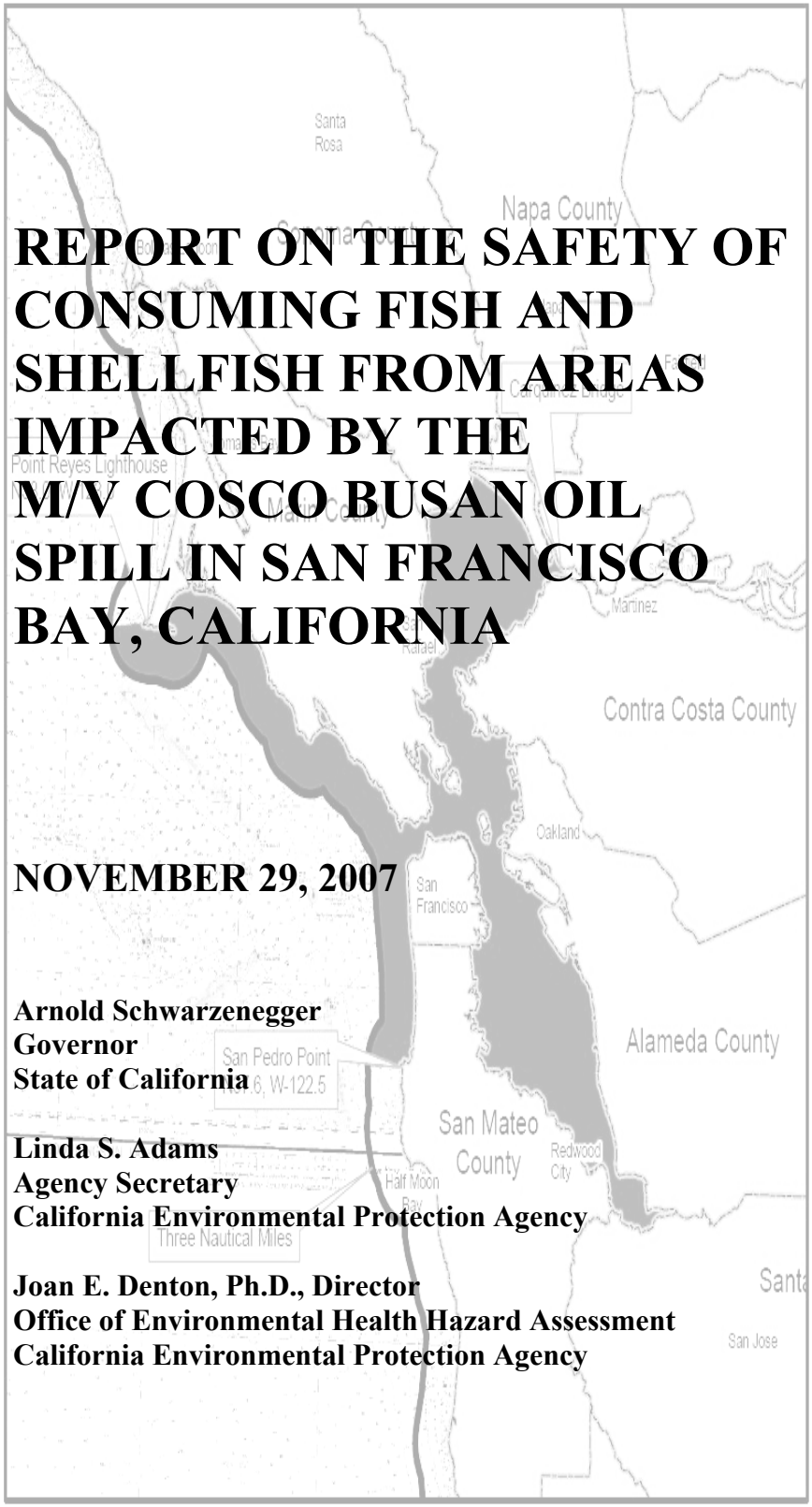

REPORT ON THE SAFETY OF CONSUMING FISH AND SHELLFISH FROM AREAS IMPACTED BY THE M/V COSCO BUSAN OIL SPILL IN SAN FRANCISCO BAY, CALIFORNIA

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Table of Contents

Executive Summary

On November 7, 2007, the container vessel M/V Cosco Busan struck the San Francisco-Oakland Bay Bridge, releasing approximately 58,000 gallons of IFO 380 bunker fuel into the San Francisco Bay. On November 9, Governor Schwarzenegger proclaimed a State of Emergency in the City and County of San Francisco and the counties of Alameda, Contra Costa, Marin, San Mateo, Solano and Sonoma. On November 13, Governor Schwarzenegger issued Executive Order S-14-07 directing the Office of Environmental Health Hazard Assessment (OEHHA), in consultation with the California Department of Public Health (CDPH), to "expeditiously review the available scientific information to determine whether a significant human health risk is posed by the human consumption of marine life caught in the area impacted by the oil spill." The Executive Order suspended all fishing for human consumption in the spill area until December 1, 2007. The California Department of Fish and Game (CDFG), in consultation with OEHHA and CDPH, can amend the ban as appropriate. Following the Executive Order, OEHHA issued an interim oil spill advisory for fish consumption for San Francisco Bay and coastal waters between Pt. Reyes lighthouse and San Pedro Point. This advisory recommended against consumption of fish or shellfish from the area affected by the oil spill.

In order for OEHHA to conclude that the marine life impacted by the oil spill were safe to eat, OEHHA had to determine three things: 1) the contaminants of concern in marine life following the oil spill, 2) the levels of these contaminants in fish tissue that pose no significant human health risk when consumed, and 3) the levels of these contaminants present in fish and shellfish in the impacted area. This report describes the results of that determination.

A sampling and analysis team was formed with staff from OEHHA, CDFG, including staff from CDFG's Office of Spill Prevention and Response (OSPR), CDPH, Department of Toxic Substances Control (DTSC), and the San Francisco Bay Regional Water Quality Control Board (SFBRWQCB). Staff from these departments met almost daily from November 13 through November 29 to discuss sampling options and priorities, analysis capabilities and timelines. This group developed a sampling plan to target the following species in the area affected by the Cosco Busan oil spill: Dungeness crab, Pacific herring, shiner surfperch, red rock crabs, and mussels. Several locations inside San Francisco Bay and on the coast were selected for collections. For comparison, samples from locations not affected by the spill were also obtained. The first round of sampling began on November 15 and was completed on November 20. The conclusions of the report are based on this sampling. Several Dungeness crab samples were sent for sensory evaluations.

Metals and polycyclic aromatic hydrocarbons (PAHs) can become concentrated in bunker fuel and may pose major human health concerns following an oil spill. Analysis of the fuel released into San Francisco Bay indicated very low levels of metal contaminants, therefore our evaluation focused on PAHs. OEHHA calculated 44 ppb (wet weight) as a level of benzo(a)pyrene equivalent (BaPE) PAHs in fish or shellfish tissue that, when

consumed, will not pose a significant human health risk. BaPE PAHs are considered the most valid measure of the cancer producing potency of the fuel. This finding assumes consumption of one 8-ounce meal (or two 4-ounce meals) per week for 30 years, which is a standard health-based approach. OEHHA considers a risk level of $1x10^{-4}$ (1 in 10,000) appropriate for use in fish consumption advisories, when considering the counterbalancing benefits of fish consumption. This risk level indicates that, for a population of 10,000 people consuming fish or shellfish containing 44 ppb (wet weight) BaPEs at 8-ounces per week for 30 years, only one additional case of cancer would be expected. This risk level is within the acceptable range of risks (1 in 10,000 to 1 in 1,000,000) used by the U.S. Environmental Protection Agency (U.S. EPA) in regulatory criteria for drinking water (Fed. Reg., 1998) and is provided as an example of a maximum acceptable risk level in U.S. EPA's Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories (U.S. EPA, 2000).

Dungeness crab, Pacific herring, and shiner surfperch were not found to contain any BaPE PAHs. Red rock crabs at Berkeley Pier were found to contain 0.4 ppb while those at Angel Island had undetectable levels. OEHHA and CDPH concluded that no significant human health risk is posed by consumption of Dungeness crab, Pacific herring, and shiner surfperch from the area impacted by the oil spill due to PAH contamination. In addition, a preliminary sensory evaluation of Dungeness crabs found no evidence that the taste and odor of the crabs had been significantly affected by the oil spill.

Mussels at Berkeley Marina and Rodeo Beach were both found to contain levels of 53 ppb BaPE, and thus they were both above the limit of health concern. For this reason, the OEHHA advisory recommending against consumption of fish or shellfish from the area affected by the oil spill will be revised to include only mussels at these two locations. This advisory will remain in place until testing indicates that consumption of mussels at these locations no longer poses a human health concern. Mussels at Angel Island and Baker Beach had levels of 12 and 2 ppb BaPE, respectively. Thus, these exposures were below our level of concern. CDPH concurs with these findings.

Although the tests found no increased risk from eating crabs or fish from the spill area because of oil contamination, it is possible that some fish or crabs may have come into contact with pockets of oil. Sport fishers should avoid eating any fish or shellfish that have an oily smell or taste. Commercial fishers and crabbers should take appropriate steps to ensure that their catches do not contact any remaining floating oil and are free of signs of contamination. Finally, it is important to note that there are other sport fish consumption advisories in the San Francisco Bay as a result of mercury and other contaminants (see http://www.oehha.ca.gov/fish/general/sfbaydelta.html).

Chapter 1. Introduction

On November 7, 2007, at 8:30 am, the container vessel M/V Cosco Busan accidentally struck into the San Francisco Bay Bridge, releasing approximately 58,000 gallons of IFO 380 bunker fuel into the San Francisco Bay. On November 9, Governor Schwarzenegger proclaimed a State of Emergency in the City and County of San Francisco and the counties of Alameda, Contra Costa, Marin, San Mateo, Solano and Sonoma; numerous beach closures ensued. On November 13, Governor Schwarzenegger issued Executive Order S-14-07 directing the Office of Environmental Health Hazard Assessment (OEHHA), in consultation with the California Department of Public Health (CDPH), to "expeditiously review the available scientific information to determine whether a significant human health risk is posed by the human consumption of marine life caught in the area impacted by the oil spill." The Executive Order suspended all fishing for human consumption including the start of crab season (Appendix 1). In response to this Executive Order, OEHHA issued an interim oil spill advisory for fish consumption for San Francisco Bay and coastal waters between Pt. Reyes lighthouse and San Pedro Point. This advisory recommended against consumption of fish or shellfish from the area affected by the oil spill. A map of the affected area is presented in Figure 1.

