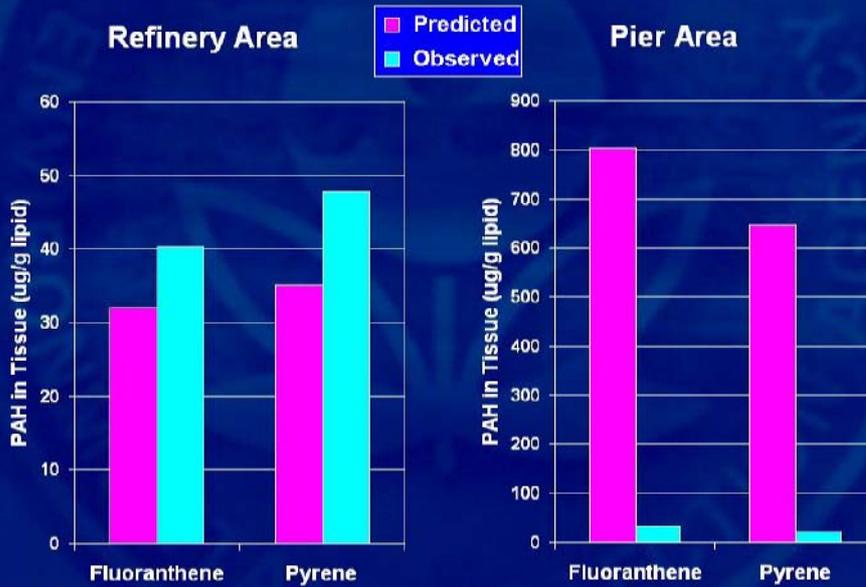


# PAH Partitioning Behavior



West et al. 2001

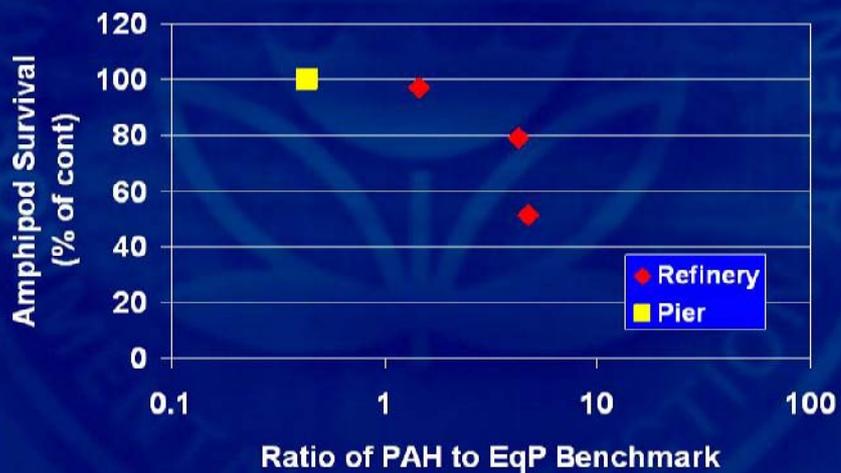
# PAH Accumulation in Oligochaetes



West et al. 2001

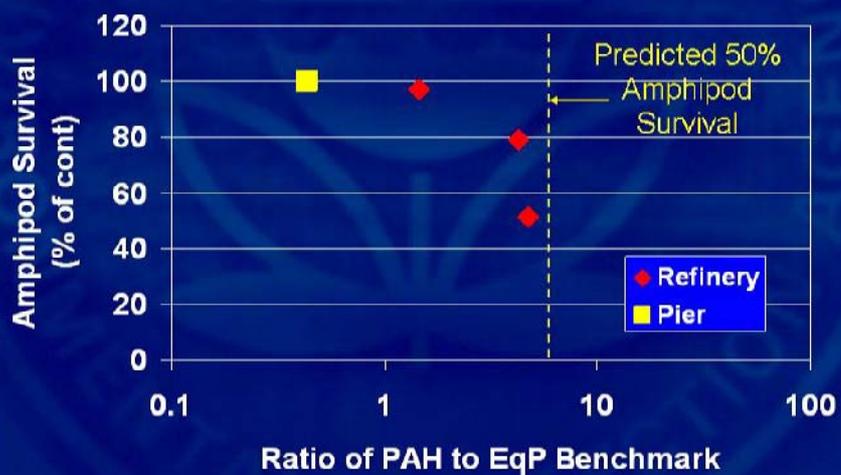
## EqP Guideline v. Toxicity -- Harbor Sites

Corrected for Alkylation and Bioavailability



## EqP Guideline v. Toxicity -- Harbor Sites

Corrected for Alkylation and Bioavailability



## Three Key Issues for Assessing PAH Potency in Sediment

- Sediment organic carbon
  - Commonly ranging from 0.5% to 15% (30-fold)
- Bioavailability (coal, soot, tire rubber)
  - Can easily create 100-fold difference
- Alkylation of PAH mixture
  - Easily a 10-fold difference
- In aggregate, the potential for a 30,000x range in threshold! (dwt, PP only)

## What Dwt PAH Concentration will Cause Effects? -- Scenario #1

- Start with 800 ug TPAH/g OC as toxicity threshold
- 0.5% OC, “normal” bioavailability, petroleum source
  - OC:  $800 \text{ ug/g OC} = 4 \text{ ug TPAH/g dwt}$
  - Bioavailability: no adjustment
  - Source:  $4 \text{ ug TPAH} / 20 = 0.2 \text{ ug ppPAH/g dwt}$
  - Expected threshold: 200 ug ppPAH/kg dwt

## What Dwt PAH Concentration will Cause Effects? -- Scenario #2

- Start with 800 ug TPAH/g OC as toxicity threshold
- 15% OC, coal present, coal tar source
  - OC:  $800 \text{ ug/g OC} = 120 \text{ ug TPAH/g dwt}$
  - Bioavailability:  $120 \text{ ug/g} \times 100 = 12,000 \text{ ug/g}$
  - Source:  $12,000 \text{ ug TPAH} / 2 = 6,000 \text{ ug ppPAH/g dwt}$
  - Expected threshold:  $6,000,000 \text{ ug ppPAH/kg dwt}$

## What To Do?

- Toxicity threshold can easily range from 200 to 6,000,000 ppb dwt as ppPAH
- How do I deal with this?
  - Measure organic carbon and normalize PAH concentrations (\$15/sample, 30x uncertainty)
  - Check for bioavailability issues (bioaccumulation, IW measurement, SPME)
  - Characterize the PAH mixture (subsets if necessary)

## What About Mechanisms Other Than Narcosis

- Some responses to PAH exposure are not those typically associated with narcosis
- Would a narcosis-based threshold protect against other effects?
- Puget Sound data for flatfish
  - Empirical effects threshold for liver lesions in sensitive species at 1,000 to 3,000 ppb dwt ppPAH
  - EPA ESB intent to prevent chronic toxicity to sensitive organisms
  - At 2% OC, normal bioavailability, and a 10x PAH source correction factor, the ESB is about 1,600 ppb dwt as ppPAH
  - But, it could also be 6,000,000 ppb!

## Strengths of EqP for Sediment Assessment

- Don't treat it as a "look-up" number – it's a framework for thinking about sediment contamination
- If you don't believe it, test it!
- Two basic assumptions
  - chemical partitions to interstitial water predictably
  - organism response is predictable from water-only toxicity data
- Both of these assumptions are completely testable
- Can be adjusted for different endpoints or management goals

## Which Sediment Guideline is Best?

- There are no bad guidelines, only guidelines used badly
- The issue is applying guidelines in ways that
  - Are consistent with their derivation
  - Recognize the implicit assumptions and uncertainties
  - Address the realities of PAH bioavailability and toxicity
  - Are consistent with the management objectives

***Todd Bridges***

Engineer Research and Development Center, US Army Corps of Engineers,  
Washington DC

**Corps National Perspective on PAH Policy and Research**

**Abstract:** Not available. Please see the author's Power Point presentation that follows.

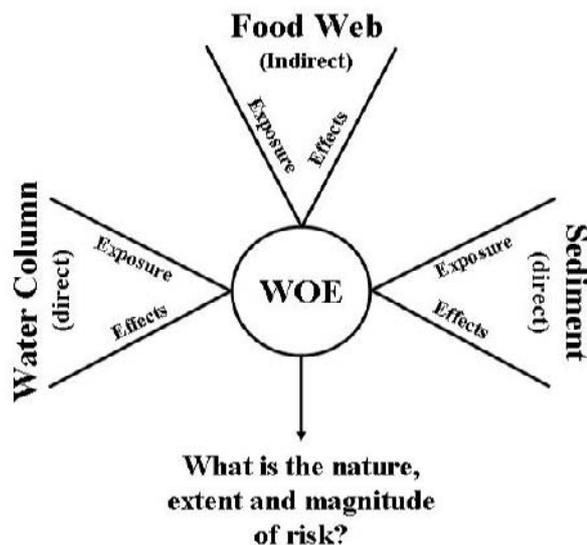
# USACE National Perspective on PAH Policy and Research

Todd S. Bridges, Ph.D.  
U.S. Army Corps of Engineers  
Engineer Research and Development Center  
Vicksburg, MS

## Collaborators

Dr. Jeffery Steevens, USACE-ERDC  
Mr. Mark Reiss, EPA Region 2  
Dr. Susan Kane-Driscoll, MCA

## General Framework for DM Evaluations: Lines and Weight of Evidence in Sediment Assessment



# PAHs

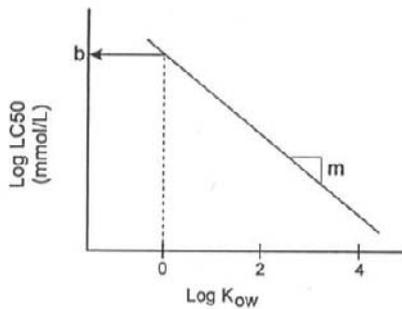
1. Existing sediment screening tools focus on protection of benthic invertebrates
2. EQP provides approach for predicting bioaccumulation
3. TLM provides approach to predict toxicology
4. Existing limitations include issues related to bioavailability, metabolism, and trophic transfer

## Use of SQG in Assessing PAHs

- Wide range of SQG values for individual and total PAHs (empirical and mechanistic): ERL/ERM, TEL/PEL, AET, EqP
  1. Focused on benthos
  2. Error rates vary in predictions about effect or lack thereof
  3. SQG values most productively used in a weight-of-evidence approach with other lines-of-evidence
- Confounding issues: Bioavailability (black carbon), Metabolism, Mixtures, Sublethal effects
- For the above reasons predicting the affects of PAHs can be problematic
  - E.g., work to-date on CA SQO

# Target Lipid Model

- Critical body burdens are determined by relationship between  $LC_{50}$  values and  $K_{OW}$  values.

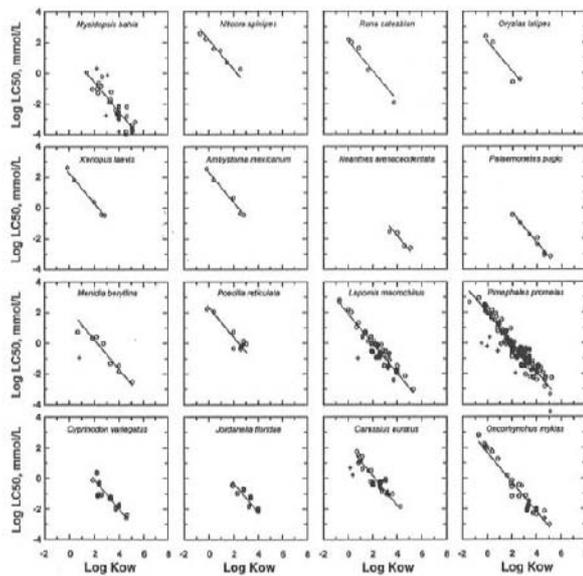


- Relationship is known as the linear free energy relationship (LFER)

- Y-intercept is the lipid normalized critical body burden

From Di Toro et al., 2000

# Target Lipid Model

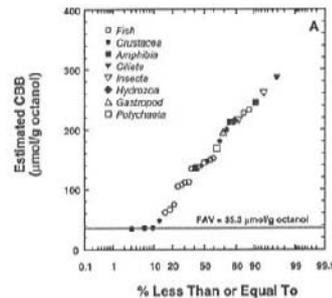


From Di Toro et al., 2000

## Target Lipid Model

- Evaluate probability distribution of critical body burdens for non-polar organics
- Determine the Final Acute Value (FAV)
  - Concentration that is protective of 95% of organisms

FAV / Acute:Chronic = FCV



From Di Toro et al., 2000

## Target Lipid Model

- Use FCV of 3.79 umoles/g octanol and normalize to wet tissue concentrations

Organism	Percent Lipid	Narcosis FCV umoles/g tissue
<i>Macoma nasuta</i> (clam) <sup>1</sup>	4.83	0.18
<i>Mercenaria sp.</i> (clam) <sup>1</sup>	0.88	0.03
<i>Leptocheirus plumulosus</i> (amphipod) <sup>2</sup>	1.78	0.07
<i>Neanthes arenaceodentata</i> (polychaete) <sup>3</sup>	2.15	0.08
<i>Nereis virens</i> (polychaete) <sup>1</sup>	7.25	0.27
Average	3.38	0.13

1. USACE BSAF Database: [www.wes.army.mil/el/dots/database.html](http://www.wes.army.mil/el/dots/database.html)
2. Lotufo et al., 2001.
3. Lotufo et al., 2000.

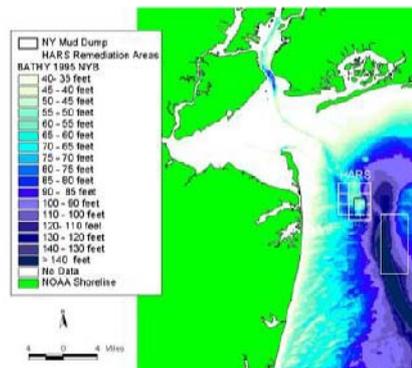
# Target Lipid Model

- Problems
  - Relies on narcosis mode of action
  - Does not adequately factor in metabolism of compounds
  - Linkage between sediment, invertebrates, and fish tissue body burden is uncertain

## Background: New York Harbor

### Issues

- Mud dump site in use since 1800's
- Variety of material
  - Garbage, sewage, sediment, industrial waste
- DM disposal site designated as the Historic Area Remediation Site (HARS) in Sept 1997
- Sediment does not go to HARS if it is not considered "remediation material"
  - Sediment is not "toxic"
  - Contaminants do not bioaccumulate to levels that pose a risk
- Development of HARS technical evaluation framework by NY District and EPA Region 2.

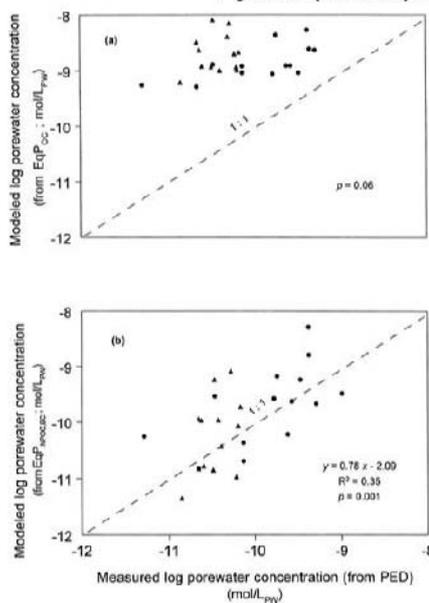


# TEF Bioaccumulation Assessment

1. Bioaccumulation test
  - 28-day bioassay
  - *Nereis virens* and *Macoma nasuta*
2. Adjust reported tissue concentration to steady-state concentration
3. Interpret result using dose-based species sensitivity distribution
4. Currently developing approach to integrate CDF to population model

## Importance of Bioavailability

Modeled versus measured porewater concentration of Pyrene (circles) and phenanthrene (triangles).