The purpose of this report is to describe the nature of the potential health risk from consuming fish and shellfish in the impacted area, the process of collecting and analyzing seafood in the affected area, the development of risk-based criteria for determining the safety of fish and shellfish consumption, the results of analytical seafood sampling, and conclusions regarding the safety of consuming fish and shellfish from the affected area.

Chapter 2. Contaminants of Concern in Marine Life Following an Oil Spill

Information on bunker fuels has recently been reviewed by OEHHA (2004). Several fuel types are used in marine vessels, including distillates, residual fuels, or a blend of both (U.S. EPA, 2003a). These blends are also referred to as intermediate fuels. "Bunker fuel" is a general term often used to refer to fuel burned in ships for propulsion, and largely consists of residual fuel. Residual fuel oils are the heavier oils that remain after the lighter fractions have been distilled away in the refining process. Residual fuel is inexpensive compared to other crude oil-derived products, and contains high levels of sulfur and nitrogen (U. S. EPA, 2003b). Polycyclic aromatic hydrocarbons (PAHs) and metals become concentrated in residual fuel. Residual fuel oils may be directly produced from the distillation process, as well as from a complex process of selection and blending of various petroleum fractions to meet definite specifications (Weisman, 1998). There are 15 residual fuel grades in national and international specifications; IFO 380 (also known as RMG35 and RMH35), which was released from the Cosco Busan, is a blend of distillate and about 98 percent residual fuel, with a viscosity of 380 centistokes at 50°C (Vis, 2003a; U.S. EPA, 1999).

The spill of bunker oil from the M/V Cosco Busan released PAH compounds into the environment. PAHs, which are usually found as complex mixtures of numerous

individual components, are a known class of potent cancer-causing chemicals (carcinogens). These chemicals are fused-ring structures of various sizes that tend to be fat or oil soluble and, as a result, may accumulate in the fatty tissue of seafood following an oil spill. PAHs are known to cause other acute or chronic health effects, but cancer is generally the health effect of concern when evaluating the risks of fish or shellfish consumption.

Several significant oil spills have occurred worldwide in the last decade or so, releasing different types and amounts of oil, including bunker fuel, into the environment. In 1996, for example, 180,000 gallons of IFO 380 bunker fuel and No. 2 fuel oil were spilled into the Fore River in Portland, Maine, after the tank vessel M/T Julie N hit a bridge. The Maine Department of Environmental Protection and the Maine Department of Maine Resources closed all fisheries, including fish, lobsters, mussels, scallops, and clams, in the Fore River and a significant portion of Casco Bay within hours of the spill. Over the next several weeks, seafood samples underwent sensory (organoleptic) testing for taint as well as chemical testing to detect a number of PAH compounds. Sensory and chemical criteria were not exceeded and, thus, the fisheries were reopened within seven weeks (Mauseth et al., 1997; Yender et al., 2002).

In 1997, after striking a dock, a fuel tank was punctured on the cargo carrier M/V Kure, resulting in a spill of approximately 4,500 gallons of IFO 180 fuel into Humboldt Bay, California. The California Department of Health Services closed commercial shellfish operations in Humboldt Bay as a precautionary measure. Although initial chemical tests revealed several samples that met or exceeded criteria set to protect the highest fish and shellfish consumers, subsequent testing showed that all samples passed sensory and chemical criteria. The fishery was reopened approximately seven weeks after the spill (Challenger and Mauseth, 1998).

Chapter 3. Identifying Levels of Contaminants Present in Fish and Shellfish

Emergency Response Team:

OEHHA coordinated a multi-agency effort to address the health impacts of the oil spill as mandated in Executive Order S-14-07 within 24 hours of the order being executed. OEHHA consulted with Office of Spill Prevention and Response (OSPR) staff deployed to Unified Command, as well as technical specialists throughout the California Department of Fish and Game (CDFG). Crucial to the efforts were laboratory staff and public health professionals from the Food and Drug Branch and the Environmental Management Office of the CDPH. Conference calls were convened daily. As the response moved into the second week, specialized sub-groups met separately on topics such as sensory (organoleptic) testing and sampling and analysis plans. Conference calls continued almost daily and provided a mechanism for information and data sharing while organizing the group's effort to meet the objectives spelled out in the Executive Order. As response activities expanded, the multi-agency coordination effort encompassed technical staff from the Department of Toxic Substances Control (DTSC), the San Francisco Bay Regional Water Quality Control Board (SFBRWQCB), the federal Food

and Drug Administration, the California Resources Agency, and the National Oceanic and Atmospheric Administration (NOAA). Representatives from the multi-agency group also reached out to answer requests from local and county jurisdictions on such matters as beach closures and public health statements.

Sampling Plan for Collection of Fish and Shellfish Species for Determination of Human Health Risk:

As noted above, fish and shellfish are exposed to a variety of PAHs following oil spills, and can become contaminated from this exposure. Fish are not only mobile, and may thus be able to avoid oil spills, but they are also able to metabolize and eliminate PAHs more effectively than shellfish. Contamination by PAHs is therefore more likely to be observed in shellfish, and particularly in bivalve mollusks such as mussels. These organisms can be exposed to PAHs in the sediments or on rocky shores where they reside.

A sampling plan was developed to target the following species in the area affected by the Busan oil spill. All samples were made by adding together tissues from individual fish or shellfish to make a "composite" sample. Shiner surfperch and Pacific herring were selected for finfish sampling. Data on background concentrations of PAHs in shiner surfperch were obtained through the Bay Protection Toxic Cleanup Program study of San Francisco Bay fish in 1994. Of the fish species tested in 1994, shiner surfperch had the greatest concentrations of PAHs. Therefore, this species was selected for evaluation and comparison to pre-oil spill conditions. Pacific herring were also targeted because they comprise a commercially harvested species. Dungeness crab were targeted in order to evaluate whether the commercial crabbing season could be opened on December 1. Red rock crabs and mussels were also selected for sampling and analysis because they are sport-harvested in the affected zone and, as indicated above, mussels are considered the most likely species to be affected.

Several locations inside San Francisco Bay and on the coast were selected for collections. A map showing the degree of oiling along the shores of San Francisco Bay was used to select the most heavily oiled areas for sampling. Consideration was also given to accessibility and feasibility of collecting the target species, and to locations where fishing is popular. Backup sampling locations were identified in the event that additional samples were needed. Rodeo Beach and Angel Island were identified as the most impacted areas. Oiling at Berkeley Pier and Marina was indicated to be light, however, it is a popular fishing area, staff reported seeing oil in this location, and it provided a sampling site on the east side of the bay. Baker Beach was selected to represent the coast south of the Golden Gate, and Muni and Hyde Street Piers were identified as additional backup locations.

Sampling began by CDFG on Thursday, November 15 with the collection of 11 small shiner surfperch. Samplers found additional shiner surfperch (34 collected) at Southhampton Shoals (located in the central bay between Angel Island and Berkeley). Bay mussels (150) were collected from Berkeley Marina and eight red rock crabs were

harvested from Berkeley Pier. Other samplers from CDFG collected 36 red rock crabs, 50 Bay mussels, and 17 black surfperch at Angel Island. Black surfperch are similar to shiner surfperch and were collected as a substitute because shiner surfperch were not available.

CDFG staff continued sampling on Friday, November 16. Thirteen Pacific herring were collected from Richardson Bay, 23 red rock crabs were collected from Muni Pier and 150 Bay mussels were obtained from Hyde Street Pier. In addition, samples of Dungeness crabs, and red and brown rock crabs were collected from Bodega Bay, which was selected as a "reference" or "control" site unaffected by the spill. Dungeness crabs were collected with the assistance of commercial crabbers along two transect lines, one north and one south of the Golden Gate. On the south transect off Pacifica, four crabs each were collected one mile and two miles off shore. On the north transect off Stinson, eight crabs, six crabs, and six crabs were collected one mile, two miles, and three miles offshore, respectively.

On Saturday, November 17, CDFG staff collected 14 red rock crabs at Berkeley Pier. An attempt was made to collect more shiner surfperch, but only one fish was caught. Eleven more Pacific herring were caught in Richardson Bay on Sunday, November 18. Collections of mussels from the coast at Rodeo Beach and Baker Beach, and from Tomales Bay, another reference site, were postponed until Tuesday, November 20, when low tides allowed safe collection.

Mussels were also collected by Natural Resources Damages Assessment (NRDA) teams from multiple locations in San Francisco Bay on November 19, 2007. Four samples were collected in the central bay. Three of these (CCY-N1-111107-4, CCY/Z-N1-111107-6, Yerba Buena –N1-111107-8) were from the eastern side in areas with documented oiling (see Figure 2). One central bay sample (MRT-N2-111207-3) was collected on the western side of the central bay in an area with no visible oiling. Another mussel sample (SMH-N1-111207-4) was collected in the south bay in an area with no visible oiling. NRDA submitted these samples for analysis of PAHs. Other mussels and oyster samples were collected, but the results are not available at this time.

A second round of Dungeness crab collections by CDFG samplers began on Friday, November 23. The two transects were expanded to include a location four miles offshore, but fewer crabs were obtained at some of the locations, especially on the north transect. Additionally, crabs were targeted in order to perform sensory (organoleptic) tests for "taint" (odor or taste of oil). A total of 33 crabs were collected on the Stinson (north) transect and 12 from the Pacifica (south) transect on Saturday November 24. Of these 45 crabs, 12 were sent for sensory testing. An additional 26 Dungeness crabs were collected from Bodega Bay (the reference site) at one, two, three, and four miles offshore; six of these crabs were sent for sensory testing.

Table 1 shows all fish and shellfish collections through Wednesday, November 27, and which of the samples were shipped to the laboratory for analysis. Remaining samples are

being held frozen pending evaluation of the results of those analyzed first. Additional sampling is also planned for Dungeness crab and mussels.

Historical Data Collected In and Around Areas Impacted by the M/V Cosco Busan Oil Spill:

Historical data collected from areas in or around those impacted by the Cosco Busan oil spill were compiled from several sources for use in comparing the analytical results obtained from recent, post-spill sampling and analysis to historical or "ambient" levels of PAHs.

The State Mussel Watch Program, directed by the State Water Resources Control Board, analyzed several species of bivalves including resident bivalves and mainly transplanted bivalves that were collected from areas away from point sources of contaminants. Transplanted bivalves were deployed, on average, for three months in San Francisco Bay and other locations throughout California. PAH data were available for resident mussels from Lake Merritt off the Oakland Harbor of San Francisco Bay between 1990 and 1999.

Fish samples were collected in 1994 as part of a pilot study of contaminant levels in San Francisco Bay fish under the Bay Protection Toxic Cleanup Program (SFBRWQCB, 1995). PAHs were analyzed in numerous species including white croaker, surfperch species, jacksmelt, California halibut, striped bass, white sturgeon, leopard shark, and brown smoothhound shark collected throughout the bay. Comparisons of levels of PAHs in all fish species tested showed the highest levels in shiner surfperch.

The City & County of San Francisco Department of Water, Southwest Ocean Outfall Regional Monitoring Program, conducts regular monitoring of fish and shellfish at their Oceanside outfall site on the San Francisco coast and at a reference station off the coast. Data on Dungeness crab were available from 1997 to 2005. Some changes in analytical methods were made during this time, and we thus chose to focus on the 2005 data, which were considered more reliable.

Analytical results from historical data collections are presented in the results and conclusions section.

Analysis Plan:

Samples of source oil were analyzed by the CDFG Water Pollution Control Laboratory using EPA Method 8270 for hydrocarbons. The source oil was also analyzed for metals by California Laboratory Services using EPA 6000/7000 Series methods. Preliminary analyses showed a number of PAHs, including several carcinogenic compounds (benz[a]anthracene, benzo[a]pyrene, and chrysene) and also non-carcinogenic compounds (anthracene, fluoranthene, fluorene, naphthalene, and pyrene) present in the source oil. Cadmium, chromium, copper, lead, zinc, mercury and selenium were not found at the limit of detection in the source oil analysis. Nickel was detected but it was

below a level of health concern. Based on these results it was decided that tissue samples should be analyzed for PAHs, but not metals.

TDI-Brooks Laboratory in College Station, Texas was selected to prepare and analyze the tissue samples for PAHs using EPA Method 8270. They had experience with fish and shellfish tissues, could produce quality analyses with low detection limits, and meet the rapid turn-around time for the large number of samples being collected. CDFG dissected and homogenized the Dungeness crab samples prior to sending them to TDI-Brooks. TDI-Brooks prepared all other samples using their standard preparation procedures supplemented with specific compositing instructions for sample collections, using CDFG Method # MPSL-105 Laboratory Preparation of Trace Metals and Synthetic Organic Samples of Tissues in Marine and Freshwater Bivalves and Fish. As part of their analysis of a series of PAHs, they were able to determine concentrations for seven carcinogenic PAHs (chrysene, benz[a]anthracene, benzo(b)fluoranthene, benzo(k)fluoranthene, dibenzo[a,h]anthracene, indeno[1,2,3,-cd]pyrene, and benzo[a]pyrene), including the three found in the source oil. Their method also determined concentrations for all of the non-carcinogenic compounds reported in the source oil.

As noted in the sampling plan, twelve Dungeness crabs were collected for sensory testing on November 24 from the same Stinson (north) and Pacifica (south) transects used to collect crabs for chemical analysis. Six Dungeness crabs were collected from Bodega Bay to serve as controls for this testing. Crabs were collected by a commercial crabber working with CDFG. These crabs were collected and packaged in accordance with National Marine Fisheries Service Seafood Inspection Program Guidance on Sensory Testing and Monitoring of Seafood for Presence of Petroleum Taint Following an Oil Spill (NOAA, 2001) provided by Dr. Carol Kelly, Chief, National Sensory Section, Technical Services, NOAA. These crabs were shipped to the U.S. Department of Commerce Seafood Inspection Program in Long Beach, California for sensory analysis by a three person panel of sensory experts.

Dr. Kelly oversaw sensory testing of these Dungeness crab samples according to NOAA protocols on November 28, and submitted preliminary test results on November 29. According to the preliminary results, there was no evidence that the taste and odor of the crab meat had been significantly affected by the oil spill.

Chapter 4. Contaminant Levels in Fish Tissue that Pose No Significant Risk

PAHs, of which benzo(a)pyrene (BaP) is the most commonly studied and measured, are formed by the burning of organic matter such as coal, gasoline, and vegetation. Humans are exposed to PAHs primarily through the diet. In addition to ambient levels in foods, cooking and curing processes can generate PAHs. Smoked or barbecued meat and fish usually contain higher levels of PAHs relative to other foods. Because of the widespread distribution of PAHs in the environment, most types of uncooked food contain measurable levels of PAHs, generally in the parts per billion (ppb), or microgram (µg) per kilogram (kg), range. A major source of PAH contamination of foodstuffs is by contact with either petroleum or coal tar products (Lijinsky, 1991).

The occurrence of BaP in seafood is primarily attributed to aquatic pollution unless the seafood was smoked or broiled/grilled well-done. Deposited PAHs can become concentrated in marine sediments, where bottom-feeding fish and filter-feeding invertebrates can be exposed. In uncontaminated waters, a low concentration of BaP detected in shellfish of no more than 3 ppb is considered a baseline level (Takatsuki et al., 1985). Levels in mollusks in polluted waters have been known to reach concentrations well into the parts per million (ppm) range. However, as noted above, fish have a greater ability to metabolize PAHs than do mollusks, so PAHs tend to be at lower concentrations in fish.

If available, the safety of commercial seafood consumption is determined by comparison of tissue contaminant concentrations to U.S. Food and Drug Administration (USFDA) action levels. Because action levels are not available for PAH compounds, risk-based criteria to determine the safety of fish and shellfish consumption impacted by the oil spill were developed by OEHHA. These risk-based criteria were determined separately for carcinogen and non-carcinogen PAH compounds likely to be found in bunker fuel.

As noted above, cancer is generally considered the health effect of concern for PAH contaminants found in seafood. Nonetheless, the non-carcinogenic risk of PAH compounds analyzed in seafood from the affected area was calculated to confirm this belief. For non-carcinogenic PAHs likely to be present in bunker oil, tissue concentrations were compared to the chronic oral reference dose (RfD) for each chemical (see Table 2), using the same assumptions as for carcinogens described below. A chronic RfD is an estimate of daily human exposure to a chemical that is likely to be without significant risk of adverse effects during a lifetime (including to sensitive population subgroups), expressed in units of mg/kg-day (IRIS, 1993). This estimate includes a factor to account for data uncertainty. The underlying assumption of a RfD is that, unlike most carcinogens, there is a threshold dose below which certain toxic effects will not occur. Using the highest PAH levels found in the most highly contaminated species (mussels), predicted exposures were vastly lower than the oral RfD ("safe" exposure level) for each chemical (data not shown). As a result, only the derivation of a risk-based criterion for carcinogenic risk is described below.

The carcinogenic activity of BaP and other PAH compounds have been extensively reviewed by OEHHA (2005), sections of which can be found in Appendix 2. BaP has been found to cause cancerous gastric papillomas and squamous cell carcinomas, pulmonary adenomas, and leukemia by the oral route in rodent studies (see OEHHA, 2005, for discussion and references.)

The following general equation was used to set the public health protective concentration (C, in μg/kg or ppb, wet weight) for carcinogenic PAH compounds potentially found in fish or shellfish:

$$C = \frac{RL \times BW \times AT \times CF}{CSF \times CR \times ED}$$

where,

RL = risk level
BW = body weight of consumer
AT = averaging time (presumed lifespan)
CF = conversion factor (1000 µg/mg)
CSF = cancer slope factor
CR = consumption rate (the daily amount of fish consumed)
ED = exposure duration

The following specific factors and assumptions were used in the above equation:

Risk Level (RL):

Risk-based criteria were designed to prevent consumers from being exposed to the carcinogenic components of spilled oil in doses that exceed a risk level (RL) of $1x10^{-4}$ (1 in 10,000). This RL is within the acceptable range of risks ($1x10^{-4}$ to $1x10^{-6}$) used by the U.S. Environmental Protection Agency (U.S. EPA) in regulatory criteria for drinking water (Fed. Reg., 1998) and is provided as an example of a maximum acceptable risk level in U.S. EPA's Guidance for Assessing Chemical Contaminant Data for Use in Fish Advisories (U.S. EPA, 2000). OEHHA considers a RL of $1x10^{-4}$ appropriate for use in fish consumption advisories, when considering the counterbalancing benefits of fish consumption. Exposure to a carcinogen at this RL would be expected to result in not more than one additional case of cancer for every 10,000 people.

Body Weight (BW):

The default value for adult body weight for these calculations was assumed to be 70 kg.

Averaging Time (AT):

The default value for averaging time for these calculations was assumed to be 70 years (the presumed lifespan).

Cancer Slope Factor (CSF, also known as a Cancer Potency Factor):

A Cancer Slope Factor (CSF) is an upper-bound estimate of the probability that an individual will develop cancer over a lifetime as a consequence of exposure to a given dose of a specific carcinogen and is expressed as $(mg/kg\text{-}day)^{-1}$ (U.S. EPA, 1989). For the purposes of this risk assessment, OEHHA will use the CSF for BaP of 11.5 $(mg/kg\text{-}day)^{-1}$ (OEHHA, 2005). For additional information on calculation of risk for other PAH compounds potentially found in fish, see the results and conclusions section below.

Consumption Rate:

The consumption rate was assumed to be 32.5 g/day (one 8-ounce meal per week, prior to cooking, or two 4-ounce meals per week, prior to cooking). The consumption rate is close to the mean fish consumption rate for "recent consumers" (those that had eaten bay fish in the prior four weeks) of 28.1 grams per day reported in the San Francisco Bay Seafood Consumption Study (SFEI, 2000), and is equivalent to the American Heart Association's recommendation for a minimum weekly fish consumption rate for healthy adults (AHA, 2006).

Exposure Duration (ED):

The Exposure Duration (ED) was assumed to be 30 years. Thirty years is considered a high-end estimate of the length of time that individuals reside at a single residence in the U.S. (U. S. EPA, 1997; OEHHA, 2000). OEHHA considers that the assumption of a 30-year ED to carcinogenic contaminants in fish is appropriate to reasonably balance the risks and benefits of fish consumption, particularly as other animal protein sources that may replace fish in the diet also contain carcinogenic contaminants.