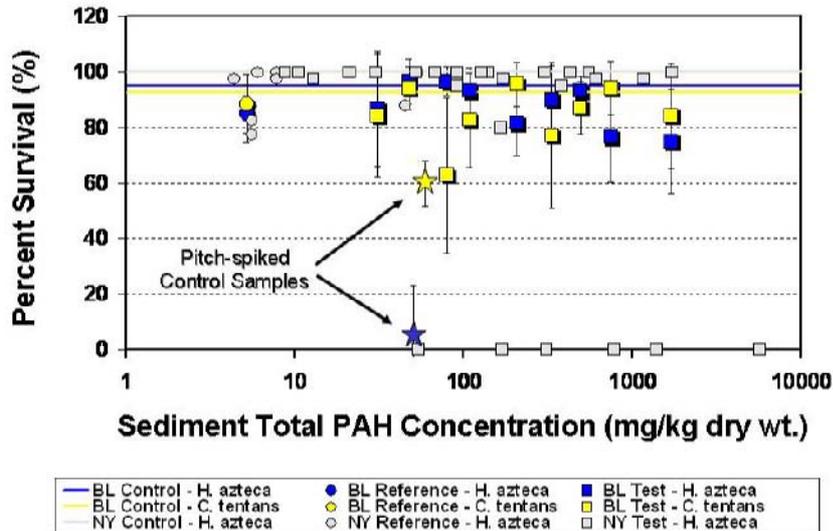
- Porewater was measured using polyethylene disk (PED) inserted in sediment.

- Top graph shows modeled concentration using nonpyrogenic carbon in normalization.

- Bottom graph shows modeled concentration using nonpyrogenic and black carbon in normalization

From Vinturella et al., 2004

## Role of Carbon on Bioavailability and Toxicity

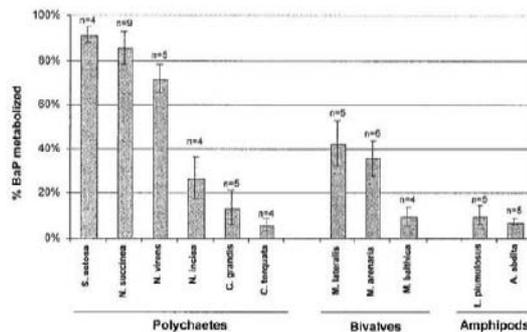


DRAFT PRELIMINARY INFORMATION  
Do not cite



## Importance of Metabolism

- PAH are metabolized at different rates (PAH and organism dependent)



From Rust et al., 2004

- Because of metabolism in fish, not appropriate to link fish tissue concentration to an effect

## Steady-State Study

- Determine kinetics for non-polar organics
  - Uptake / elimination kinetics
  - Time-to-steady state (TSS)
  - 28 day fraction of steady state (fss)
  - Steady state BSAF
- Study design
  - *Macoma nasuta* (120 day exposure)
  - *Nereis virens* (56 day exposure)
  - Two sediments: Arthur Kill and Newark Bay
  - 7 sample points during study
  - 16 PAHs, 15 pesticides, 33 PCBs, 17 dioxins / furans

### Results

#### *Macoma nasuta*

Analyte	Sediment	K <sub>ow</sub>	TSS <sub>95%</sub> (days)	F <sub>ss</sub> (28 day)	BSAF <sub>ss</sub>
Fluoranthene	Arthur	4.22	32	0.93	0.73
	Newark	4.22	18	1.00	0.18
Pyrene	Arthur	5.07	<b>43</b>	0.86	0.09
	Newark	5.07	18	1.00	0.32
PCB 28	Arthur	5.67	<b>58</b>	<b>0.76</b>	0.27
	Newark	5.67	25	0.97	0.38
Total DDT	Arthur	6.22 – 6.96	<b>102</b>	<b>0.56</b>	0.24
2,3,7,8-TCDD	Arthur	6.46	<b>144</b>	<b>0.44</b>	0.78
	Newark	6.46	<b>55</b>	<b>0.78</b>	1.19
PCB 138	Arthur	6.83	<b>63</b>	<b>0.73</b>	0.20
	Newark	6.83	26	0.96	0.29

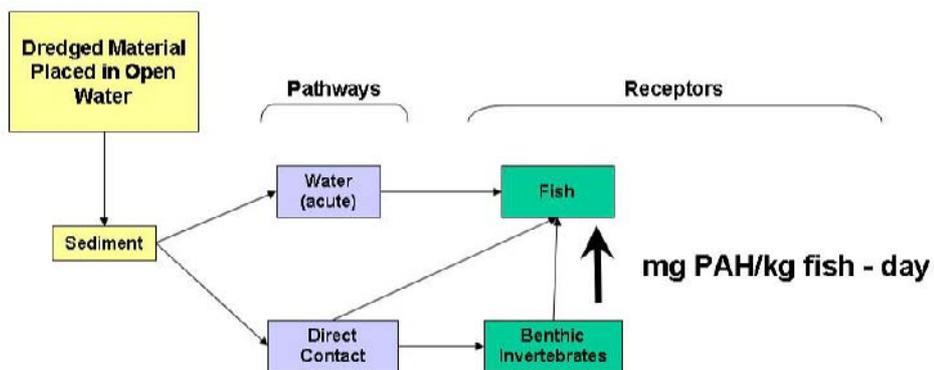
Boese *et al.* (1997): TSS for PCBs ranged from 63 – 267 days

# Results

## *Nereis virens*

Analyte	Sediment	K <sub>ow</sub>	TSS <sub>95%</sub> (days)	F <sub>ss</sub> (28 day)	BSAF <sub>ss</sub>
Fluoranthene	Arthur	4.22	4	1.00	0.02
	Newark	4.22	NA	NA	NA
Pyrene	Arthur	5.07	11	1.00	0.03
	Newark	5.07	15	1.00	0.06
PCB 28	Arthur	5.67	18	1.00	0.19
	Newark	5.67	25	1.00	0.15
Total DDT	Arthur	6.22 – 6.96	<b>85</b>	<b>0.63</b>	0.79
2,3,7,8-TCDD	Arthur	6.80	<b>72</b>	<b>0.69</b>	0.40
	Newark	6.80	<b>82</b>	<b>0.64</b>	0.51
PCB 180	Arthur	7.36	<b>41</b>	0.87	0.26
	Newark	7.36	<b>37</b>	0.90	0.23

## Metabolized Compounds/Inorganics: Benchmark Dose for PAH



**Advantages of Dietary Dose:**  
**Captures sediment bioavailability**  
**Addresses metabolism**

# Benchmark Dose for PAH

## Appropriate toxicity studies:

- 11 Studies
- 8 species at various life stages
- Individual PAHs
- Measures of Exposure
  - Concentration in water or tissue of food
  - Doses in diet (5) or water (6)
- Measures of Effect
  - Survival (2), growth (5), reproduction (4)
  - Neoplasms (3)

## Data set for derivation of PAH CDF

Common Name	Species	Life Stage at start of Experiment	Chemical	Exposure Route	Exposure Duration	Effect	Measured NOAEL	Estimated NOAEL	LOAEL	Geometric Mean of NOAEL and LOAEL	Citation
							mg/kg-d	mg/kg-d	mg/kg-d	mg/kg-d	
English sole	<i>Parophrys vetulus</i>	Juvenile	BaP	Diet	10 or 12 days	Reduced growth		0.14	0.45	0.25	Rice et al., 2000.
Rainbow trout	<i>Salmo gairdneri</i>	Adult	BaP	Diet	18 months	Increased liver neoplasms, reduced body weight		0.26	0.92	0.46	Hendricks et al., 1985
Chesook salmon	<i>Oncorhynchus tshawytscha</i>	Juvenile	Mixture of PAHs	Diet	18 days	Growth		0.37	1.15	0.65	Meador et al., in prep.
Fathead minnow	<i>Pimephales promelas</i>	21-d larvae	BaP	Water	4 months	Reduced survival of F2 larvae	0.6		1.0	0.77	White et al., 1999
Atlantic croaker	<i>Micropogonias undulatus</i>	Adult	BaP	Diet	30 days	Reduced ovarian growth, decreased estradiol and testosterone		0.79	2.4	1.4	Thomas, 1988
Guppy	<i>Poecilia reticulata</i>	6-11 day larvae	DMBA	Water	4 weeks	Increased hepatic neoplasms 24 and 37 weeks after exposure	0.71		8	2.4	Hawkins et al., 1989
Fathead minnow	<i>Pimephales promelas</i>	Adults (> 2 months)	Fluoranthene	Water	14 weeks	Reduced egg production		2.1	6.6	3.7	Diamond et al., 1995
Fathead minnow	<i>Pimephales promelas</i>	Adult (> 4 months)	Anthracene	Water	4 weeks	Reduced hatching success in eggs exposed to SUVR	5.6		11	8.9	Tilghman Hill and Cris, 1991
Fathead minnow	<i>Pimephales promelas</i>	23-33-d larvae	Fluoranthene	Water	32 days	Reduced survival and growth	13		26	18	Spohr et al., 1999
Zebrafish	<i>Danio rerio</i>	Juveniles (2 months)	DMBA	Diet	12 weeks	Increased neoplasms and decreased body weight	11		56	25	Spitzbergen et al., 2000, J.M. Pearce-Enoch et al., 1995
Rainbow trout	<i>Oncorhynchus mykiss</i>	1-7 d Fry	Phenanthrene	Water	60 days	Decreased growth		27	84	41	

## Conversion of PAH Tissue and Water Concentrations to PAH Dose

$$\text{Dose} = \text{mg PAH ingested/kg fish-d}$$

$$\text{Dose} = (E_w * G_V / W_B) * C_w$$

$E_w$  = Kow derived gill chemical uptake efficiency

$G_V$  = Gill ventilation rate (L/d)

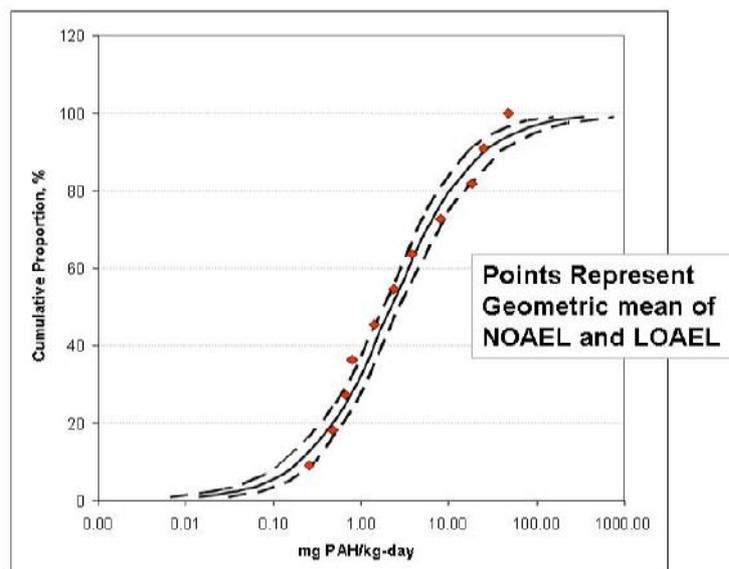
- weight of organism,  $O_2$  saturation, temperature

$W_B$  = wet weight of the organism (kg)

$C_w$  = Concentration of PAH in water (mg/L)

(Arnot and Gobas, 2004)

## Cumulative Distribution Function-PAHs Dose

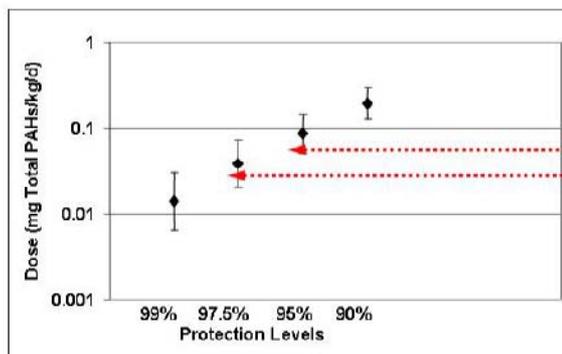


# PAH Benchmark Dose

- Derived a dose-based toxicity reference value
- Can be used to back calculate to:
  - Sediment concentrations (BSAF, OC, lipid)
  - Invertebrate tissue (prey ingestion rate)

Protection Level	Doses (mg/kg/d)		
	LCL	Mean	UCL
99%	0.00646	0.0140	0.0305
97.5%	0.0208	0.0391	0.0736
95%	0.0511	0.0863	0.146
90%	0.129	0.196	0.298

## Benchmark-derived Tissue Concentrations: Comparison to Reported Residue Effect Levels

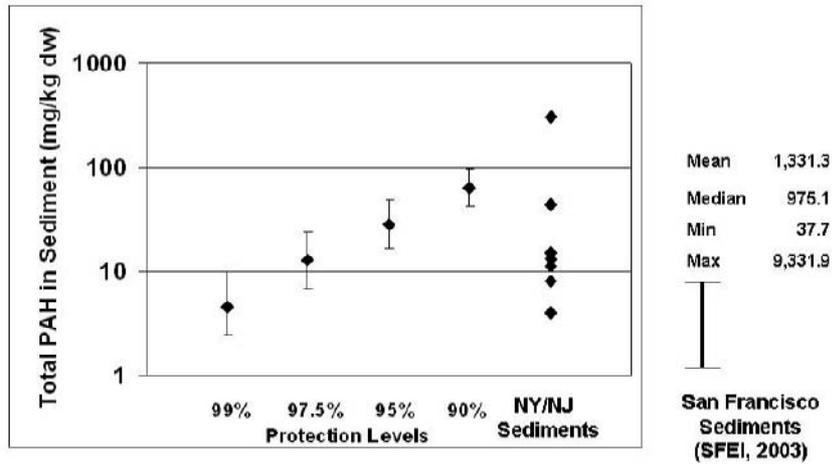


Reduced growth in English Sole (*Pleuronectes vetulus*) fed *Armandia* exposed to sediment, (Rice 2000)

Reduced growth of Pink salmon (*Oncorhynchus gorbuscha*) fed food spiked with crude oil, (Carls 1996)

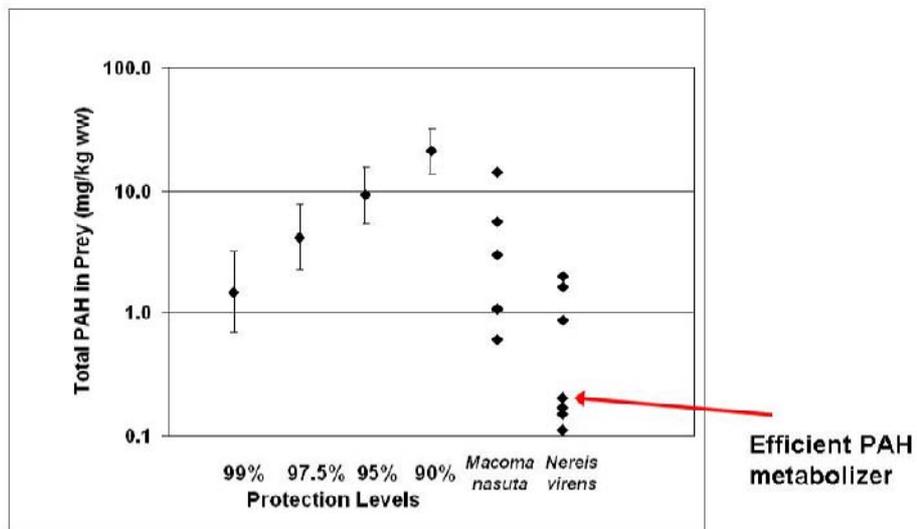
*When have you achieved sufficient protection?*

## Benchmark-derived Sediment Concentrations: Measured Concentrations in NY/NJ Sediments



*Some sediments exceed benchmark; potential for effects on fish*

## Benchmark-derived Prey Concentrations: Measured Concentrations in NY/NJ Bioaccumulation Tests



# Conclusions and Issues

- **Importance of the exposure in calculating risk**
  - Bioavailability of PAHs will vary among sediments depending on the nature of carbon
  - Length of exposure will vary depending on the physical processes relevant to the scenario under consideration
    - Navigation channels are sediment traps
    - Disposal sites may be net erosional or depositional
  - Behavior of target receptors: demersal v pelagic fish
  - Calculating exposure point concentrations: Total PAHs v 34 PAHs v alkylated PAHs
- **Importance of toxicology in calculating risk**
  - Which toxicological endpoints are most relevant
  - The role of other contaminants
- **Realities: 1/3 of all sediments in US exceed 1 ppm (EPA 1997)**