Calculation of the Public Health Protective Concentration:

Applying the specific factors and assumptions to the above equation results in the following risk-based criteria to determine the safety of fish and shellfish safety following the M/V Cosco Busan oil spill:

$$C = \frac{(1\times10^{-4})(70\text{ kg})(70\text{ yr})(1000\mu\text{g/mg})}{[11.5\text{ (mg/kg-day)}^{-1}](0.0325\text{ kg/day})(30\text{ yr})} = 43.7\text{ ppb (wet weight)}$$

This criterion estimates that, for every population of 10,000 people consuming fish or shellfish containing 43.7 ppb (wet weight) benzo(a)pyrene equivalent (BaPE) at 8-ounces per week for 30 years, only one additional case of cancer would be expected.

Chapter 5. Results and Conclusions

In order to interpret the results for individual PAH compounds that do not have established CSFs, the carcinogenic activity relative to BaP is estimated as the potency equivalency factor, or PEF (OEHHA, 2005). PEFs for PAH compounds likely to be found in bunker oil are listed in Table 3. Tissue concentrations of PAHs other than BaP are multiplied by their respective PEF and then added to the tissue concentration of BaP to determine the BaPE concentration. BaPE concentration is considered the most valid measure of the cancer producing potency of the fuel. BaPE concentration for each species is then compared to the risk-based criterion of 43.7 ppb (wet weight). All BaPE concentrations are reported in wet weight.

Analysis of levels of PAHs by TDI-Brooks Laboratory in composite samples of fish and shellfish collected after the Cosco Busan spill for assessment of human health risks

yielded the following results. Results (in BaPE) are shown in Table 4 and post-spill sampling locations are shown in Figure 3. For Dungeness crab, samples taken from the South Coast transect at one, two, and three miles offshore were all below detection limits. North Coast samples from two miles and three miles offshore were also below detection limits. The reference samples of Dungeness crab from Bodega Bay were below detection limits. An historical sample from the Oceanside Outfall on the San Francisco coast tested in 2006 by the City & County of San Francisco Department of Water contained 0.65 ppb BaPE.

Red rock crabs from Angel Island and from the Bodega Bay reference site were below detection limits. Red rock crabs from Berkeley Pier had 0.42 ppb BaPE. Brown rock crabs were also collected from Bodega Bay and were below detection limits.

Shiner surfperch from Berkeley Pier and Southhampton Shoals were analyzed together and the results were below detection limits. Black surfperch collected at Angel Island were below detection limits. Twelve composite samples of shiner surfperch from San Francisco Bay tested in 1994 under the Bay Protection Toxic Cleanup Program were also below detection limits for BaPE.

All Pacific herring caught in Richardson Bay were below detection limits.

Mussels from Berkeley Marina and Angel Island had 53.19 and 11.85 ppb BaPE, respectively. Mussels from Rodeo Beach had 53.05 ppb BaPE and 1.67 ppb BaPE from Baker Beach. Mussel samples were compared to historic samples from Lake Merritt in Oakland analyzed through the State Mussel Watch Program. The mean value for samples collected between 1990 and 1999 was 23.1 ppb BaPE. Reference samples from Bodega Bay were below detection limits and 0.19 ppb BaPE from Tomales Bay.

Additional samples were collected by NRDA and analyzed by Alpha Labs in Woods Hole, Massachusetts. Mussels were collected from four sites within San Francisco Bay (Figure 2). Results can be found in Table 5. Three samples from various locations in the central bay had 13.4 ppb, 20.34 ppb, and 2.59 ppb BaPE. A sample from Yerba Buena Island had 20.19 ppb BaPE, and a sample from the south bay had 4.59 ppb BaPE. Results for the other NRDA sampling sites in San Francisco Bay were not available.

Based on the findings from sampling and analysis of fish and shellfish from the area impacted by the spill, fish and crabs are well below the risk-based criterion (Figure 4). Therefore, consumption of fish or shellfish from the impacted area would not pose a human health risk from exposure to PAHs. Analysis of mussels showed that two of the nine locations sampled exceeded the risk-based criterion (Figure 5). Consumption of mussels from these two locations, Berkeley Marina and Rodeo Beach, is not recommended. Continued monitoring of mussels is necessary to track concentrations of PAHs at these locations. The revised advisory for mussels is presented in Figure 6.

Based on this risk assessment, OEHHA recommends that CDFG lift the ban on fishing that occurred as a result of the oil spill, with the proviso that OEHHA's no-consumption

advisory for mussels remains in effect at the Berkeley Marina and Rodeo Beach. However, it is important to note that there are other sport fish consumption advisories in the San Francisco Bay because of mercury and other contaminants (see http://www.oehha.ca.gov/fish/general/sfbaydelta.html). Although the tests found no increased risk from eating crabs or fish from the spill area because of oil contamination, it is possible that some fish or crabs may have come into contact with pockets of oil. Sport fishers should avoid eating any fish or shellfish that have an oily smell or taste. Commercial fishers and crabbers should take appropriate steps to ensure that their catches do not contact any remaining floating oil and are free of signs of contamination.

Chapter 6. Other Health Concerns

OEHHA was asked to provide advice on potential public health risks from skin contact with oil on the beaches or floating in the water. Local jurisdictions (local health departments, counties, cities and park districts) wanted to consider this information when deciding to reopen beaches, including allowing swimming or surfing, and for public health advisories. We developed additional advice regarding direct contact with oil or tar balls (http://www.oehha.ca.gov/public_info/pdf/FishImpactSFBayOil112107.pdf).

Volatile components of bunker fuel tend to dissipate fairly quickly after a spill. The primary hazard from contact with the remaining high viscosity, less volatile components, is skin irritation. In theory, high viscosity petroleum distillates encountered when swimming in an oil slick could, if aspirated, produce a lipoid pneumonia, but this exposure scenario is unlikely. We determined that, for this oil spill, an additional health advisory should focus on simple prudent measures to avoid prolonged skin contact with oil residues and tar balls. Language for the advisory was adapted from a fact sheet developed by the Alaska Department of the Environment after the Exxon Valdez oil spill (http://www.epi.hss.state.ak.us/bulletins/docs/b1989_06.htm). This information was shared with the San Mateo County environmental health officer who made the initial inquiry.

Local environmental health officers wanted to develop a unified message to the public on advice as people returned to the beaches after the oil spill, and San Mateo developed a draft health advisory incorporating OEHHA's recommendations. OEHHA staff also participated in a phone conference organized by the local environmental health officers on November 20 to clarify any remaining issues related to health advisories after the oil spill. OEHHA subsequently posted a health advisory on seafood consumption and avoiding skin contact with oil deposits on November 21. OEHHA will also be participating in a meeting with the Unified Command addressing criteria for beach reopening on November 30.

The health advice regarding potential acute hazards after the oil spill are summarized below:

- If you encounter oil or tar balls either in the water or on the beaches, avoid contact. Direct contact with the oil can cause skin irritation.

- If you do get oil or tar balls on your skin, wash it off with soap and water. Wash your hands before eating to avoid ingestion of oil.
- If you get oil on clothing, wash it in the usual way. Harsh detergents, solvents or other chemicals can increase skin irritation and should not be necessary to remove oil from skin or clothing.
- Don't burn debris, driftwood or other materials contaminated with oil.

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Table 1. Fish and Shellfish Sample Collected through November 27, 2007

Site/Locations	Species	Number Collected	Number of Composites Sent to Lab
Berkeley Pier	Red Rock Crabs	22	2
Berkeley Marina	Bay Mussels	150	3
Berkeley Pier & Southhampton Shoals	Shiner Surfperch	45	2
Angel Island	Black Surfperch	17	3
Angel Island	Red Rock Crabs	36	3
Angel Island	Bay Mussels	150	3
Richardsons Bay	Pacific Herring	24	2
Richardsons Bay	Shiner Surfperch	1	0
Muni Pier	Red Rock Crabs	23	
Hyde Street Pier	Bay Mussels	150	
Bodega Bay (reference site)	Red/Brown Rock Crabs	30	2 brown, 1 red
Spill Zone Transect – South of Gate 1 mile out	Dungeness Crabs	4	1
Spill Zone Transect – South of Gate 2 miles out	Dungeness Crabs	4	1
Spill Zone Transect – North of Gate 1 mile out	Dungeness Crabs	8	1
Spill Zone Transect – North of Gate 2 miles out	Dungeness Crabs	6	1
Spill Zone Transect - North of Gate 3 miles out	Dungeness Crabs	6	1
Bodega Bay - Clean Zone	Dungeness Crabs	12	3
Tomales Bay at Shell Beach	Bay Mussels	150	3
Rodeo Beach – North End	Bay Mussels	150	3
Baker Beach – West End	Bay Mussels	150	3
Total Number of Samples		1138	38

Table 2. Reference Doses (RfDs) for Selected PAH Compounds

Compound	RfD*	Critical Effect
Anthracene	$3x10^{-1}$	NOAEL
Fluoranthene	$4x10^{-2}$	Nephrotoxicity, increased liver weight, hematological effects, clinical effects
Fluorene	$4x10^{-2}$	Decreased RBCs, PCV, Hb
Naphthalene	$2x10^{-2}$	Decreased mean terminal BW for males
Pyrene	$3x10^{-2}$	Renal tubular pathology, decreased kidney weight

*RfDs (mg/kg-day) were obtained from U.S. EPA's Integrated Risk Information Service (IRIS) in November, 2007

Legend for Table 2:

NOAEL: No Observable Adverse Effect Level, RBCs: red blood cells, PCV: packed cell volume, Hb: hemoglobin, BW: body weight

Table 3. Potency Equivalency Factors and Cancer Slope Factors for Selected PAH Compounds

Chemical	Potency Equivalency Factor (PEF)	Cancer Slope Factor (CSF) $(mg/kg\text{-}day)^{-1}$
benzo[a]pyrene	1	11.5
benz[a]anthracene	0.1	
benzo[b]fluoranthene	0.1	
benzo[k]fluoranthene	0.1	
Indeno[1,2,3-cd]pyrene	0.1	
chrysene	0.01	
dibenzo[a,h]anthracene	0.36	4.1

Table 4. Benzo(a)pyrene Equivalent (BaPE) Concentrations in Fish and Shellfish Collected from Areas Impacted by the Oil Spill

Species	Location	BaP Equivalent (µg/kg, wet weight)
Dungeness crab	South Coast – 1mile	< D.L.
	South Coast – 2mile	< D.L.
	South Coast – 3mile	< D.L.
	North Coast – 2mile	< D.L.
	North Coast – 3mile	< D.L.
	Bodega Bay	*< D.L.*
	Oceanside Outfall[1]	*0.65*
Red rock crab	Berkeley Pier	0.42
	Angel Island	< D.L.
	Bodega Bay	*< D.L.*
Brown rock crab	*Bodega Bay*	*< D.L.*
Shiner surfperch	Berkeley Pier/ Southhampton Shoal	< D.L.
	Historic Bay[2]	*< D.L.*
Black surfperch	Angel Island	< D.L.
Pacific herring	Richardson's Bay	< D.L.
Mussels	Berkeley Marina	53.19*
	Angel Island	11.85
	Rodeo Beach	53.06
	Baker Beach	1.67
	Lake Merritt historic[3]	*23.1*
	Bodega Bay	*< D.L.*
	Tomales Bay	*0.19*

1 Data from 2005; mean of outfall and reference; Southwest Ocean Outfall Regional Monitoring Program, City & County of San Francisco, Department of Water.