**Daniel R. Oros**

San Francisco Estuary Institute, Oakland, CA

## **PAH in San Francisco Bay: A 10-year Retrospective of Monitoring in an Urbanized Estuary**

**Abstract:** Polycyclic aromatic hydrocarbons (PAH) are widespread contaminants in the San Francisco Bay. Several exceedances of water quality criteria raise the possibility that PAH may be impacting aquatic biota. The Regional Monitoring Program for Water Quality (RMP) has been monitoring PAH in the San Francisco Bay sediments since 1993. PAH concentrations, spatial distributions, and temporal trends were determined in San Francisco Bay sediments samples that were collected at fixed stations over the period 1993-2001. Surface sediments (top 5 cm) were collected at 26 sampling stations. The mean total PAH ( $\Sigma$ PAH) concentration in sediments was spatially distributed as Central Bay (230 mg/kg of organic carbon, OC), South Bay (217 mg/kg OC), North Estuary (96 mg/kg OC), Extreme South Bay (87 mg/kg OC), and Delta (31 mg/kg OC). Overall, the mean  $\Sigma$ PAH concentrations were significantly higher in the Central Bay and South Bay segments compared to the North Estuary, Extreme South Bay and Delta segments, and the Delta was significantly lower than all other segments (Kruskal-Wallis,  $H=156.94$ ,  $df=4$ ,  $p=0.000$ ). In addition, no significant difference in  $\Sigma$ PAH concentration was found between the Central Bay and South Bay. Temporal trend analysis showed a statistically significant temporal trend in  $\Sigma$ PAH concentration at only 1 of the 26 sampling sites located throughout the Bay (San Pablo Bay, significant decrease,  $p=0.024$ ,  $r^2=0.314$ ,  $n=16$ ), which suggests that  $\Sigma$ PAH concentrations in San Francisco Bay surface sediments generally remained constant from 1993-2001. Based on their relative contribution to the estimated total maximum PAH loading (10,700 kg/yr) into the Bay, the PAH loading pathways are ranked as storm water runoff (~51%) > tributary inflow (~28%) > wastewater treatment plant effluent (~10%) > atmospheric deposition (~8%) > dredged material disposal (~2%). Source analysis using PAH isomer pair ratios as indicators showed that PAH are derived primarily from combustion of fossil fuels/petroleum (gasoline, crude oil, and coal) and biomass (wood and grasses), with minor amounts of PAH derived from direct petroleum input. The total PAH sediment quality threshold of 1000 ppb, which has been previously suggested to protect estuarine fish against adverse health effects (Johnson et al., 2002), was frequently exceeded at individual monitoring stations (19 of the 26 stations or 73%) throughout the sampling period. The adverse health effects that might occur in English sole and other bottom dwelling fish include DNA damage, liver lesions and reproductive abnormalities due to exposure to PAH in sediments. Modeling results have shown that the predominant loss pathway for PAH is degradation in sediments, and unless external loading levels of PAH are controlled, the Bay is not expected to recover rapidly.

## **PAH in San Francisco Bay: A 10-year Retrospective of Monitoring in an Urbanized Estuary**

Daniel R. Oros

San Francisco Estuary Institute  
Oakland, CA



### **Recent Papers on PAH in SF Bay**

Oros, D.R., Ross, J.R.M. PAH in SF Estuary sediments. Marine Chemistry 2004, 86, 169-184.

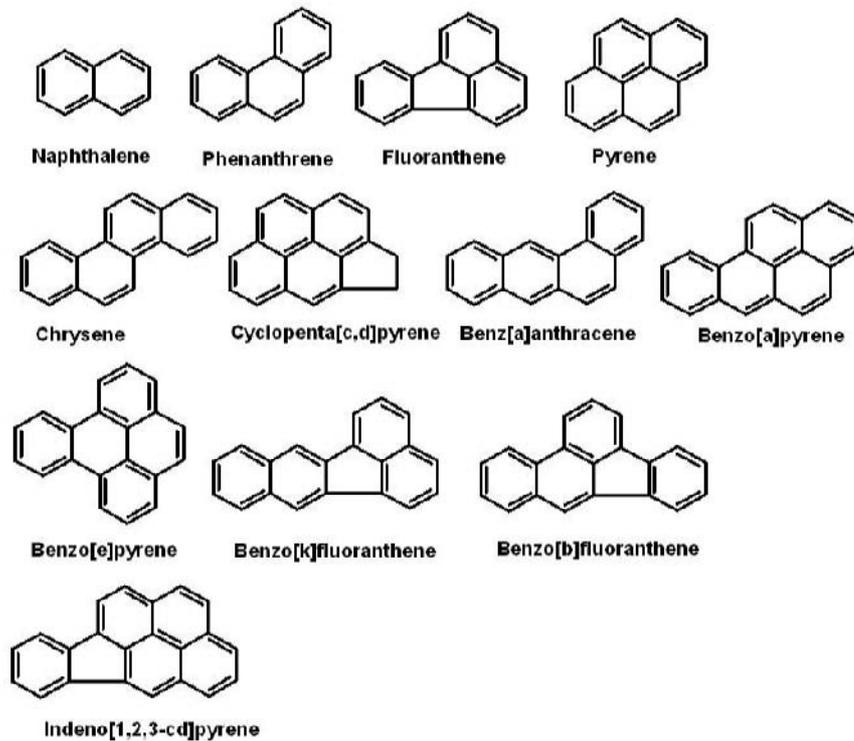
Ross, J.R.M., Oros, D.R. PAH in the in SF Estuary water column. Chemosphere 2004, 57, 909-920, 2004.

Oros, D.R., Ross, J.R.M. PAH in SF Estuary bivalves. Marine Environmental Research 2005, 60, 466-488.

Oros, D.R., Ross, J.R.M., Spies, R.B., Mumley, T. Polycyclic aromatic hydrocarbon (PAH) contamination in San Francisco Bay: A 10-year retrospective of monitoring in an urbanized estuary (submitted to Environmental Research)

## Why are PAH of concern?

- Genotoxic
- Mutagenic
- Carcinogenic
- Ubiquitous
- Limited or no control of input



## How do PAH enter the estuary?

### Permitted Dischargers

- Wastewater Treatment Plants – 40 in the Bay Area
- Industry – 30 in the Bay Area (e.g., chemical manufacturers, steel producers, refineries and utilities)
- Municipalities – 30 in the Bay Area

### Major Mobile Sources

- Vehicular traffic (Bay Area 2nd in congestion in U.S.)
- Marine Vessels (tankers, freighters, container ships, cruise ships, military, tugs, pilots, fishing, and ferries)
- Trains (passenger and commerce)

## Combustion of Refined Petroleum Products



Vehicular Traffic



Trains



Industrial Emissions



Ferries



Fishing Boats



Container ships and Tankers

## Natural and Intentional Burning of Biomass



Fireplaces



Natural Fires



Campfires

## Uncontrolled, Accidental, and Intentional Input



Street Runoff



Spills (e.g., crude oil)



Creosote Treated Pilings



Piped Discharges

What do we know about PAH in SF Bay sediments  
(upper 5 cm)?

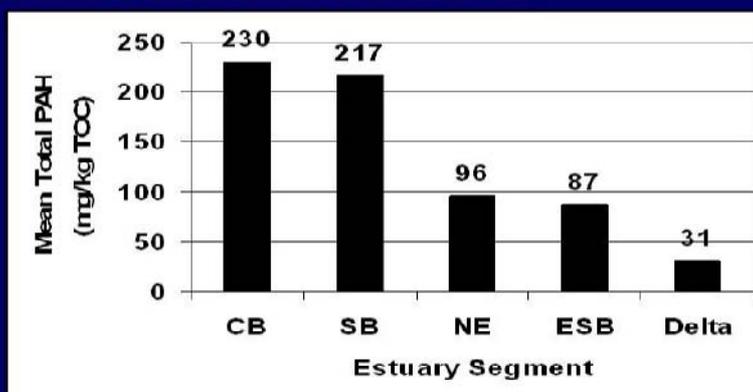
- Spatial distributions
  - Individual stations
  - Bay segments
- Temporal trends
  - Individual stations
  - Seasonal influences
- Sources
  - Combustion
  - Fossil fuels
  - Biomass burning



## Determination of Spatial Distributions

- 25 PAH were summed ( $\Sigma$ PAH) for each station
- $\Sigma$ PAH concentrations were TOC normalized (statistically significant relationship). Mean TOC in Bay in 1.1%
- Stations were grouped into 5 segments: Delta, North Estuary, Central Bay, South Bay, and Extreme South Bay
- Comparisons between segments, seasons, and stations were conducted using the non-parametric Kruskal-Wallis test

## Results: Spatial Distributions



- CB and SB  $\Sigma$ PAH were significantly higher than NE, ESB and Delta segments
- SB and CB were not significantly different
- Delta was significantly lower than all other segments

### Distributions of PAH in sediments (1993-2001)

	Mean	Mean	Mean	Mean	Mean
Segment	Total	Wet	Dry	%OC	%Fines
Delta	31	51	17	0.6	33
NE	96	111	84	1.2	72
CB	230	237	224	0.9	58
SB	217	259	184	1.2	79
ESB	87	83	90	1.4	71
Baywide	132	148	120	1.1	63

### HPAH / LPAH in Sediments (1993-2001)

	Mean	Mean	Mean	Mean	Mean
Segment	Total	Wet	Dry	%OC	%Fines
Delta	14	16	12	0.6	33
NE	7	7	7	1.2	73
CB	6	7	6	0.9	58
SB	7	7	7	1.2	79
ESB	6	6	6	1.4	71
Baywide	8	9	7	1.1	63

## Results: Spatial Distributions

- Individual station mean Total PAH range:  
31-347 mg/kg OC (highest at Horseshoe Bay)  
120-3769 ng/g dry wt (highest at San Pablo Bay)
- Highest mean Total PAH were found at:  
Horseshoe Bay (347 mg/kg OC or 2544 ng/g dry wt)  
Richardson Bay (316 mg/kg OC or 2708 ng/g dry wt)  
both significantly ( $p < 0.0005$ ) higher than other stations
- Lowest mean Total PAH were found at:  
San Joaquin River (31 mg/kg OC or 120 ng/g dry wt)  
Sacramento River (31 mg/kg OC or 241 ng/g dry wt)

## Results: Spatial Distributions

### Seasonal Influences

- Wet season (Jan-March): total PAH at Horseshoe and Richardson Bays were significantly ( $p < 0.0005$ ) higher than the San Joaquin River and Davis Point stations
- Dry season (July-Sept): total PAH at Horseshoe Bay, Richardson Bay, San Pablo Bay and Alameda stations were significantly higher ( $p < 0.0005$ ) than San Joaquin River and Sacramento River stations (31 mg/kg OC)
- No other significant differences were found

## Determination of Temporal Trends

- $\Sigma$ PAH were first normalized to OC by multiple linear regression analysis
- Trends for  $\Sigma$ PAH were examined for each station by linear regression analysis using the  $\ln(\text{rescaled residual})$  as the dependent variable and sampling date as independent variable
- A significant positive slope ( $p < 0.05$ ) indicated an increase, a significant negative slope a decrease, and a lack of significance no detectable trend in  $\Sigma$ PAH at a station over time

## Results: Temporal Trends

### Station Analysis

- A significant ( $p < 0.05$ ) decreasing trend in  $\Sigma$ PAH was found only at San Pablo Bay (1 of 26 stations)
- Trends were not found at any other stations, which suggests that  $\Sigma$ PAH levels remained constant over the period 1993-2001

### Seasonal Comparison

- Sacramento River and Oyster Point showed significantly higher  $\Sigma$ PAH in the wet season than the dry season
- No other significant differences were found

## Determination of Possible Sources

- PAH isomer pair ratios were used as diagnostic indicators to identify possible sources (isomers have similar partitioning behavior and solubility)

Anthracene / Anthracene + Phenanthrene

Benz[*a*]anthracene / Benz[*a*]anthracene + Chrysene

Fluoranthene / Fluoranthene + Pyrene

Indeno[1,2,3-*c,d*]pyrene / Indeno[1,2,3-*c,d*]pyrene + Benzo[*g,h,i*]perylene

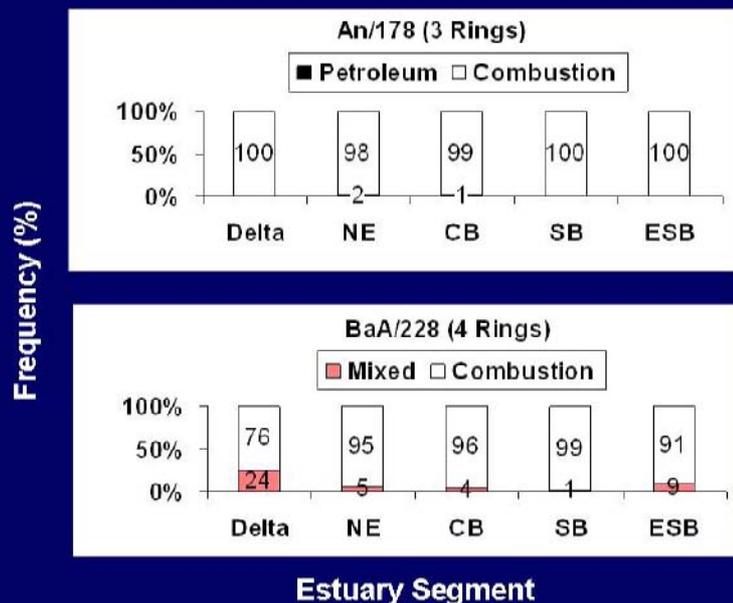
## Determination of Possible Sources (cont'd)

- Bar plots of PAH isomer pair ratios were generated to show estimated frequency (%) of PAH from the various sources in each segment
- PAH isomer pair ratios determined from SF Estuary were compared to PAH isomer pair ratios from known environmental, petroleum, and single-source combustion sources (Yunker *et al.*, 2002)

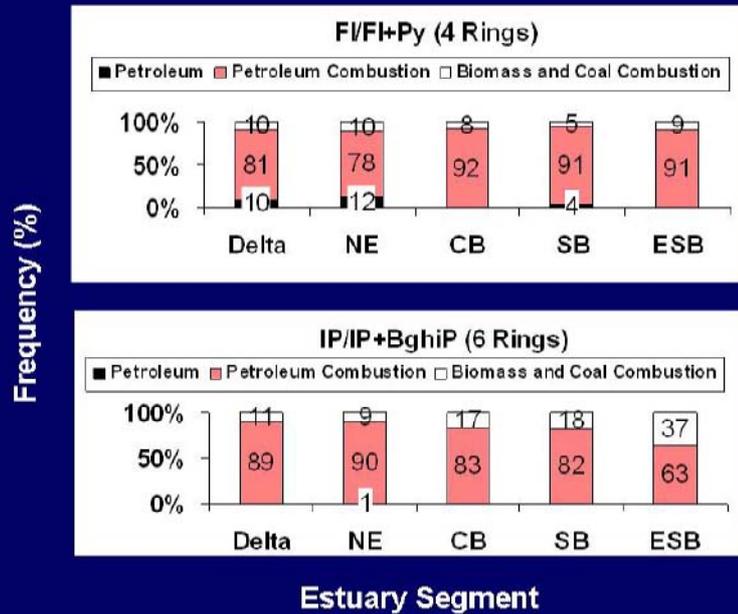
## Use of PAH isomer Pair Ratios to Identify Sources

Source	PAH Isomer Pair Ratio			
	An/178	BaA/228	Fl/Fl+Py	IP/IP+BghiP
Petroleum (unburned)	<0.10	<0.20	<0.40	<0.20
Petroleum combustion			0.40-0.50	0.20-0.50
Petroleum and combustion (mixed)		0.20-0.35		
Combustion	>0.10	>0.35		
Biomass and coal combustion			>0.50	>0.50

Based on Yunker et al. (2002)



Bar plots showing frequency (%) of source material occurrence in each estuary segment



Bar plots showing frequency (%) of source material occurrence in each estuary segment

## Summary of PAH Source Analysis

### Major Sources

Petroleum and Fossil Fuel Combustion

### Minor Sources

Biomass Burning

wood, wood soot and grasses

Unburned Petroleum

Shale Oil, Lube oil and Creosote

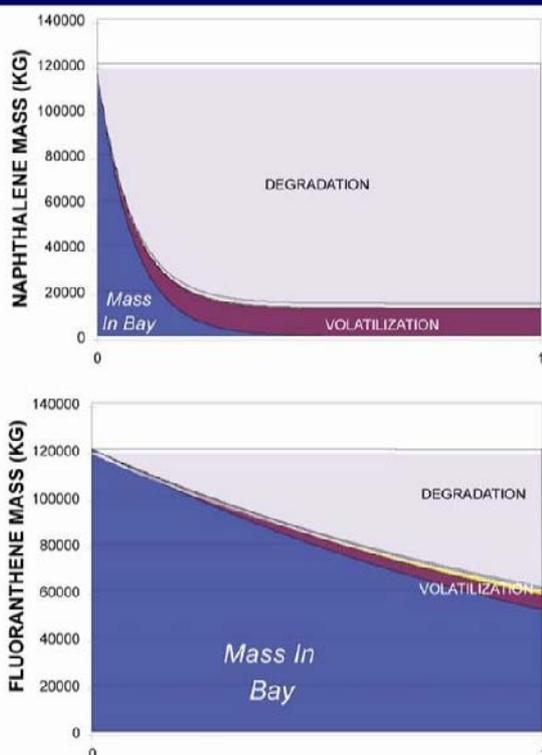
## Estimation of total PAH loads (kg/yr) from various sources

Source	Estimated Minimum Load	Estimated Maximum Load	% of $\Sigma$ PAH Load <sup>2</sup>
Stormwater Runoff	130	5500	51
Tributary Inflow		3000	28
Effluent Discharge	200	1100	10
Atmospheric Deposition		890	8
Dredged Material Disposal		210	2
<b>Total PAH Load<sup>1</sup></b>	<b>330</b>	<b>10700</b>	<b>100</b>

<sup>1</sup>This table cited from Greenfield and Davis (2005).