2 Historic samples (12 composites) from Contaminant Levels in Fish Tissue from San Francisco Bay, 1994 San Francisco Regional Water Quality Control Board, State Water Resources Control Board, and California Department of Fish and Game.

3 Mean value from Mussel Watch Program (1990-1999), State Water Resources Control Board, and California Department of Fish and Game.

< D.L. = less than detection limit for carcinogenic PAHs.

Reference site outside of spill area or historic data showing pre-spill levels are italicized.

*One composite was dropped because of quality assurance considerations. The remaining composites were acceptable.

Table 5. Benzo(a)pyrene Equivalent (BaPE) Concentrations in Fish and Shellfish Collected from Areas Impacted by the Oil Spill and Reference Sites

Species	Location	BaP Equivalent (µg/kg, wet weight)
Mussels	NRDA SF Bay mid CCY-N1-111107-4	13.4
Mussels	NRDA SF Bay mid CCY/Z-N1-111107-6	20.34
Mussels	NRDA SF Bay mid YerbaBuena-N1-111107-3	20.19
Mussels	NRDA SF Bay mid MRT-N2-111207-3	2.59
Mussels	NRDA SF Bay South SMH-N1-111207-4	4.59

Figure 1. Fishing Ban Zone Following the M/V Cosco Busan Oil Spill

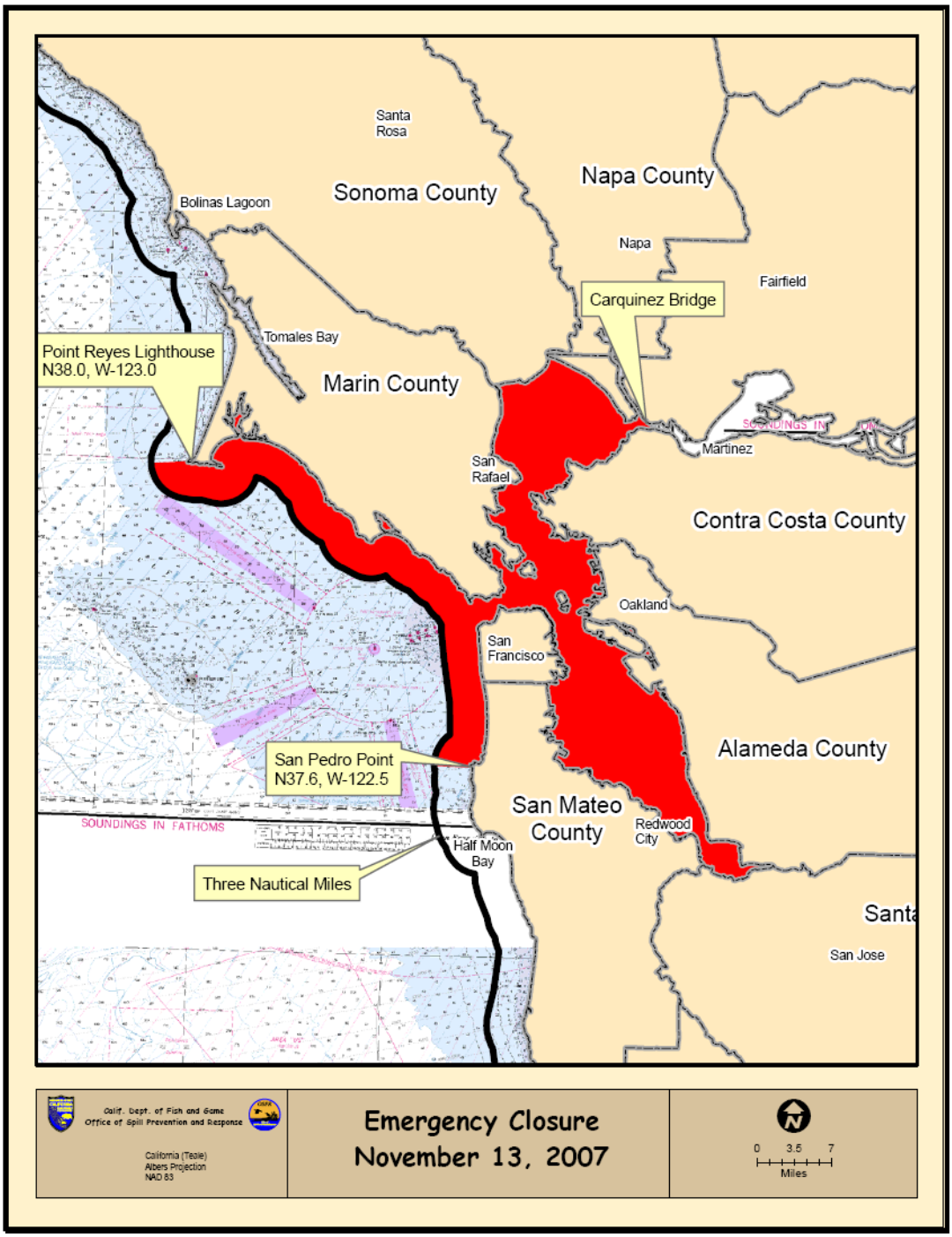


Figure 2. NRDA Mussel and Oyster Sampling in San Francisco Bay

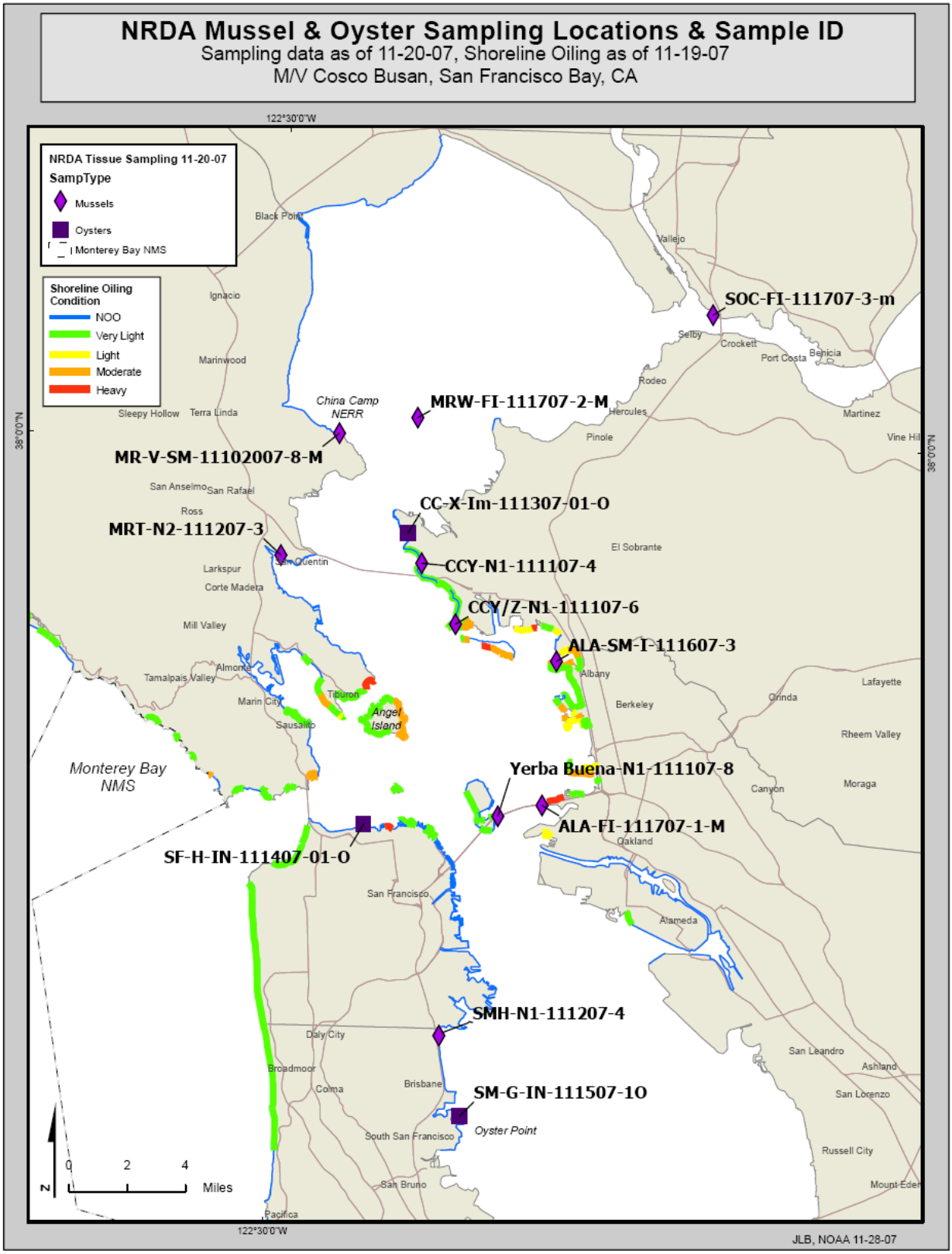


Figure 3. Post-Spill Sampling Locations

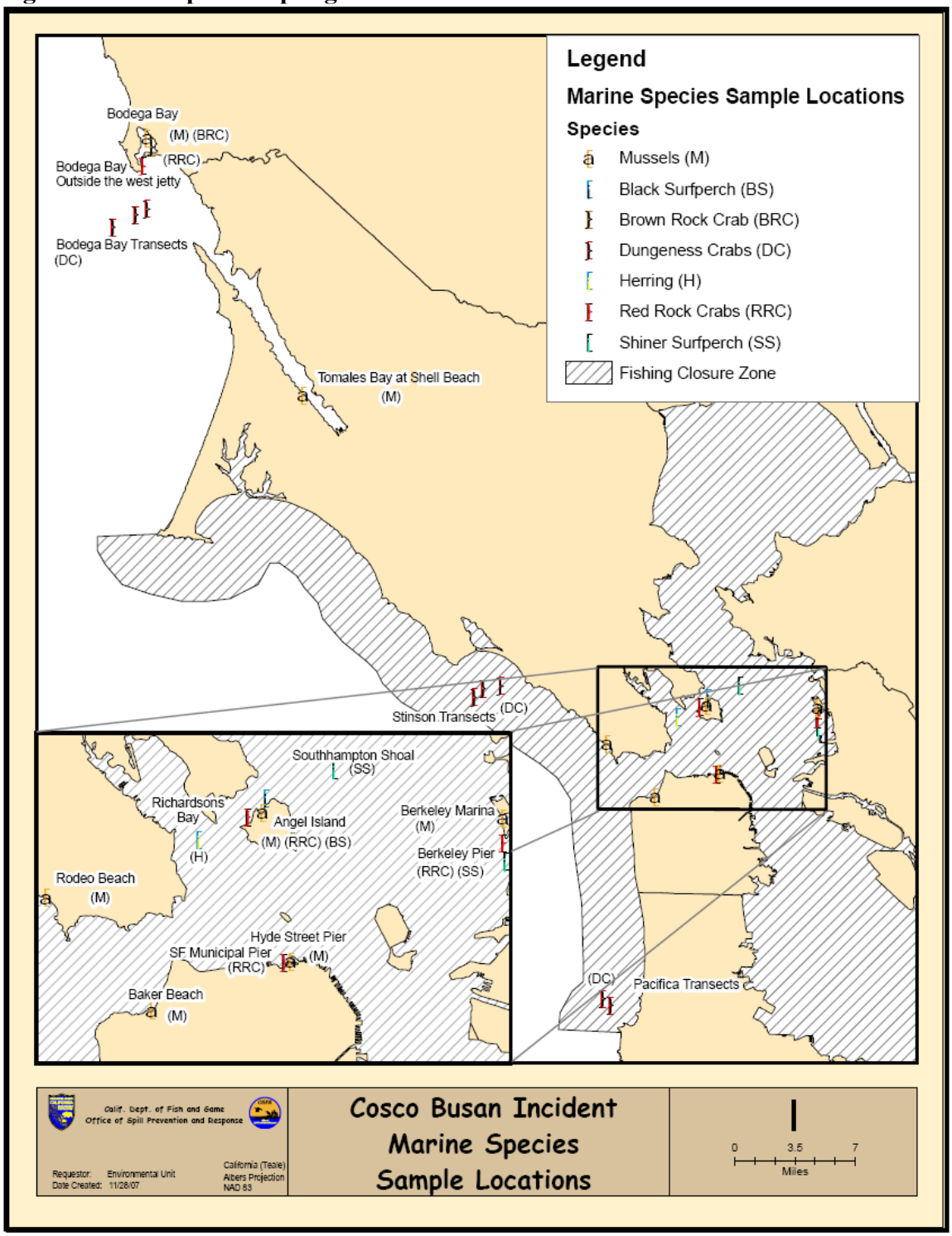
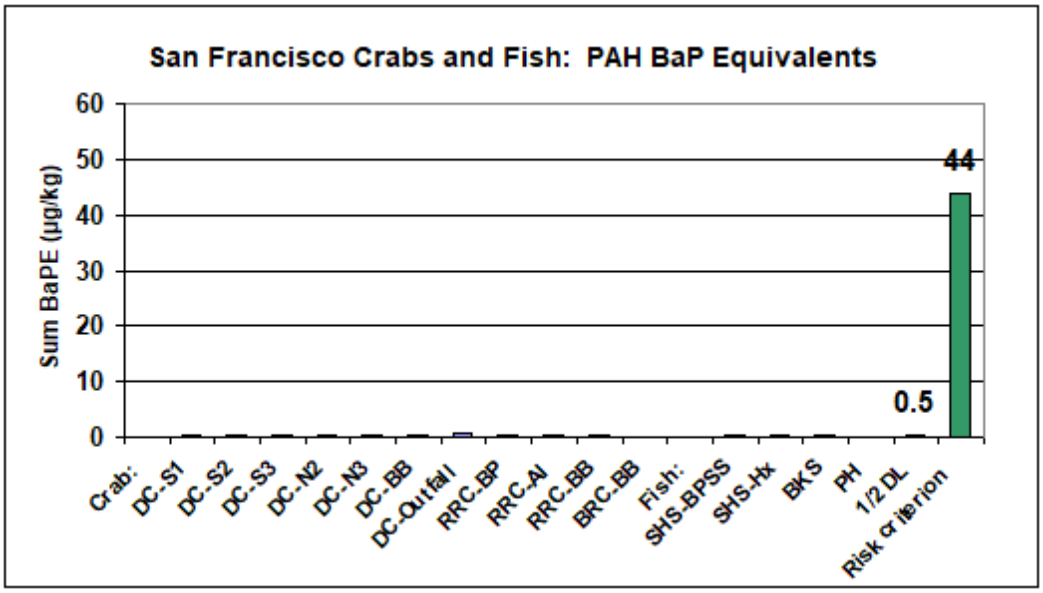


Figure 4. Analytical Results for Fish and Crabs

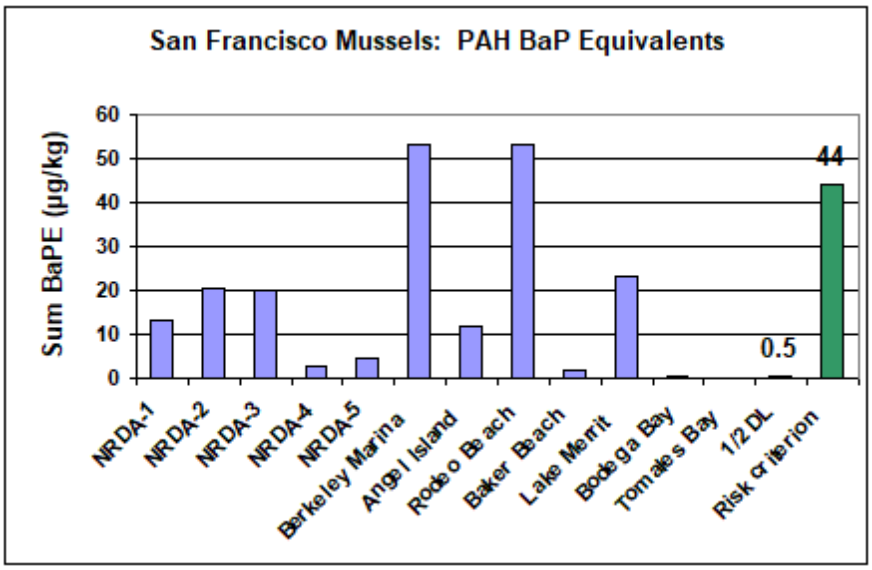


The risk criterion of 44 µg/kg (wet weight) in this graph has been rounded from 43.7 µg/kg (ppb).

Legend for Figure 4:

DC: Dungeness crab, S1: South Coast-1 mile, S2: South Coast-2 mile, S3: South Coast-3 mile, N2: North Coast-2 mile, N3: North Coast-3 mile, BB: Bodega Bay, Outfall: Oceanside Outfall; RRC: red rock crab, BP: Berkeley Pier, AI: Angel Island; BRC: brown rock crab; SHS: shiner surfperch, BPSS: Berkeley Pier/Southhampton Shoal, Hx: Historic Bay; BKS: black surfperch; PH: Pacific herring; ½ DL: ½ Detection Limit

Figure 5. Analytical Results for Mussels



The risk criterion of 44 µg/kg (wet weight) in this graph has been rounded from 43.7 µg/kg (ppb).

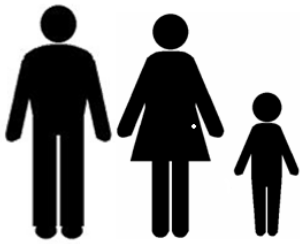
Legend for Figure 5:

NRDA: National Resources Damages Assessment; NRDA-1: SF Bay mid CCY-N1-111107-4; NRDA-2: CCY/Z-N1-111107-6; NRDA-3: YerbaBuena-N1-111107-3; NRDA-4: MRT-N2-111207-3; NRDA-5: SMH-N1-111207-4; ½ DL: ½ Detection Limit

Figure 6.

Health Advisory

ALL CONSUMERS



For Berkeley Marina
And Rodeo Beach

Do not eat mussels from these locations until further notice

Appendix 1

Gov. Schwarzenegger Suspends Fishing, Expedites Review of Environmental Health Concerns Relating to San Francisco Bay Oil Spill

Governor Schwarzenegger today issued the following executive order to suspend all fishing for human consumption including the start of crab season in the San Francisco Bay in response to last week's oil spill. Additionally, the Governor directed the Office of Environmental Health Hazard Assessment in consultation with the California Department of Public Health to expeditiously review the available scientific information on whether a significant human health risk is posed by the consumption of marine life caught in the threatened area.

"We must protect public health and that is why I am signing this executive order today that will suspend harvesting of all marine life for human consumption in the areas affected by the spill. And we will continue to look at any other steps we need to take right now to protect the public and the Bay Area," said Governor Schwarzenegger.

"Our priority must be getting the oil cleaned up as quickly as possible, rescuing all marine life and most importantly protecting the public health."

The suspension is for all fishing for human consumption in the areas affected by the oil spill beginning November 15, 2007 until December 1, 2007 or when the Department of Fish and Game and state health officials determine the fishing season can be opened.

On Friday, the Governor proclaimed a State of Emergency in response to the disaster to help expedite the cleanup effort.

On Saturday, Bay Area crab fishermen voted to officially request that the season be delayed.