<sup>2</sup>Based on the estimated maximum load.

•Total PAH annual load to the Bay is estimated at 10,700 kg/yr



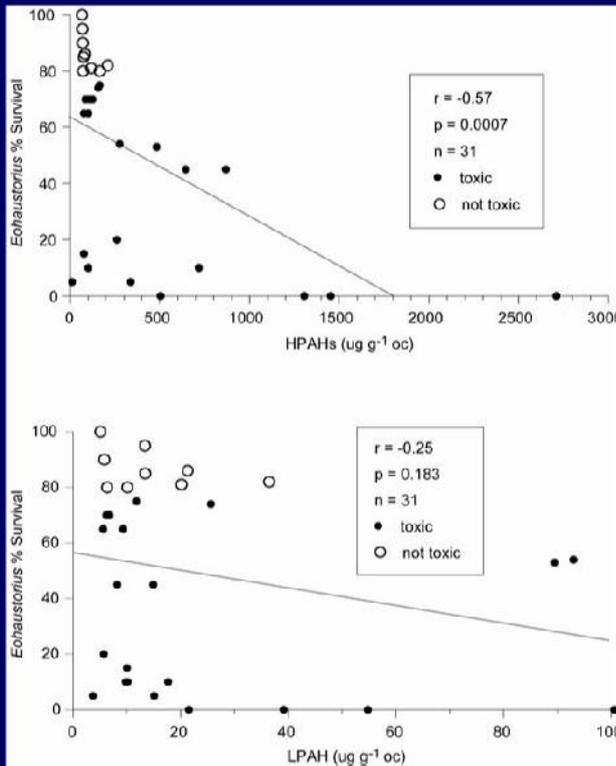
### Predicted Loss of PAH Over 1 Year (Greenfield and Davis, 2005)

#### Assumptions:

- no external PAH load
- initial mass = 120,000 kg

#### Time for loss of one-half of initial mass:

- 21 d Naph (2 rings)
- 63 d Phen (3 rings)
- 302 d Fluor (4 rings)
- 6 y Benzo[b]fluor (5 rings)



Plots of *Eohaustorius* % Survival vs. HPAH and LPAH in Alameda and Castro Cove sediments. PAH were statistically associated with amphipod toxicity (Thompson et al., 1999).

**Relationship of sediment PAH with biological abnormalities. Based on experiments with English sole.**

Effect <sup>a</sup>	Threshold <sup>b</sup> (ppb)	Threshold Confidence Limits (ppb)
<i>DNA Damage</i>	288	6-1318
<i>Liver Lesions</i>		
Neoplasms	2800	11-5500
Foci of cellular alteration	54 (ns)	na-870
Specific degeneration/necrosis	940	600-1400
Proliferative lesions	230	1.4-830
Any lesion	620	300-1000
<i>Reproductive Abnormalities</i>		
Inhibited gonadal growth	4000	nd
Inhibited spawning	630	nd
Infertile eggs	630	nd
Abnormal larvae	630 (ns)	nd

Source: Johnson et al. (2002)

**Sediment PAH levels (1993-2001) and potential effects**

Station	# Samples	% >1000 ppb <sup>a</sup> Threshold	% >4022 ppb <sup>b</sup> ERL
Delta Sacramento River	17	0	0
Delta San Joaquin River	16	0	0
NE San Pablo Bay	18	100	17
NE Napa River	18	22	0
NE Petaluma River	18	22	0
NE Honker Bay	14	7	0
NE Grizzly Bay	16	6	0
NE Pacheco Creek	16	0	0
NE Davis Point	18	0	0
NE Pinole Point	16	0	0
CB Point Isabel	16	100	0
CB Richardson Bay	16	100	0
CB Horseshoe Bay	16	81	0
CB Yerba Buena Is.	16	50	0
CB Red Rock	14	0	0
SB Redwood Creek	16	94	0
SB Dumbarton Bridge	16	94	0
SB San Bruno Shoal	14	93	0
SB South Bay	16	88	6
SB Alameda	14	86	7
SB Oyster Point	16	69	0
SB Coyote Creek	12	42	0
SB San Jose	10	30	0
SB Sunnyvale	7	14	0
EI Guadalupe River	8	38	0
EI Standish Dam	10	0	0

<sup>a</sup>Johnson et al. (2002) suggested level to protect estuarine fish against health effects: At >1000 ppb risk of liver disease, reproductive impairment & effects on growth is substantially increased

<sup>b</sup>Long et al. (1995) used to protect benthic invertebrates

### Sum PAH Thresholds

- *Rhepoxynius abronius* Sum PAH (Page et al., 2002)  
LC50 = 10,750 ng/g; Lower Threshold = 2,600 ng/g
- Sum PAH Consensus-based Guideline Value = 1,800 µg/g OC (Swartz et al., 1995)

### PAH Toxicity Models

- Target Lipid Model (Di Toro et al., 2000; Di Toro and McGrath, 2000)
- Sum PAH Model (Swartz et al., 1995)

## Questions Remaining To Be Addressed

- Should we be primarily concerned with PAH mixtures or individual PAH in sediments?
- Are PAH causing toxicity to critical estuary species?
- What are the best indicator species?
- What levels are safe for aquatic biota and humans?
- What can be done to control PAH input?
- Who will follow up on monitoring in the Bay? in Air?

## Acknowledgements

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San Francisco Estuary Institute, Oakland, CA

John Ross, SFEI



## **Lyndal Johnson**

Northwest Fisheries Science Center, National Oceanic and Atmospheric Administration, Seattle, WA

### **Polycyclic Aromatic Hydrocarbon Exposure Levels Associated with Injury in Marine Fish**

**Abstract:** Under the U.S. Endangered Species Act and the Essential Fish Habitat provisions of the Sustainable Fisheries Act, it is the responsibility of the National Marine Fisheries Service (NMFS) to safeguard the health of fish in estuarine and coastal waters. This includes assessment of the impacts of toxic chemicals such as polycyclic aromatic hydrocarbons (PAHs) on fish and their critical habitat. However, regulatory guidance for marine and estuarine fish is limited, as most sediment evaluation guidelines and sediment testing procedures are based on effects on benthic invertebrates. These guidelines may not adequately protect fish, because the metabolism and toxicology of PAHs differs in these two groups of organisms. While invertebrates generally bioaccumulate PAHs, fish metabolize and excrete them, producing toxic intermediates that can be mutagenic and carcinogenic. In Puget Sound, NMFS researchers have linked PAH exposure with DNA damage, cancer and related liver lesions, reproductive impairment, and reduced growth in bottomfish. We used these data in analyses designed to help NMFS resource managers determine when fish are exposed to potentially harmful PAH concentrations. Effects thresholds were estimated through segmented regression of site-specific sediment PAH concentrations and associated DNA damage and disease prevalences in a resident bottomfish, English sole. Both effects were minimal at sediment PAH concentrations below 1000 ppb. Above 1000 ppb, the risk of contaminant-related injury to English sole increased, with substantial proportions of animals showing effects at concentrations above ~5000 ppb. More limited data indicated a similar pattern for PAH-related impacts on sole growth and reproduction. In NOAA's National Benthic Surveillance Project, liver lesions like those in sole were found in starry flounder from San Francisco Bay, with highest lesions prevalences at sites with the greatest PAH contamination. Segmented regression yielded a PAH sediment threshold of 1000 ppb for specific degeneration/necrosis, the most common lesion in these fish. Similar analyses for winter flounder from the Northeast Coast of the United States yielded comparable liver lesions thresholds. Recent studies suggest that PAHs may also affect disease resistance, early development and cardiac function of fish at relatively low environmental concentrations, although specific thresholds have not yet been determined. The effects thresholds based on flatfish liver lesions are comparable to some existing guidelines, including the NOAA Effect Range Low (4022 ppb total PAHs) and the threshold effects level used by the Florida and Environment Canada (1684 ppb total PAHs). However, they are substantially lower than the typical sediment screening guidelines used for dredged material management, indicating a need to refine current sediment evaluation procedures to better assess PAH toxicity to fish. In applying the fish threshold data to the current sediment management framework, factors to be considered include differences in species sensitivity, the influence of PAH mixture composition and type on

toxicity, effects of co-occurring contaminants, the effects of chronic vs. short-term impacts, and the geographical extent of PAH contamination vs. its likely biological impact. Another potential approach would be the development of dietary PAH exposure thresholds for fish that could be incorporated into the existing bioaccumulation testing framework.

# Polycyclic Aromatic Hydrocarbon Exposure Levels Associated with Injury in Marine Fish

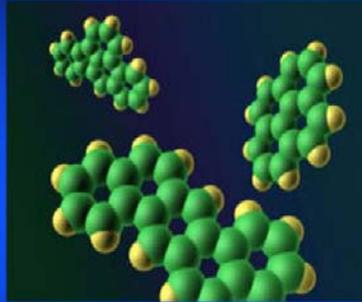
Lyndal L. Johnson

Environmental Conservation Division,  
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Seattle, Washington

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Tracy Collier

## Polycyclic aromatic hydrocarbons (PAHs)



- Fused aromatic rings
  - Low molecular weight (e.g. 1-2 aromatic rings)
    - Typically, do not bind to Ah receptor
  - High molecular weight (3 or more rings, usually 3-5)
    - Often are AhR agonists, potential carcinogens
- 
- Low water solubility, decreases with size
  - Alkyl substitution common; -N, -S, and -O heterocycles
  - Generally deposited in sediments in aquatic systems, slow rates of degradation

## Sources of PAHs



- Petrogenic (LMW) and pyrogenic (HMW) sources
- Natural sources (seeps, fires)
- Anthropogenic sources (spills, internal combustion engines, coal burning, wood preservatives)
- Variety of routes (point sources, non-point sources)
  - Urban runoff, atmospheric deposition, riverine

# PAHs in fish



- Benthic invertebrates typically accumulate PAHs in tissues
- Originally (late 60s and early 70s) fish were thought to have limited ability to metabolize PAHs
- Analyses showing little or no PAHs in fish tissues following oil exposure led some to conclude that PAHs were not bioavailable
- Extensive metabolism of PAHs, and effects in fish, shown in mid-1970s
- Toxicology of PAHs different in fish than in invertebrates because of metabolism
- Linkages of environmental PAH exposure to tumor formation in fish demonstrated in 1980s



# Liver disease

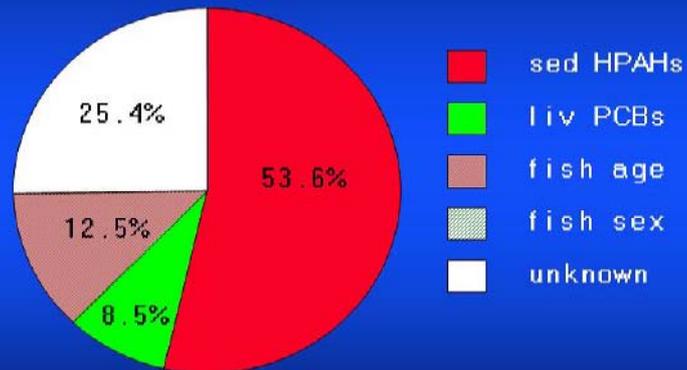
Liver tumors in benthic fishes from urban sites

Strong link to PAHs in sediment

Chemicals extracted from urban sediment caused liver lesions in healthy laboratory fish

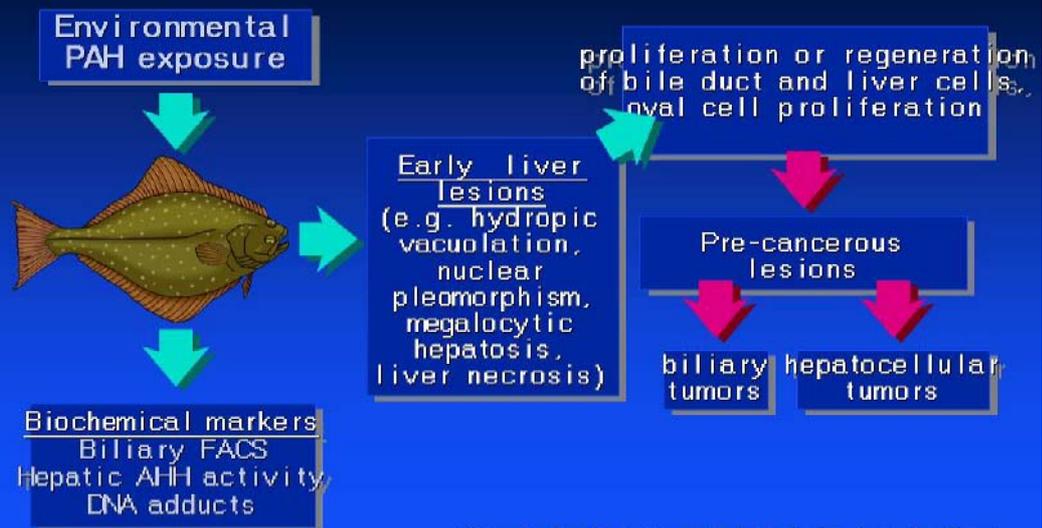


## Optimal Risk Model for Toxicopathic Liver Lesions in English Sole (PSAMP, '89-95)

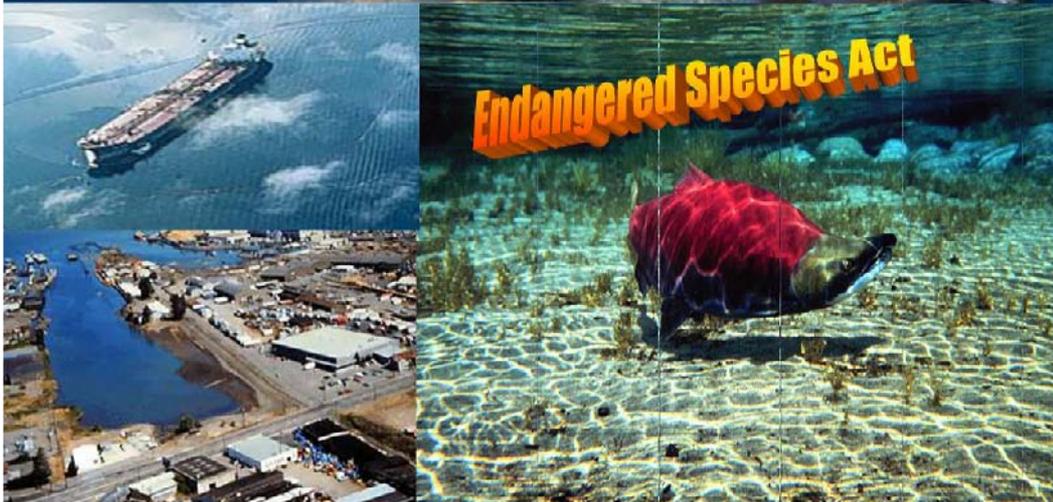


Total variance explained by model: 71.1%  
N = 49 sites

## Biologic responses in liver to PAH exposure\*



## Damage Assessment, Remediation, and Restoration Program



## NOAA Responsibilities for Marine Res

Under environmental laws in the United States, NOAA has stewardship of marine and anadromous resources, and is authorized to recover money or services to be used for restoration of resource injuries.

Under the ESA, NMFS is responsible for protecting threatened and endangered marine and anadromous fish against any activities that may kill or injure them or interfere with breeding, spawning, rearing, migrating, feeding, or sheltering.

## The Challenge: Protect Marine Resources from Impacts of Contaminated Sediments

- What are acceptable levels of contaminants in marine sediments for minimal biological and ecological damage?
- What levels are safe for protection of threatened or endangered species?
- What are realistic target levels for sediment contaminants in remediation projects?

## Goals

- Assess links between chemicals and biological effects
- Identify chemicals that are risk factors
- **Determine “thresholds” for effects**

## Dredged Material Evaluation Framework

Lower Columbia River Management Area  
November 1998



## Current Approaches to Sediment Quality Guidelines

- Based on effects mainly on effects on benthos
- Bulk sediment bioassays
- Benthic community structure
- Effects on fish not incorporated
- May not be appropriate for ESA species

## PAHs are a special concern

- Common in urban areas at relatively high concentrations
- In contrast to legacy pollutants (e.g. PCBs, DDTs) use is not banned, and sources are diffuse. Levels appear to be increasing
- Toxicology in fish may be different than in benthic invertebrates because of metabolism
- Associated with biological effects in fish such as liver cancer

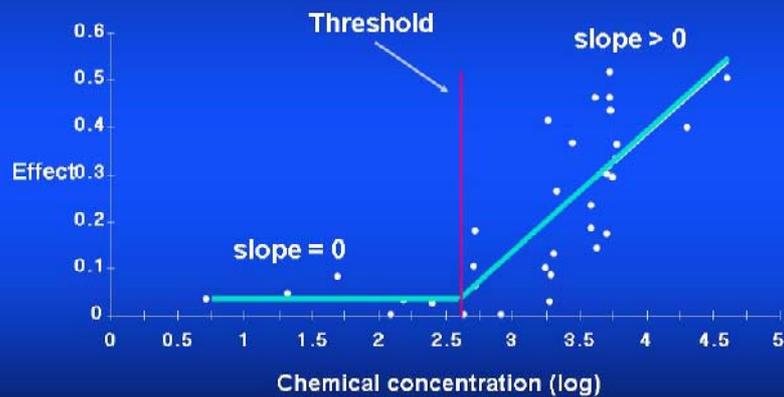
## Our Approach

- Estimate sediment toxicity thresholds from field data on chemical contaminant concentrations and associated biological effects
- Focus on sublethal effects
- Focus on effects that occur in indigenous organisms
- Use segmented regression to estimate threshold values

## Segmented Regression

- Has been used to model physiological responses
- An alternative dose-response or dose-extrapolation model
- Hockey Stick Regression is a special case
  - Two-segment, linear curve
  - Below the threshold, slope is zero
  - Segments are constrained to join at the threshold

## Sample Hockey Stick Regression Plot



## Biological Assumptions

- Adaptive physiological mechanisms (e.g., detoxification, DNA repair) control contaminant insult at low concentrations.
- Beyond some threshold the mechanism is overwhelmed, resulting in a linear population response

## Parameters Used in Threshold Model

### •Aromatic Hydrocarbons

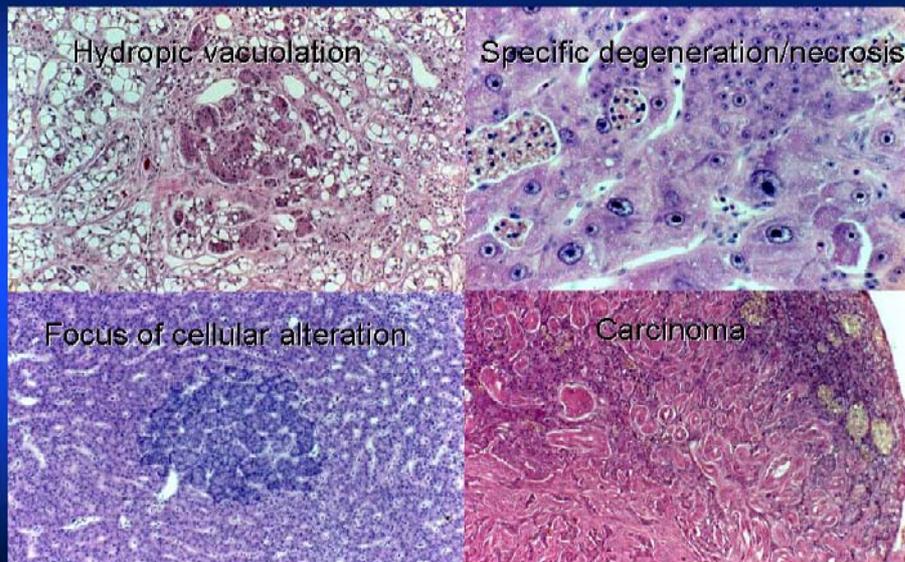
- Known carcinogens in mammals
- Cause liver lesions in bottomfish in laboratory exposures
- Linked to other biological effects (impaired growth and reproduction; altered immune function)

### •Pathological Conditions in Fish

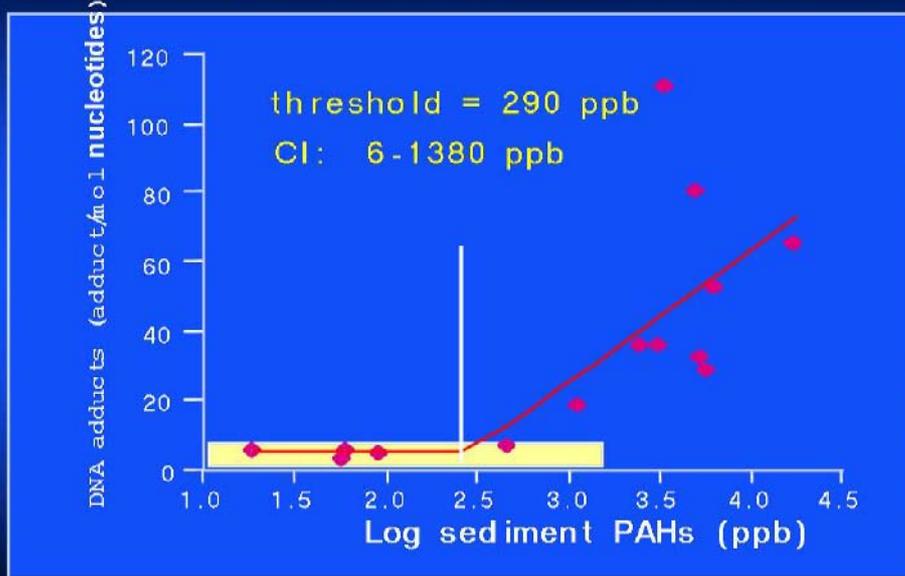
- DNA Damage
- Liver tumors and precancerous lesions



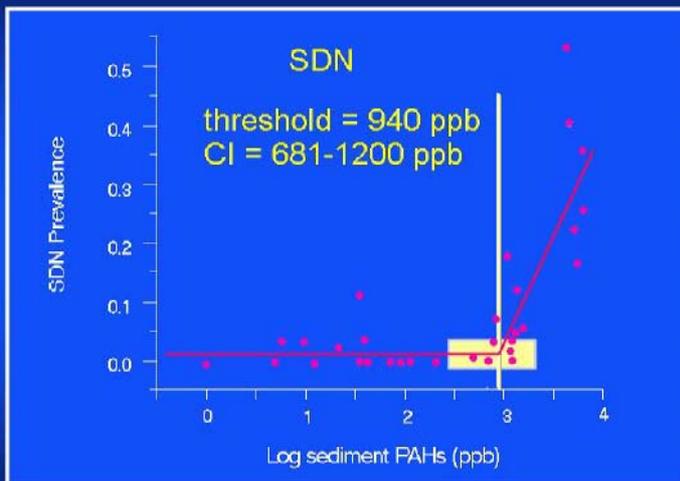
## Liver Lesions



### Sediment PAH threshold for DNA adducts in English sole



### Sediment PAH threshold for English sole Lesions: NBSP data



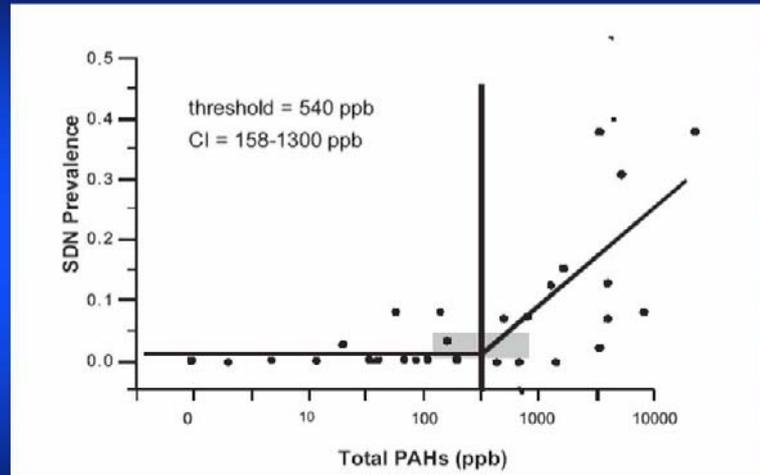
Neoplasms  
2800 ppb  
(11-5500)

FCA  
54 ppb  
(na-780)

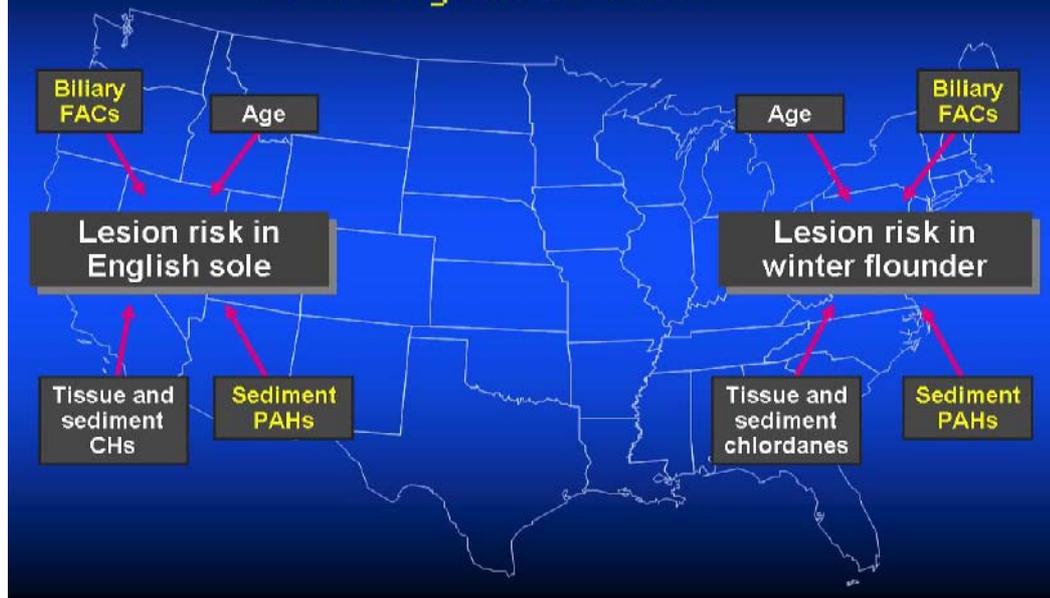
Proliferative  
Lesions  
230 ppb  
(1.4-830)

Any Lesions  
620 ppb  
(300-1000)

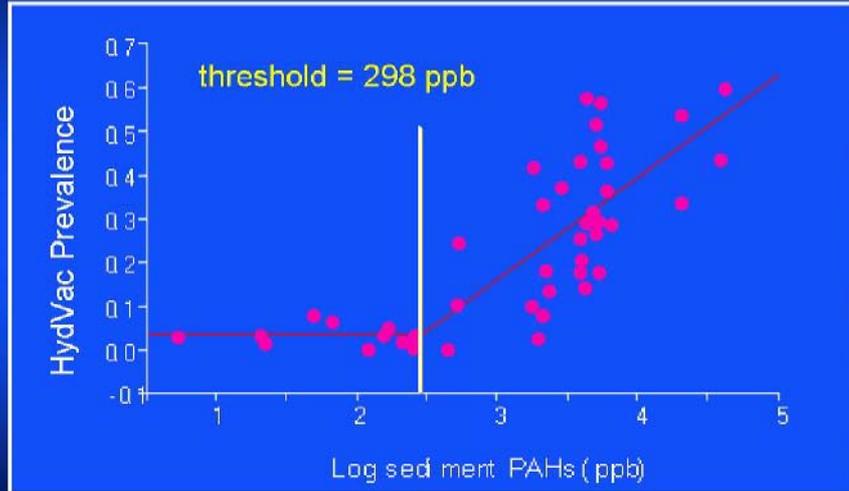
### Sediment PAH threshold for SDN in English sole: PSAMP data



### Identification of risk factors for lesions in indigenous fish



### Sediment PAH threshold for hydropic vacuolation in winter flounder



Plasma estradiol (ng/ml)



%vitellogenic females

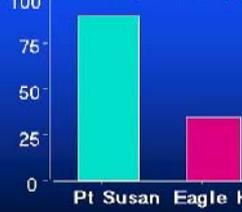


### Reproductive Problems

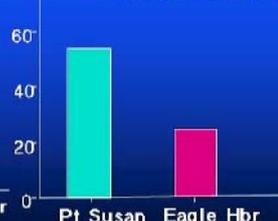
PAH exposure can cause resorption of eggs

Female sole in areas with high PAH levels have lower levels of reproductive hormones and are less likely to mature

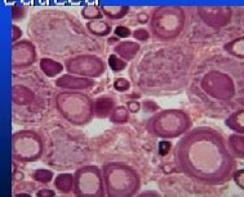
%spawning



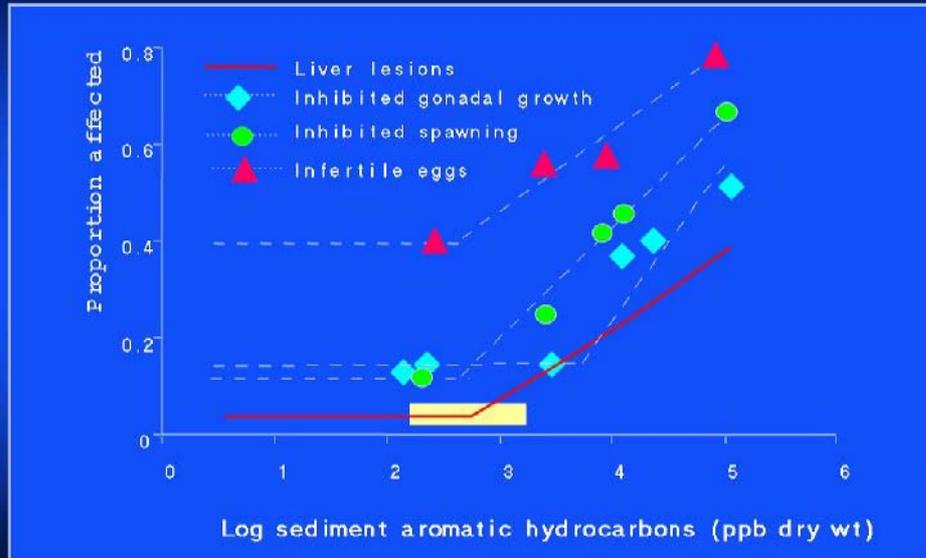
%fertilization



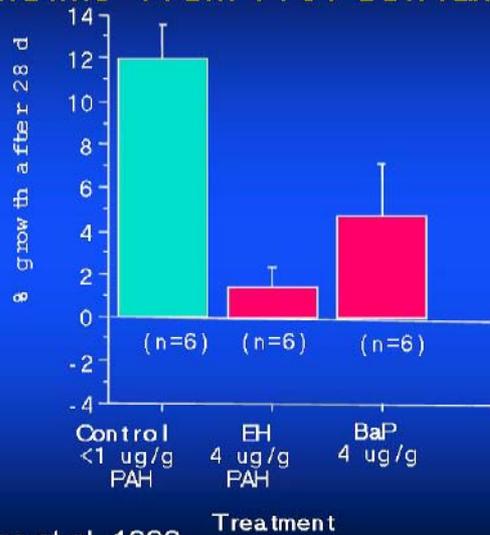
Spawning may be impaired, and egg and larval quality are reduced