The text of the Governor's executive order is below:

EXECUTIVE ORDER S-14-07

WHEREAS, on November 8, 2007, a major oil spill occurred when the container ship COSCO BUSAN struck the fender surrounding a footing of the western span of the Bay Bridge in the San Francisco Bay; and

WHEREAS, on November 9, 2007, I proclaimed a State of Emergency in the City and County of San Francisco and the counties of Alameda, Contra Costa, Marin, San Mateo, Solano and Sonoma due to the effects of this major oil spill; and

WHEREAS, the conditions caused by the oil spill continue to create conditions of extreme peril to the safety of persons and property in the area; and

WHEREAS, the oil spill continues to threaten marine life in the area, including marine mammals, birds, crabs, herrings and other fish populations; and

WHEREAS, sea water contaminated from the oil spill continues to move in and around the San Francisco Bay due to tide and weather; and

WHEREAS, at this time, the human health risk posed by the human consumption of crab, herring and other marine life caught in the oil spill area is unknown.

NOW, THEREFORE, I, ARNOLD SCHWARZENEGGER, Governor of the State of California, in accordance with the authority vested in me by the State Constitution, statutes of the State of California, including the Emergency Services Act and in particular Government Code sections 8567 and 8571, do hereby issue the following order to become effective immediately:

IT IS HEREBY ORDERED THAT:

1. The Department of Fish and Game, in consultation with the Office of Oil Spill Prevention and Response (OSPR), shall determine the geographic area impacted by the oil spill that poses a potential risk to human health that may come from the human consumption of marine life as a result of the oil spill.

2. The Office of Environmental Health Hazard Assessment (OEHHA), in consultation with the Department of Public Health, shall expeditiously review the available scientific information to determine whether a significant human health risk is posed by the human consumption of marine life caught in the area impacted by the oil spill.

3. The applicable sections of the California Fish and Game Code are suspended for all fishing seasons that are open or scheduled to open between November 8, 2007 and December 1, 2007, to the extent that such marine life is being taken for human consumption in the area impacted by the oil spill, such area to be determined by the Department of Fish and Game, in consultation with OSPR. This suspension shall remain in effect until December 1, 2007 unless modified by the Director of the Department of Fish and Game upon consultation with OEHHA and the Department of Public Health on whether a significant human health risk is posed by the human consumption of marine life caught in the area impacted by the oil spill.

This Order is not intended to, and does not, create any rights or benefits, substantive or procedural, enforceable at law or in equity, against the State of California, its departments, agencies, or other entities, its officers or employees, or any other person.

I FURTHER DIRECT that as soon as hereafter possible, this Order shall be filed with the Office of the Secretary of State and that widespread publicity and notice be given to this Order.

IN WITNESS WHEREOF I have hereunto set my hand and caused the Great Seal of the State of California to be affixed this 13th day of November 2007.

_____
ARNOLD SCHWARZENEGGER
Governor of California

ATTEST:

_____
DEBRA BOWEN
Secretary of State

Appendix 2

*Benzo[*A*]pyrene AND POLYCYCLIC AROMATIC HYDROCARBONS.*

This secion was prepared by Prepared by: John D. Budroe, Ph.D., James F. Collins, Ph.D., Melanie A. Marty, Ph.D., Andrew G. Salmon, M.A., D. Phil., George V. Alexeeff, Ph.D.

CAS No: 50-32-8

I. PHYSICAL AND CHEMICAL PROPERTIES (From HSDB, 1998)

Molecular weight	252.3
Boiling point	360° C
Melting point	179° C
Vapor pressure	1 mm Hg at 20° C
Air concentration conversion	1 ppm = 10.3 mg/m^3

II. HEALTH ASSESSMENT VALUES

Unit Risk Factor:		1.1 E-3 $(ug/m^3)^{-1}$
Slope Factor:	(inhalation)	3.9 E+0 $(mg/kg\text{-}day)^{-1}$
	(oral)	1.2 E+1 $(mg/kg\text{-}day)^{-1}$

[Inhalation: male hamster respiratory tract tumor incidence (Thyssen *et al.*, 1981), unit risk calculated using a linearized multistage procedure (OEHHA, 1993). Oral: male and female gastric tumor (papillomas and squamous cell carcinomas) incidence (Neal and Rigdon, 1967), cancer potency factor calculated using a linearized multistage procedure (OEHHA, 1993).]

III. CARCINOGENIC EFFECTS

Human Studies

The predominant sources of airborne benzo[*a*]pyrene (BaP) are combustion processes. Thus, this compound rarely enters the environment alone but rather is associated with additional PAHs and other components frequently present in both vapor phase and particulate form. Available epidemiological information, therefore, is from persons exposed to mixtures such as tobacco smoke, diesel exhaust, air pollutants, synthetic fuels, or other similar materials. Several IARC publications have been dedicated to the analysis of cancer in processes which involve exposure to polynuclear aromatic compounds (PAHs) (IARC, 1983; 1984a; 1984b; 1985; 1987). The types of cancer reported are often consistent with the exposure pathway: scrotal cancer and lung cancer in chimney sweeps exposed to soot; skin cancer (including scrotal cancer) where shale oils are used; and lung cancer where airborne exposure of PAHs occurs, such as in iron and steel foundries.

Shamsuddin and Gan (1988) examined several human tissues collected at surgery or autopsy using rabbit high-specificity antibody to benzo[*a*]pyrene diol epoxide (BPDE)-DNA adducts and light immunocytochemistry. Antigenicity was detected in the lung, ovary, placenta, uterine cervix, and white blood cells. Their results indicated that the tissue concentration of adducts varies substantially in the human population and that BPDE-DNA adducts can be detected in human tissues by immunochemical techniques.

Five of twelve human lung samples obtained at surgery, from smokers or former smokers, showed positive antigenicity for BPDE-DNA adducts (Garner *et al.*, 1988). Higher DNA-adduct levels were detected in the white blood cells of Finnish iron workers with jobs in high PAH exposure areas than in the white blood cells of workers with jobs in low PAH exposure areas (Perera *et al.*, 1988; Hemminki *et al.*, 1990). Workers were classified as high, medium, or low BaP exposure and there was a highly significant correlation between BaP exposure and DNA-adduct levels (Reddy *et al.*, 1991). A similar observation was noted by Ovrebo *et al.* (1992) in a study of workers exposed around coke ovens. Perera *et al.* (1993) extended the technique and found that PAH adducts were higher in an industrialized area in winter than both in a more rural area in winter and in the same urban area in summer (when less burning of fuel would occur).

In studies looking at PAH-derived adducts bound to serum protein, higher levels of PAH-albumin adducts were found in foundry workers and in roofers than in their respective reference groups (Lee *et al.*, 1991). Smokers had higher levels of BaP-derived adducts bound to serum protein than non-smokers, and workers in high BaP exposure areas (foundry) had two to three times the levels of workers in low exposure areas (Sherson *et al.*, 1990).

Studies with human placental tissues have shown that aryl hydrocarbon hydroxylase (AHH) activity is several times higher in smokers than non-smokers and that this activity increases in a sigmoidal manner with increased numbers of cigarettes smoked (Gurtoo *et al.*, 1983). Genetic factors probably contribute to this variability and, ultimately, to susceptibility of individuals to tumor development (Manchester and Jacoby, 1984).

Animal Studies

BaP is carcinogenic by intratracheal, inhalation, and dermal exposure, by intraperitoneal injection, and when given in the diet.

(a) Inhalation and Intratracheal Exposures

Early experiments by Saffiotti *et al.* (1968) indicated that PAHs are at least weakly carcinogenic to the respiratory tract. A mixture of BaP (3 mg) and Fe_2O_3 (hematite, 0.25 μm) (3 mg) in a saline suspension was administered to Syrian golden hamsters by intratracheal instillation, once per week for 15 weeks. Most surviving animals receiving BaP plus Fe_2O_3 developed tumors of the respiratory tract (mostly bronchogenic carcinoma) whereas control animals receiving Fe_2O_3 only or those receiving no treatment did not develop tumors.

Subsequently, Saffiotti *et al.* (1972) determined the carcinogenic dose-response relationship after intratracheal instillation of a suspension of BaP and Fe_2O_3 in male and female Syrian golden hamsters. Test materials were administered once weekly for 30 weeks at 2.0, 1.0, 0.5, and 0.25 mg BaP/animal and an equivalent weight of Fe_2O_3 (hematite) as particulate carrier. Tumors were not present in animals receiving ferric oxide or in untreated controls. Respiratory tract tumors (including squamous cell carcinomas of the larynx, of the trachea, and of the bronchi, adenocarcinomas of the bronchi, and adenomas of the bronchi and of the bronchioles and alveoli) developed in all groups of BaP/Fe_2O_3 treated animals. The response was dose related.

In another experiment, Feron *et al.* (1973) gave male Syrian golden hamsters intratracheal doses of 0, 0.0625, 0.125, 0.5, or 1 mg BaP weekly for 52 weeks. A variety of tumors were produced throughout the respiratory tract, including bronchoalveolar adenomas and carcinomas.

Thyssen *et al.* (1980) conducted an inhalation study in which male Syrian golden hamsters were exposed to BaP condensation aerosol (in 0.1% saline; particle size ranging from 0.2 to 1.5 μm) for 10 to 16 weeks at a concentration of 9.8 to 44.8 mg BaP/m^3. Neoplastic changes in the respiratory tract were not seen.

In a subsequent experiment, Thyssen *et al.* (1981) exposed male Syrian golden hamsters to BaP condensed onto sodium chloride particles at BaP concentrations of 2.2, 9.5, and 46.5 mg BaP/m^3. Tumors were not observed in the respiratory tract of the unexposed control group or the group that received 2.2 mg/m^3. The incidence of tumors in this organ system increased in a dose dependent manner for the 9.5 and 46.5 mg/m^3 exposure groups. Papillomas, papillary polyps, and squamous cell carcinomas were seen in the nasal cavity, larynx, trachea, pharynx, esophagus, and forestomach. Lung tumors were absent.

(b) Feeding Studies

Feeding of pelletized chow containing BaP (50 to 250 ppm BaP for 4 to 6 months) to male and female CFW mice caused gastric tumors (papillomas and squamous cell carcinomas), pulmonary adenomas, and leukemia (Rigdon and Neal, 1966; 1969; Neal and Rigdon, 1967). The pulmonary adenomas, gastric tumors, and leukemia occurred independently of each other (Rigdon and Neal, 1969). The overall data strongly suggest a positive carcinogenic effect since there were no gastric tumors in 289 control mice while 178 out of 454 mice fed various levels of BaP had gastric tumors (Neal and Rigdon, 1967).