## English sole reproductive success vs. sediment PAH concentrations



## Growth of juvenile English sole fed worms from PAH-contaminated sediment



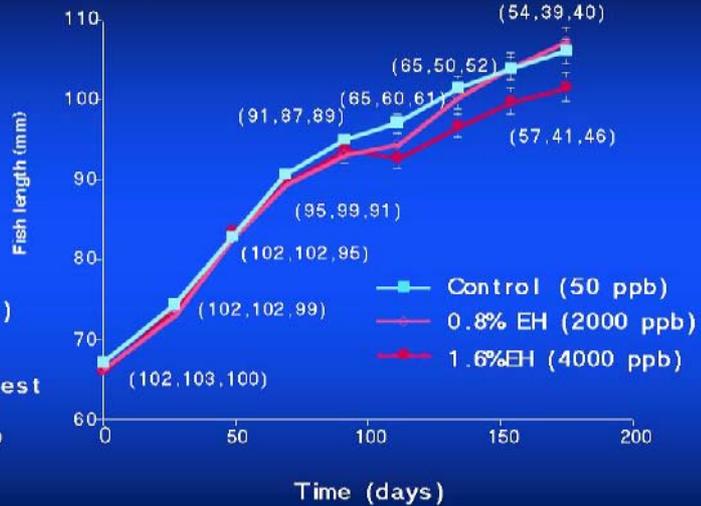
Rice et al. 1999

## Growth of juvenile English sole exposed to creosote contaminated sediment

Growth of juvenile English sole exposed to creosote contaminated sediment from Eagle Harbor, WA

No dietary exposure

Final size (in length) was reduced by approximately 15% in fish from the highest exposure group (Total PAHs ~4000 ppb dry wt)



Kubin 1997

## Sediment $\Sigma$ PAH concentration vs biological effects in English sole

### Biological Effect

PAH (ppb dry wt)	↑Liver Lesions (%)	↓Gonad Dev (%)	↓spawn (%)	infertile eggs (% eggs)	DNA damage (nmol adducts per mol bases)	↓Growth (%change in wt per day)
50	0	15	12	38	5	1.1-1.2
100	0	15	12	38	5	--
1000	9	15	17	42	25	--
2000	18	15	25	48	36	--
3000	24	15	30	51	43	--
5000	31	18	35	55	51	0.05-0.10
10000	40	27	43	61	63	--
100000	71	58	69	80	100	--



## Applications of the Threshold Approach

- Refinement of Sediment Quality Criteria
- Identification of high risk areas through sediment screening for PAHs
- Development of adaptive management strategies for ongoing restoration and remediation studies
- Could also be applied to generate thresholds from other exposure data (e.g., PAHs in diet, bile metabolites)

## Liver Lesions In San Francisco Bay



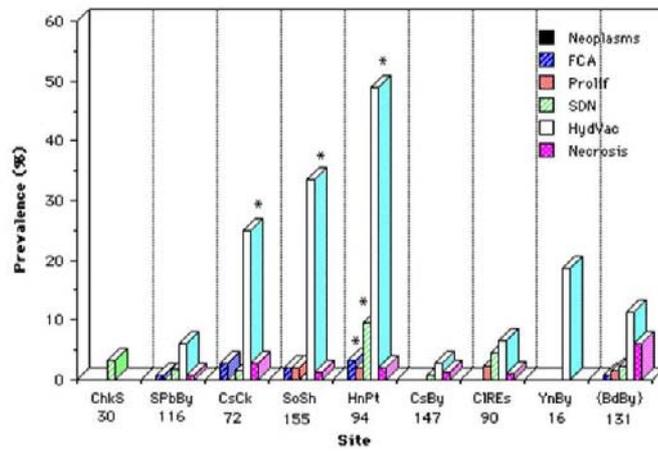
Starry flounder



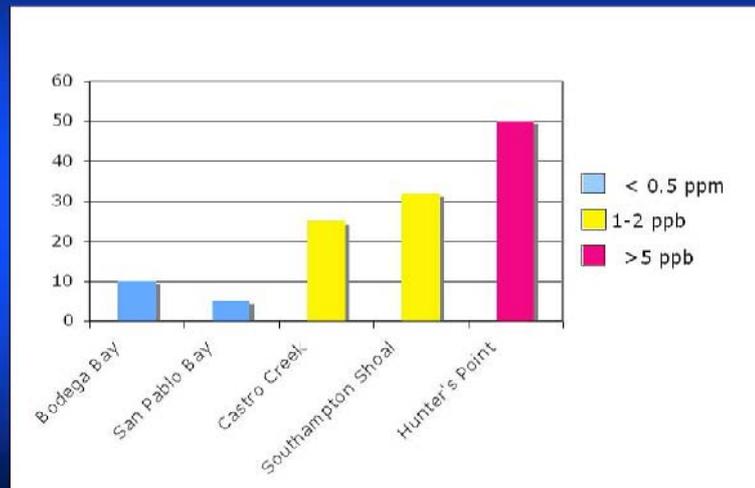
White croaker



## Liver Lesions in Starry Flounder



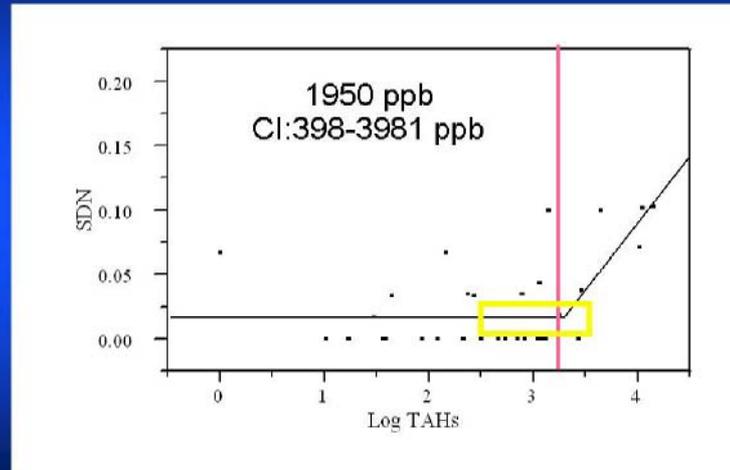
## SDN vs. PAHs in starry flounder



## PAHs and Lesion risk in Starry flounder

	FCA	SDN	HydVac	Necrosis
<b>Starry flounder</b>				
LAHs	ns	(+) 17% P<0.001	(+) 12% P<0.001	ns
HAHs	ns	(+) 19% P<0.001	(+) 13% P<0.001	ns
TAHs	ns	(+) 19% P<0.001	(+) 13% P<0.001	ns

### SDN vs. sediment PAHs in starry flounder

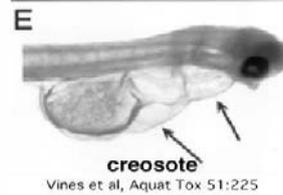
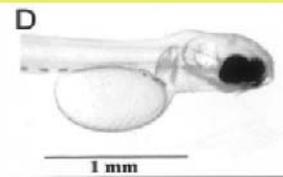


## Recent findings concerning effects of PAHs on fish



### Effects of PAHs on Pacific herring embryos from San Francisco Bay (creosote) and Prince William Sound (weathered crude oil):

- Spinal curvature (lordosis)
- Craniofacial abnormalities
- Small eyes
- Swelling of the neural tube
- Pericardial and yolk sac edema



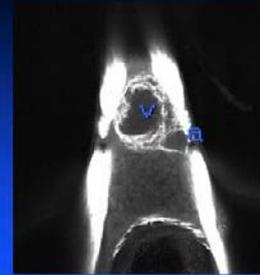
### Effects of PAHs: developmental defects in fish embryos and larvae

Impacts related to heart  
defects

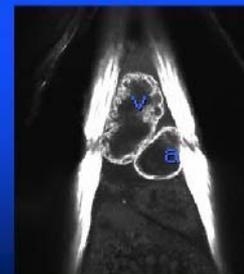
Low level exposure can  
cause heart problems but no  
obvious deformity

Studies following the Exxon Valdez spill  
found that pink salmon exposed as eggs  
to low levels of weathered crude oil (1-10  
ppb in water; ~1000 ppb in oiled gravel)  
appeared normal, but had lower adult  
return rates  
compared to unexposed fish.

Could be related to impaired cardiac function?



Normal



Phenanthrene

## Recent findings concerning effects of PAHs on fish

- Exposure of fertilized salmon eggs to low levels (1-10 ppb in water; ~1000 ppb in oiled gravel) of total PAHs from weathered oil is linked to reduced adult returns 2 years after exposure—possibly due to impaired cardiac function (work of Rice, Short, Heinz, Incardona)
- Juvenile salmon migrating through urban estuaries show reduced disease resistance and increased PAH exposure, and similar results are seen with PAH exposed animals in lab studies.
- Juvenile salmon migrating through urban estuaries show changes in growth and metabolism, and similar results are seen with PAH exposed animals in lab studies. Fish at higher doses experience delayed mortality several months after exposure ended.

## In Summary . . .

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- Chronic exposure to sediment PAHs is associated with various biological effects (liver lesions, impaired growth and reproduction) in English sole and other flatfish
- Thresholds for effects in 1,000-2,000 ppb range or below; significant effects in the 5,000-10,000 ppb range
- Similar relationships for liver lesions in San Francisco fish species
- More recent research shows PAHs affect growth and metabolism, development, immune function
- Thresholds uncertain but effects occur in field populations at sites where PAHs are in 5,000-10,000 ppb range

## Uncertainties for Management Application

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- Differences in species sensitivity
- Geographic extent of PAH hotspot vs. biological impact
- Influence of PAH mixture composition and type
- Effects of co-occurring contaminants
- Effects of chronic vs. short-term impact
- What level of impact constitutes biological damage?

## **Tom Gries**

Washington Department of Ecology, Environmental Assessment Program, Toxics Studies Unit, Seattle, WA

### **Risks from Exposure to Sediment PAHs**

**Abstract:** Sediment quality in Washington State is regulated using standards adopted by rule and by the Dredged Material Management Program guidelines. Both define two levels of protection: no and minor adverse effects. Numeric chemical and biological criteria and guidelines that are based on regional Apparent biological Effects Thresholds (AETs) are believed to protect benthic infaunal communities. However, these numeric criteria and guidelines are not intended to address the risk associated with exposures of fish, wildlife or humans to sediment contaminants. Standard risk assessment approaches are used for this purpose and will be illustrated using two cleanup site case studies. At the Eagle Harbor site, exposure of resident bottom fish to high concentrations of sediment PAHs was reduced through several remedial actions. The actions contributed to a large reduction in the observed incidence of liver lesions and other biomarkers, e.g., presence of hepatic DNA adducts. For the second site - the Lower Duwamish Waterway Superfund cleanup - a baseline risk assessment is currently being prepared that will address certain fish and human populations in addition to benthic communities. For some fish, tissue PAHs have been measured and for others they are being modeled using PAHs measured in gut contents and/or prey tissues. Tissue PAHs will be compared to threshold reference values that have yet to be defined. As with most risk assessments, levels of uncertainty about the concentrations of bioavailable PAHs in sediment that represent no or minor effects levels in populations of sensitive fish, wildlife and humans is expected to be high. It is evident from the Eagle Harbor and other case studies that merely having sediment quality standards and guidelines can facilitate actions that reduce exposure to PAHs in sediment and thereby improve benthic communities and fish health. However, a high level of uncertainty remains about the concentrations of sediment PAHs at a specific discharge, dredging or cleanup site that results in exposures still safe for fish populations. This is in part due to the fact that the mixtures of sediment PAH and their bioavailability differ between sites. Additional studies and policies that might reduce this uncertainty include a) new evaluation procedures that assess bioavailability of PAHs, b) new toxicity and/or bioaccumulation tests (e.g., exposing fish in a sensitive stage of development to sediment PAHs) and c) region or site-specific studies of the incidence of fish lesions and biomarkers.

## Risk from Exposure to Sediment PAHs

### Outline

- Washington's Sediment Management Standards or "SMS" rule (Chapter 173-204 WAC)
- Assessing Risk to Benthic Communities
- Assessing Risk to Human Health
- Assessing Risk to Fish and Wildlife
- Case Studies
- Conclusions, Uncertainties

## Risk from Exposure to Sediment PAHs

### Washington's SMS

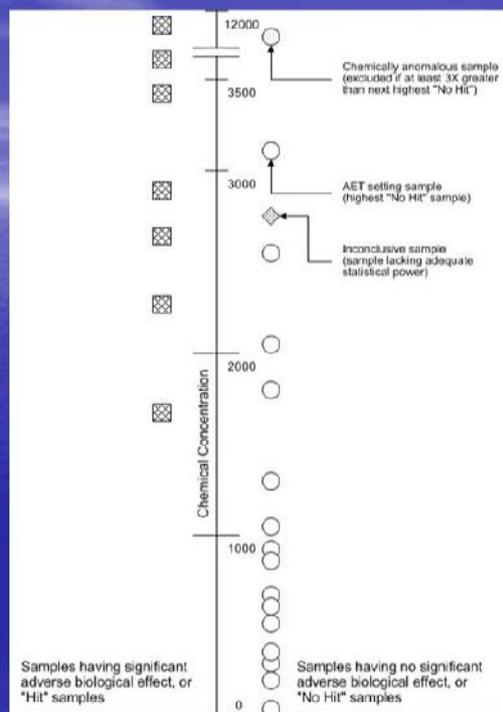
- Authorities - cleanup and water quality laws
- Applicable to cleanup, source control and dredging
- Narrative standards, e.g., protection of human health and wildlife, freshwater environments, antidegradation, etc.
- Numeric criteria for chemical constituents in *and biological properties of* marine sediments to protect benthic communities
- Two levels of criteria: goal (no effects) & upper regulatory limit (minor effects)

# Risk from Exposure to Sediment PAHs

## Risk to Benthic Communities

- AET-based numeric criteria for PAHs as well as acute and chronic toxicity in marine sediment
- Perceived as "High" SQGs:
  - LPAH AETs of 5.2, 13 and 24 mg/kg dry wt. versus TEL of 0.31 and ER-M of 3.2
  - HPAH AETs of 12, 17 and 69 mg/kg dry wt. versus TEL of 0.66 and ER-M of 9.2
- But more conservative when used in concert with other sediment standards/guidelines

Risk from Exposure to Sediment PAHs  
AET for PAH



## “Regulatory Beauty”



## Risk from Exposure to Sediment PAHs

### Risk to Benthic Communities

- Chemical criteria and guidelines used in dredging, cleanup and sediment source control programs
- May trigger toxicity and/or bioaccumulation tests
  - Modified protocol for UV-enhanced toxicity  
<http://www.ecy.wa.gov/biblio/0309043.html>
  - 45-day 'standard' Corps bioaccumulation tests
- How predictive of benthic impairment?

## Risk from Exposure to Sediment PAHs

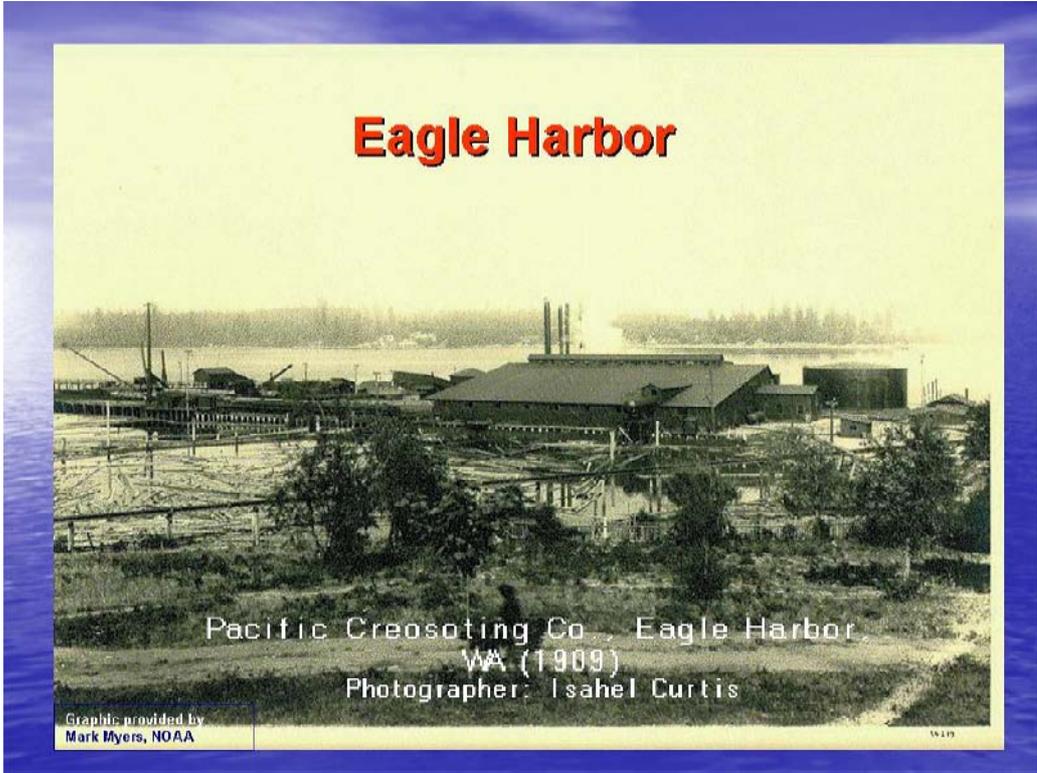
### Risk to Human Health

- Primarily sediment cleanup program
  - Identify target population and acceptable risk level
  - Identify direct and indirect exposure pathways
- Identify receptors of concern (fish, shellfish?)
  - Measure or model tissue PAHs
  - Link to sediment regulatory thresholds
- Less important for other programs?
  - Compliance with WQC during dredging, may be monitored
  - Compliance with WQC for discharge permits

## Risk from Exposure to Sediment PAHs

### Risk to Fish and Wildlife

- Eagle Harbor Superfund Site, Case Study
- Washington's numeric sediment quality standards *not* intended to protect fish and wildlife, except by virtue of protecting prey species
- Various Puget Sound salmonids listed under ESA
- NOAA/NMFS 'White Paper' and responses
- Lower Duwamish Superfund Site, Case Study



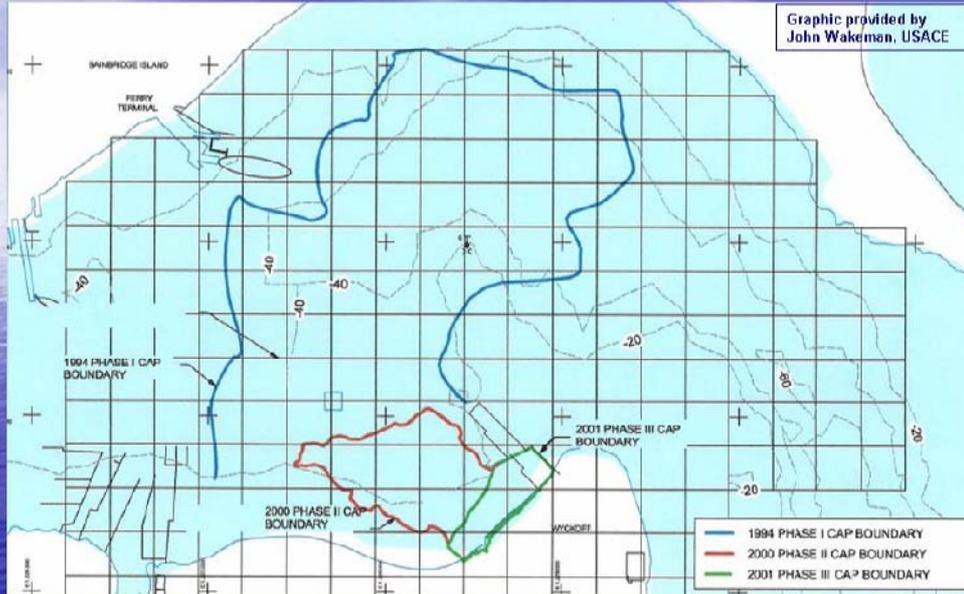


## Risk from Exposure to Sediment PAHs

### **Eagle Harbor Superfund Site**

- Pre-remedial site characteristics
  - Maximum sediment PAHs nearly 120,000 ug/kg
  - Acute toxicity in many locations
  - Incidence of hepatic lesions/tumors in flatfish 75%
- Remedial action objectives/cleanup goals
- Actions taken
  - 1994 capping
  - 2000 cap
  - 2001 cap
  - 10+ years monitoring

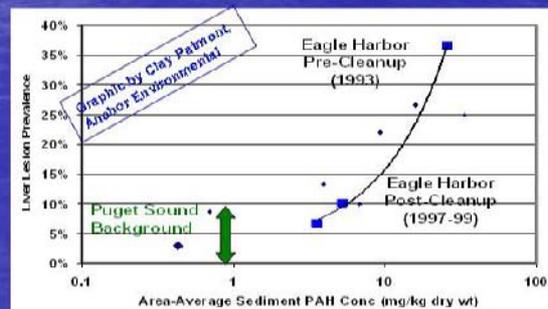
## Eagle Harbor Cap Boundary (2004)



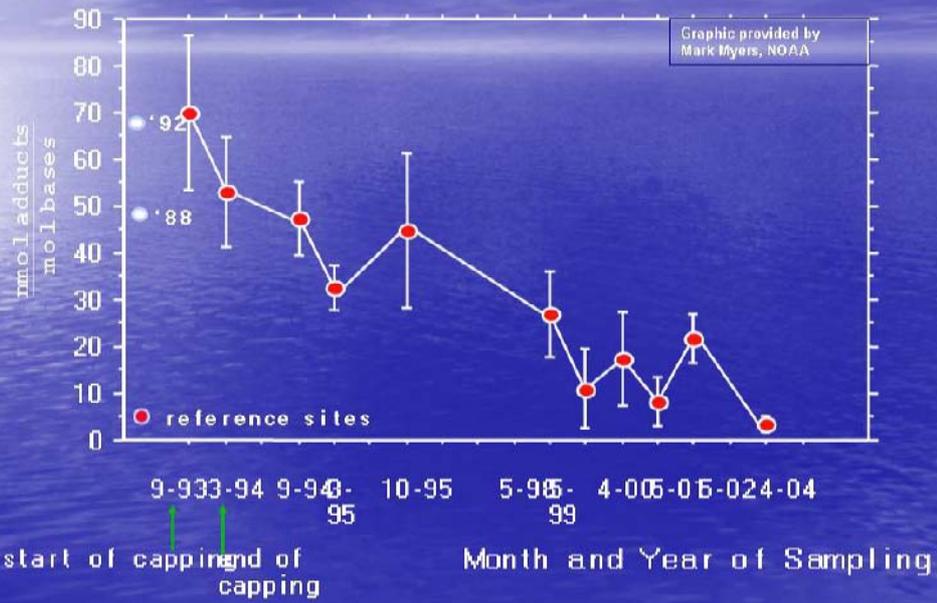
## Risk from Exposure to Sediment PAHs

### Eagle Harbor Superfund Site

- Post-remedial action monitoring results
  - Exposure to sediment PAH reduced
  - Reduced lesions (below), biliary FAC<sub>BAP</sub>, hepatic DNA adducts,



### Eagle Harbor Superfund Site: Hepatic DNA adducts in English Sole



### Lower Duwamish Waterway





## Lower Duwamish Waterway

Kellogg Island

## Risk from Exposure to Sediment PAHs

### Lower Duwamish Waterway Superfund Site

- Pre-remedial site characteristics
- Preliminary ecological risk assessment
  - Chemicals of potential concern
  - Likely pathways and receptors of concern
  - Tissue effects levels
  - Final determination for baseline RA

## Risk from Exposure to Sediment PAHs

### Lower Duwamish Waterway Superfund Site

- Phase 2 investigations
  - Risk to benthic communities assessed as exceedances of standards and/or laboratory toxicity
  - Risk to human health assessed via fish consumption, measured concentrations of PAH in fish tissue
  - Risk to fish themselves
    - Assessed using TRV approach (not biomarkers or “hockey-stick” regression)
    - Measured tissue PAH of benthic “market basket” and gut contents of juvenile Chinook salmon
    - Derivation of TRV in progress

## Risk from Exposure to Sediment PAHs

### “Conclusions”

- Sediment quality criteria/guidelines developed to protect benthic communities may or may not benefit fish populations
- Having sediment criteria/guidelines helps reduce exposures to sediment PAHs
- Reducing exposure to sediment PAHs can improve benthic communities and fish health
- Exposure reduction needed to protect fish populations is unknown
- Not all PAHs are created equal
- Need process for modifying program guidance

## Risk from Exposure to Sediment PAHs

### “Uncertainties”

- Evidence that reduction of sediment PAHs benefits fish populations?
- What PAH threshold level is needed to improve populations?
- Applicability of region/site-specific sediment PAHs thresholds elsewhere?
- How to weigh magnitude of management implications vs. uncertainty?

## Risk from Exposure to Sediment PAHs

### Acknowledgments *(by entity)*

- Clay Patmont, Daniel Hennesey (Anchor Env.)
- Lawrence McCrone, Dreas Neilson (Exponent)
- Pete Adolphson (Ecology)
- Justine Barton, Erika Hoffman et al (EPA)
- Jeff Stern (King County)
- Tracy Collier, Lyndal Johnson, Mark Myers et al (NOAA Fisheries)
- John Wakeman, Brenda Bachman (USACE)



***Fred Hetzel***

San Francisco Regional Water Quality Control Board, Oakland, CA

## **Ambient Concentrations of PAH in San Francisco Bay**

**Abstract:** In the mid 1990s, waterboard staff identified the need to determine ambient concentrations of chemicals in San Francisco Bay, in part to help evaluate disposal options for dredged sediments. Data collected for the Bay Protection and Toxic Cleanup Program (BPTCP) and the Regional Monitoring Program (RMP) were selected for the statistical analysis. The sediment samples used for this analysis were collected far from known or potential sources of contamination in order to characterize the least contaminated sediments. Statistical analyses were performed on the data to calculate the upper bound of the distributions for each analyte. Individual PAH were evaluated as well as total PAH. The upper 85th percentile of the distribution for total PAH is 3.4 mg/kg (Smith and Riege, 1999). This is not surprising as sediments in more remote areas often have total PAH concentrations above 1 mg/kg. However, several locations in the Bay were placed on the 2002 303(d) list as being impaired in parts due to elevated total PAH concentrations in surface sediments, and there are known areas around the Bay where elevated PAH concentrations are found in deeper sediments due to historical activities.

References: Smith R.W. and L. Riege. 1999. San Francisco Bay Sediment Criteria Project: Ambient Analysis Report. Prepared For the San Francisco Bay Regional Water Quality Control Board, Oakland, CA.



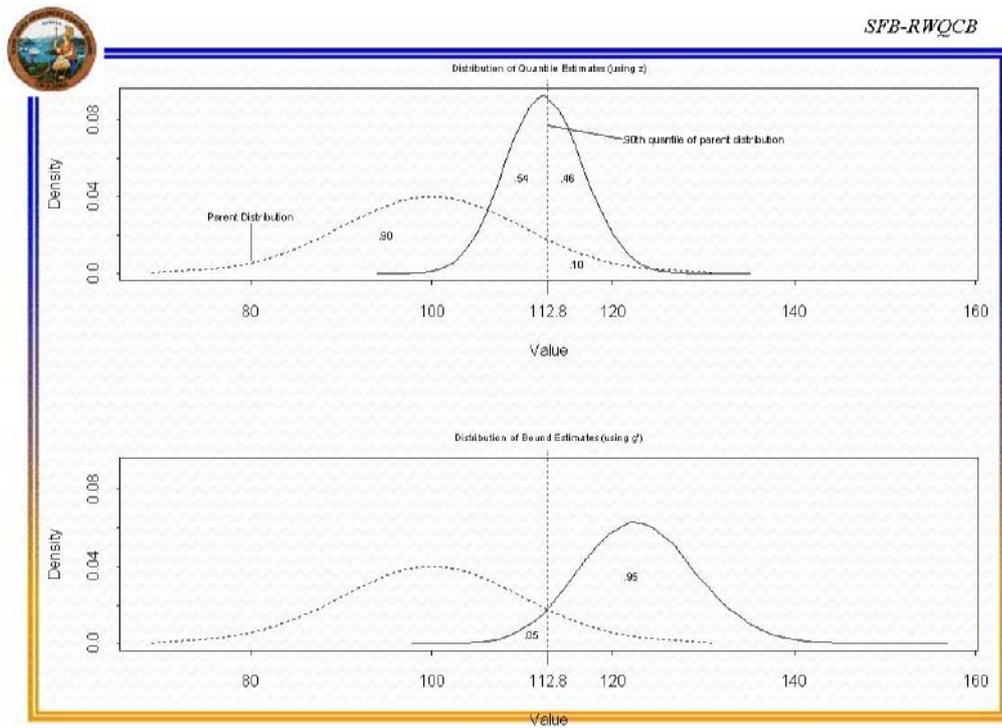
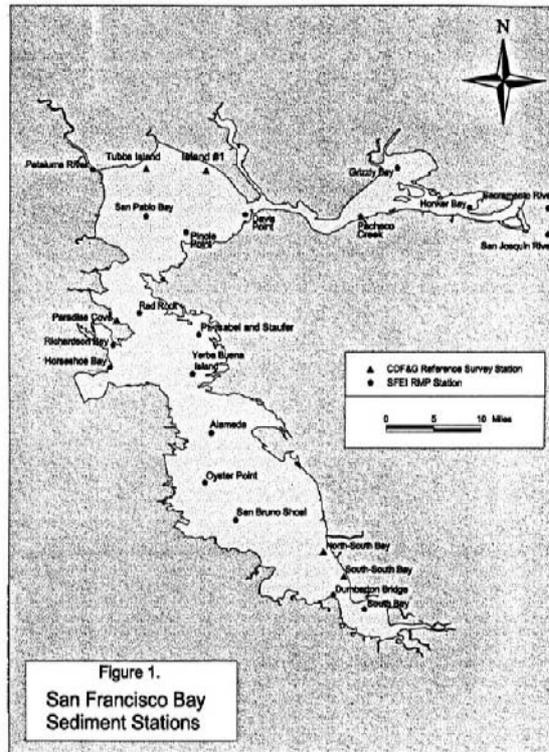
## **Ambient PAHs in San Francisco Bay**

**Fred Hetzel  
SFB-RWQCB**



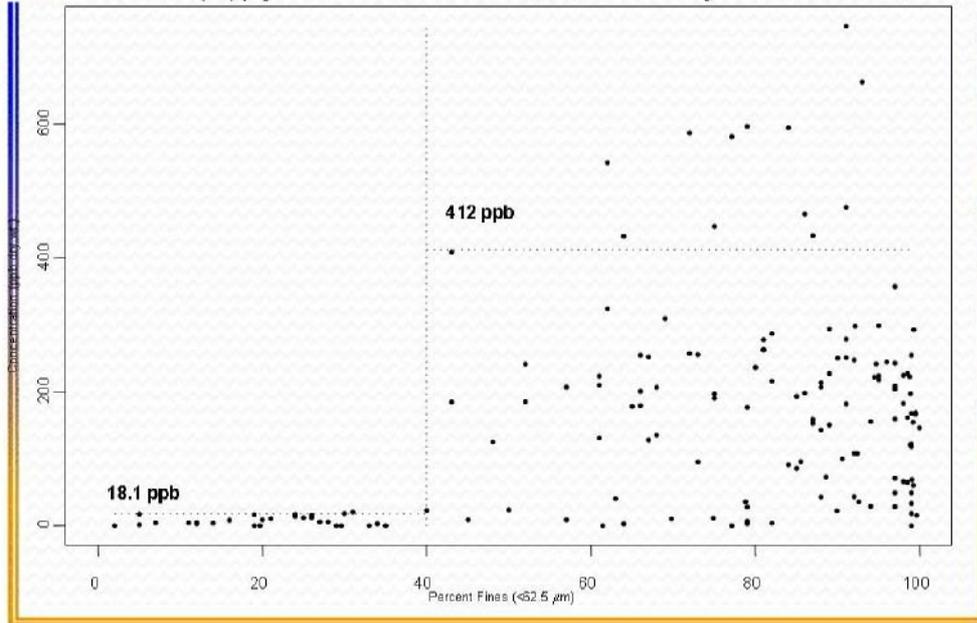
## **Reference**

Smith, R.W. and L. Riege. 1998. San Francisco Bay sediment criteria project: Ambient analysis report. Prepared for the California Regional Water Quality Control Board, San Francisco Region.

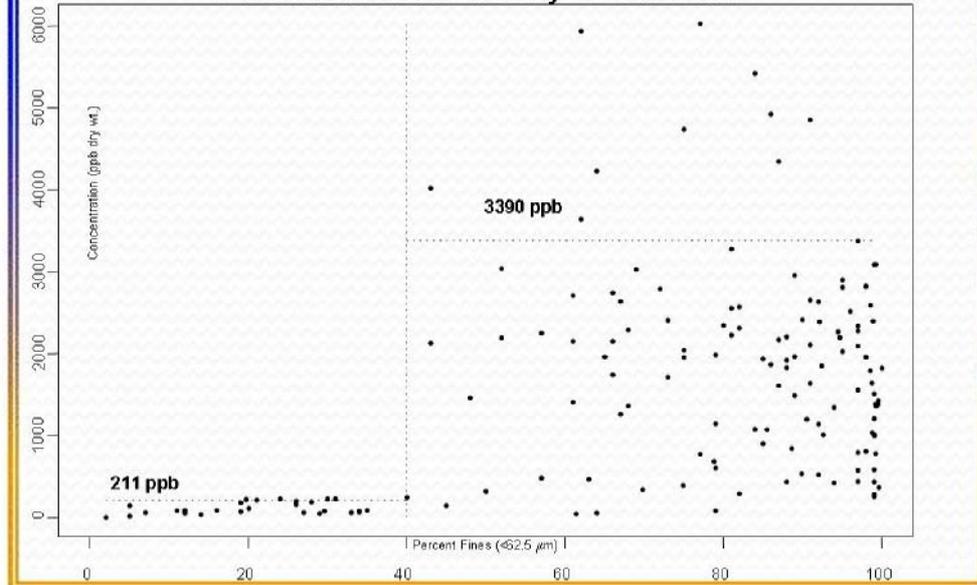




### Benzo(a)pyrene Concentrations in Bay Sediments



### Total Polynuclear Aromatic Hydrocarbons Concentrations in Bay Sediments





Analyte	S.F. Estuary Sediment Ambient Numbers	
	<40 % fines	<100 % fines
High molecular weight PAHs	256	3060
Low molecular weight PAHs	37.9	434
PAHs, total	211	3390

Analyte	S.F. Estuary Sediment Ambient Numbers	
	<40 % fines	<100 % fines
1-Methylnaphthalene	6.8	12.1
1-Methylphenanthrene	4.5	31.7
2,3,5-Trimethylnaphthalene	3.3	9.8
2,6-Dimethylnaphthalene	5	12.1
2-Methylnaphthalene	9.4	19.4
Acenaphthene	11.3	26.6
Acenaphthylene	2.2	31.7
Anthracene	9.3	88
Benz(a)anthracene	15.9	244
Benzo(a)pyrene	18.1	412
Benzo(b)fluoranthene	32.1	371
Benzo(e)pyrene	17.3	284
Benzo(g,h,i)perylene	22.9	310
Benzo(k)fluoranthene	29.2	258
Biphenyl	6.5	12.9
Chrysene	19.4	289
Dibenz(a,h)anthracene	3	32.7
Fluoranthene	78.7	514
Fluorene	4	25.3
Indeno(1,2,3-c,d)pyrene	19	382
Naphthalene	8.8	55.8
Perylene	24	145
Phenanthrene	17.8	237
<b>Total</b>	<b>64.6</b>	<b>665</b>

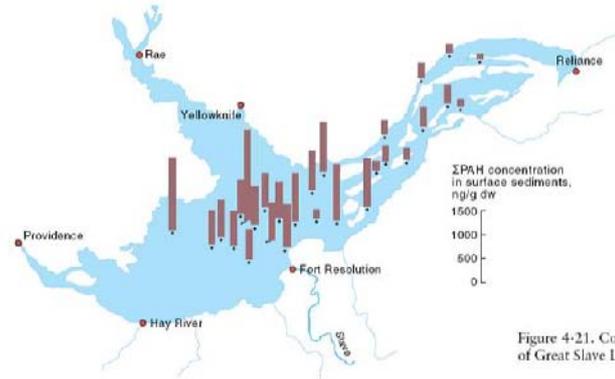


Figure 4-21. Concentrations of PAHs in surface sediments of Great Slave Lake (NWT, Canada) (Evans *et al.*, 1996).

J. V. Macías-Zamora

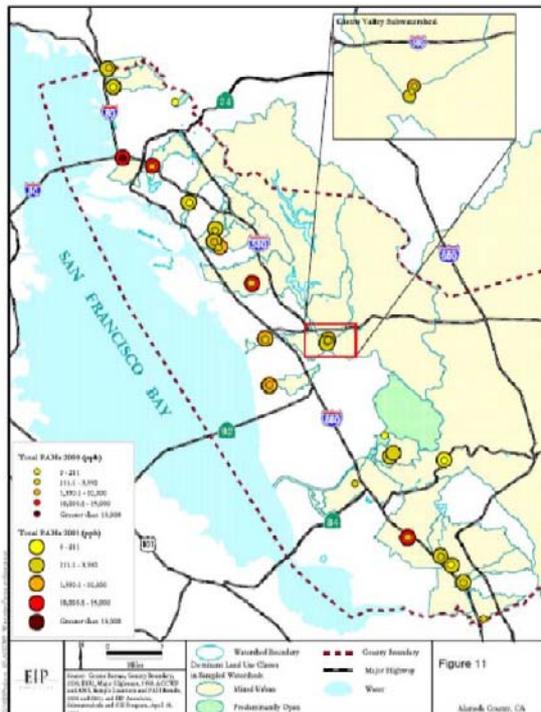


Fig. 1. Location of stations and main isobaths for the area of study.

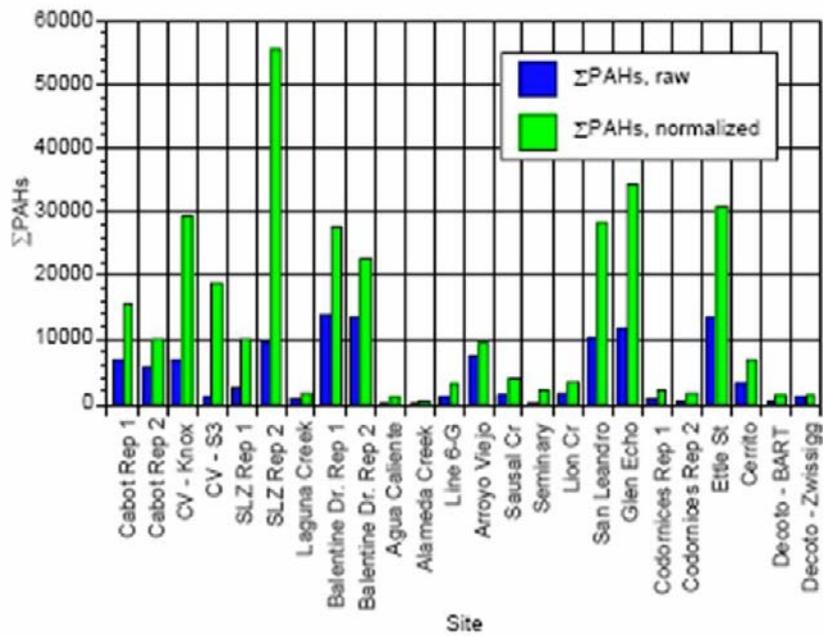
**Table 1. Concentration of linear hydrocarbons, unresolved carbon mixture and total polyaromatic hydrocarbons for recent marine sediments off the coast of Baja California**

Station	Total n-hydrocarbons (ppm)	Total UCM (ppm)	Total PAHs (ppm)	CPI	Depth (m)	Grain size ( $\phi$ )	% Org. carbon
A	14.14	26.23	0.14	1.06	29	3.621	0.19
B	1.80		0.55	1.09	40	1.526	0.05
C	34.37	220.5	1.93	0.92	55	1.830	0.05
D	30.48	114.3	0.60	0.93	89	4.385	0.04
F	12.24		0.82	0.98	70	3.513	0.32
G	24.35	74.5	1.03	1.00	48	3.498	0.26
H	0.77		0.28	1.14	31	2.117	0.08
I	70.64	49.02	0.54	1.03	22	2.038	0.17
J	14.07		0.22	0.88	31	2.525	0.29
K	2.55	8.90	0.12	0.98	30	1.303	0.27
L	2.40	4.15	1.51	1.11	68	5.048	0.68
M	6.57	23.61	0.16	1.01	58	0.337	0.68
N	70.64	151.10		1.12	35	2.084	0.20
O	2.12	12.12	0.55	1.14	33	2.264	0.16
P	7.03	78.88		0.96	38	3.021	0.30
Q	32.82	54.80	0.17	0.91	36	2.715	0.10
R	2.79	4.36	0.08	1.04	53	4.028	0.77
S	1.63	2.97	0.18	0.99	50	3.105	0.49
T	12.94			0.96	68	5.054	0.85
U	1.81	9.41	0.44	1.00	65	0.733	
V	1.55	5.28		0.95	48	2.225	0.25
W	1.58		0.16	1.12	42	5.215	1.91
X	6.87	29.46	0.20	1.30	62	4.289	1.94
Z	2.50	17.07	0.14	1.55	73	4.606	0.72

Empty spaces indicate that the measured level was below the limit of detection. Grain size in  $\text{mm} = 2^{-\phi}$ .



## Alameda County Creek Sediment Sampling Sites



## PAHs in Bay Water



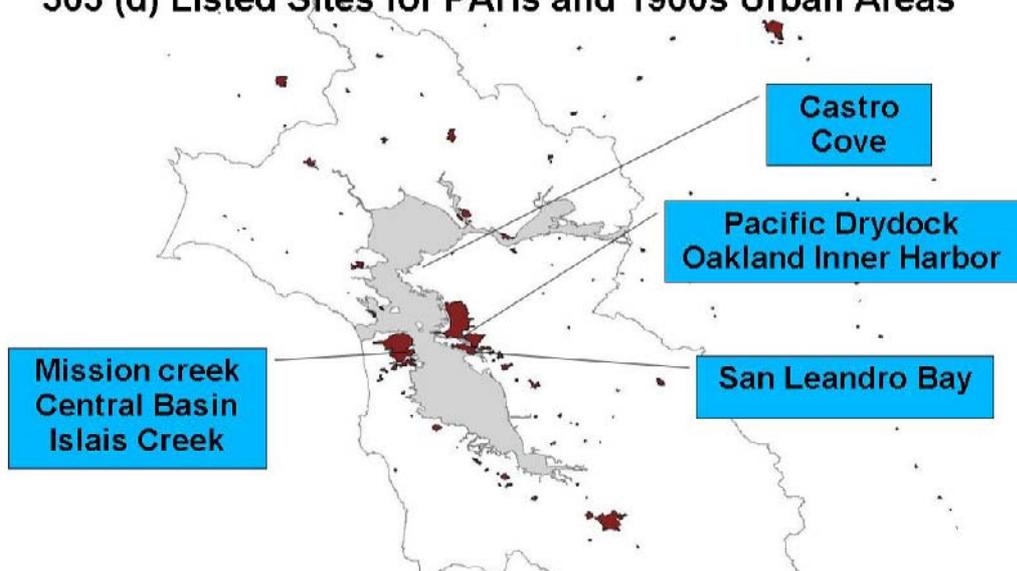
	Acenap hthene	Anthrace ne	Fluorene	Ben z(a) ant hra cen e	Chr yse ne	Pyrene	Be nz o(a) jpy ren e	Be nz o(b) jflu ora nth en e	Ben zo(k) flu oran thene	Flu ora nth ene
	ng/L	ng/L	ng/L	ng/L	ng/L	ng/L	ng/ L	ng/ L	ng/ L	ng/ L
	2700000	110000000	14000000	49	49	11000000	49	49	49	370
	0	0	0	2	0	0	3	4	1	0
N	144	187	177	240	294	265	116	296	281	281
Mean	2.6	2.0	2.5	5.2	4.3	9.0	6.8	8.2	3.8	7.7
Medi an	0.6	0.3	1.0	1.3	1.5	3.7	1.1	2.9	1.2	4.3
Stdev	3.2	2.2	2.0	10.1	5.4	12.7	10. 7	13. 9	6.9	8.4
95% CI	8.9	6.2	6.5	25.0	14.9	33.9	27. 7	35. 5	17.4	24.1



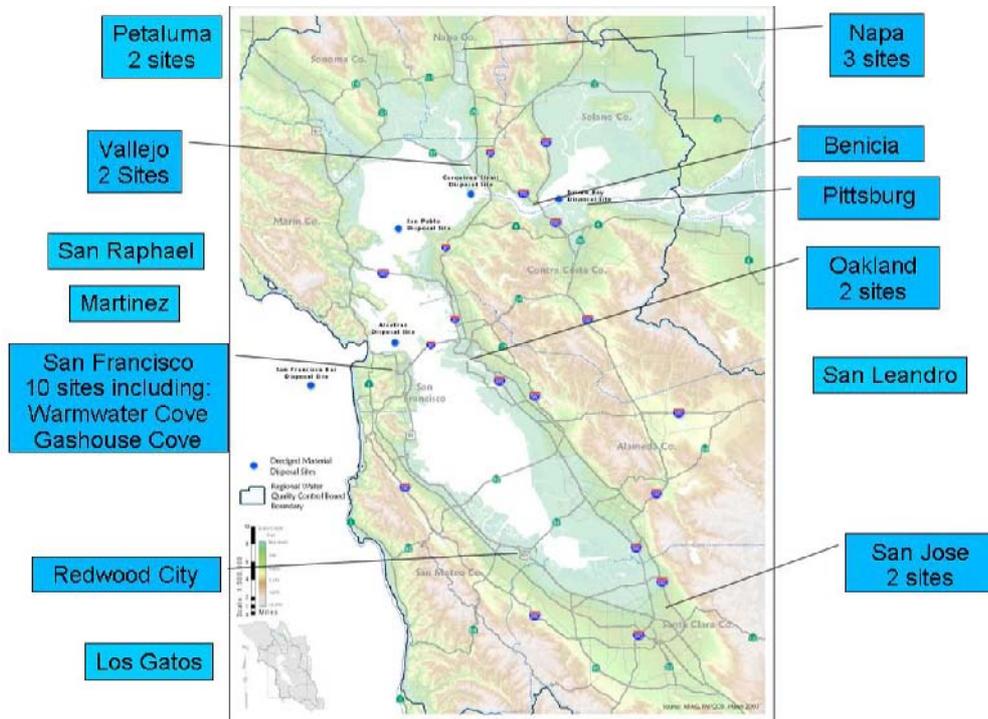
## Two narrative water quality objectives

- “All waters shall be maintained free of toxic substances in concentrations that are lethal to or that produce other detrimental responses in aquatic organisms.”
- and
- “Many pollutants can accumulate on particles, in sediment, or bioaccumulate in fish and other aquatic organisms. Controllable water quality factors shall not cause a detrimental increase in concentrations of toxic substances found in bottom sediments or aquatic life. Effects on aquatic organisms, wildlife, and human health will be considered.”

**303 (d) Listed Sites for PAHs and 1900s Urban Areas**



**Not Listed: HPS, ANAS, Mare Island NS, Moffett Field AS, Hamilton Army Airfield, Warmwater Cove, Gashouse Cove, and Waldo Point**



**Selected Locations of Coal Gasification Plants**