(c) Dermal Application

BaP has been shown to be carcinogenic by dermal application (ATSDR, 1990). Wynder and associates demonstrated a positive dose-response relationship for BaP-induction of skin tumors in Swiss and in C57BL mice and showed a tumor response at doses as low as

0.001% BaP applied topically in acetone every 2 weeks for up to 2 years (Wynder and Hoffmann, 1959; Wynder *et al.*, 1957; 1960). In addition, incidences of 95% for papillomas and carcinomas of the skin were obtained by chronic administration (3 times weekly for 1 year) of 0.001% BaP to the skin of Swiss mice (Wynder and Hoffman, 1959). Extensive experiments conducted by Conney and associates demonstrated the tumor initiating activity of BaP and several of its epoxide and hydroxy derivatives (summarized by US EPA, 1979 and by Conney, 1982).

IV. DERIVATION OF CANCER POTENCY

Basis for Cancer Potency

A very large number of experiments have demonstrated that BaP causes tumors at several sites, by several routes of administration, in both sexes, and in several animal species. Many studies, however, are very limited in scope or in data reported and are not suitable for risk assessment (Zeise and Crouch, 1984).

OEHHA guidelines prescribe that risk assessment use the most sensitive sex, site, and species where a significant increase in cancer incidence is observed (CDHS, 1985). Since there is no adequate information regarding the carcinogenicity of BaP to humans from epidemiological studies, data from animal bioassays were extrapolated to estimate human cancer risk. Potency estimates were derived by OEHHA (1993) from gastric tumors (papillomas and squamous cell carcinomas) observed in male and female mice due to feeding of BaP (Neal and Rigdon, 1967), respiratory tract tumors in hamsters from the inhalation bioassay of Thyssen *et al.* (1981), and from data obtained after intratracheal administration of BaP (Saffiotti *et al.*, 1972; Feron *et al.*, 1973). The dose-response data from these studies are presented in Tables 1-4 below.

Table 1: Gastric tumors in mice from feeding benzo[*a*]pyrene[a].

Exposure (ppm)	Calculated daily dose (mg/kg-day) (animal)	Incidence of gastric tumors
0	0	0/289
1	0.078	0/25
10	0.781	0/24
20	1.563	1/23
30	2.344	0/37
40	3.126	1/40
45	3.516	4/40
50	3.908	24/34
100	7.815	19/23
250	19.538	66/73

[a]Source: OEHHA (1993). Adapted from Neal and Rigdon (1967) and US EPA (1984).

Table 2: Respiratory tract tumors in hamsters from benzo[*a*]pyrene inhalation[a]

Exposure (mg/m^3)	Hamster dose (mg/kg-day)		Tumor incidence
	based on U.S EPA (1994)	based on US EPA (1988)	
0	0	0	0/27
2.2	0.089	0.152	0/27
9.5	0.385	0.655	9/26
46.5			13/25[b]

[a]Source: OEHHA (1993). Adapted from Thyssen *et al*. (1981) and US EPA (1984)
[b]These data were not used due to shortened lifespan of the hamsters in the exposure group. The carcinogenic response, however, is apparent.

Table 3: Respiratory tract tumors from intratracheal instillation of benzo[*a*]pyrene in hamsters – 30 week exposure[a].

Weekly Dose (mg)	Average Daily Dose (mg)	Lifetime Adjusted Daily Dose (mg/kg-day)	Human Equivalent Dose (mg/kg-day)	Tumor Incidence (Males)	Tumor Incidence (Females)
0	0	0	0	0/47	0/45
0.25	0.036	0.119	0.013	6/47	4/41
0.5	0.071	0.239	0.027	10/33	9/30
1.0	0.143	0.477	0.054	22/33	20/34
2.0	0.286	0.953	0.107	17/28	17/29[b]

[a]Source: OEHHA (1993). Adapted from Saffiotti *et al.*, 1972.
[b]Data group was not used since exposure started 7 weeks after other groups.

Table 4: Bronchoalveolar tumors from intratracheal instillation of benzo[*a*]pyrene in hamsters – 52 week exposure[a].

Weekly dose (mg)	Average daily dose (mg)	Lifetime adjusted daily dose (mg/kg-day)	Human equivalent dose (mg/kg-day)	Tumor incidence
0	0	0	0	0/29
0.0625	0.009	0.0495	0.0059	1/30
0.125	0.018	0.0989	0.0118	4/30
0.25	0.036	0.198	0.0237	6/30
0.5	0.071	0.395	0.0473	17/30
1.0	0.143	0.791	0.0947	19/30

[a]Source: OEHHA (1993). Adapted from Feron *et al.*, 1973.

Methodology

Cancer risk associated with exposure to ambient levels of BaP was estimated by extrapolating from the experimental data to ambient levels by means of the best fitting linearized multistage procedure GLOBAL86 (Howe *et al.*, 1986). In addition, other models were fit to the data for comparison. In its risk assessment, the US EPA used the data for stomach tumors from oral exposure to BaP in mice and the data for respiratory tract tumors from inhalation exposure in hamsters to estimate cancer potency and unit risks associated with exposure to BaP (US EPA, 1984).

For BaP there is compelling evidence that it is genotoxic and an initiator of tumorigenesis. Therefore, OEHHA staff treated BaP-induced carcinogenesis as a nonthreshold phenomenon and, as such, applied a nonthreshold, linear extrapolation model for cancer potency estimation.

The linearized multistage model was fit to the respiratory tract tumor data resulting from inhalation exposure of hamsters to BaP (OEHHA, 1993; Thyssen *et al.*, 1981). The data from the highest dose group were not used since these animals had an appreciably shortened lifespan (59 weeks versus 96 weeks in other groups) (Thyssen *et al.*, 1981; US EPA, 1984). By considering the conditions of exposure given in the report and using an inhalation rate of 0.063 m^3/day and a "standard" body weight of 0.12 kg for hamsters (US EPA, 1988), a dose of BaP in mg/kg-day was estimated. A q_1^* (animal) equal to 0.43 $(mg/kg\text{-}day)^{-1}$ is obtained. Multiplying by the interspecies surface area correction factor of $(70/0.1)^{1/3}$ yields a human equivalent $q_1^* = 1.1 \times 10^{-3}$ $(\mu g/m^3)^{-1}$ for inhalation.

Because of the limited amount of data currently available for risk assessment of BaP, the inhalation unit risk of 1.1×10^{-3} $(\mu g/m^3)^{-1}$ based on respiratory tract tumors in hamsters is used as a best value for inhalation exposures. For exposures to BaP by other routes, the potency of 11.5 $(mg/kg\text{-}day)^{-1}$ based on gastric tract tumors in mice can be used (Neal and Rigdon, 1967).

Cancer Potency for Other PAHs

IARC (1987; 1989) has classified a number of PAHs, their mixtures and derivatives, as carcinogens (Group 1, Groups 2A and Group 2B) and a large number of PAHs into Group 3, a class of chemicals for which there are no human data but limited or inadequate data in animals (Tables 5 and 6). The US EPA has classified several PAHs in Group B2, possibly carcinogenic to humans and Group D, unclassifiable as to carcinogenicity (Table 7).

In their risk assessment, OEHHA staff concluded that while the studies available for carcinogenic risk assessment of BaP are not ideal for risk assessment, those for practically all other individual PAHs are less complete for risk assessment (OEHHA, 1993). However, there are extensive data establishing the genotoxicity, and in some cases the carcinogenicity, of many PAHs or their genotoxic metabolites. In other cases, some PAHs are not considered carcinogens. Several authors have used mutagenicity and

various tests of carcinogenicity to rank several PAHs for their relative carcinogenicity (e.g., Deutsch-Wenzel *et al.*, 1983; Bingham and Falk, 1969; Habs *et al.*, 1980; Wynder and Hoffman, 1959; Wislocki *et al.*, 1986) and their relative genotoxicity (Brown, 1989). Many of these comparisons were summarized by Clement Associates (1988) and Krewski *et al.* (1989). In these analyses dibenz(*a,h*)anthracene was shown to be more potent than BaP, while other PAHs tested were less or much less potent. These comparisons indicated that considering all PAHs to be equivalent in potency to BaP would overestimate the cancer potency of a PAH mixture, but such an assumption would be health protective and is likely to be helpful in a screening estimate of PAH risks (OEHHA, 1993).

If one assumes that PAHs are as carcinogenic as they are genotoxic, then their hazard relative to BaP would be dependent on their concentration in the environment. In light of the limited information available on other PAHs, BaP remains an important representative or surrogate for this important group of chemically diverse air pollutants.

Selection of Risk Values for Other PAHs

BaP was chosen as the primary representative of the class because of the large amount of toxicological data available on BaP (versus the relatively incomplete database for other PAHs), the availability of monitoring techniques for BaP, and the significant exposure expected (and found). Nisbet and LaGoy (1992) presented a Toxic Equivalency Factor (TEF) scheme for 17 PAHs. The paper was an extension of earlier work by other investigators (Clement Associates, 1987; 1988; Krewski *et al.*, 1989). Along similar lines, OEHHA has developed a Potency Equivalency Factor (PEF) procedure to assess the relative potencies of PAHs and PAH derivatives as a group. This would address the impact of carcinogenic PAHs in ambient air since they are usually present together.

Table 5: IARC groupings of PAHs, mixtures with PAHs, and derivatives.

Group 1	Group 2A	Group 2B
Coal-tar pitches Coal-tar Coke production Mineral oils Shale-oils Soots Tobacco smoke	Benz[*a*]anthracene Benz[*a*]pyrene Creosotes Dibenzo[*a,h*]anthracene	Benzo[*b*]fluoranthene Benzo[*j*]fluoranthene Benzo[*k*]fluoranthene Carbon black extracts Dibenz[*a,h*]acridine Dibenz[*a,j*]acridine 7H-Dibenzo[*c,g*]carbazole Dibenzo[*a,e*]pyrene Dibenzo[*a,h*]pyrene Dibenzo[*a,i*]pyrene Dibenzo[*a,l*]pyrene Indeno[1,2,3-*cd*]pyrene 5-Methylchrysene 5-Nitroacenaphthene 1-Nitropyrene 4-Nitropyrene 1,6-Dinitropyrene 1,8-Dinitropyrene 6-Nitrochrysene 2-Nitrofluorene

Source: OEHHA (1993)
Abstracted from IARC Supplement 7 (1987) and IARC Volume 46 (1989).
Group1: carcinogenic to humans.
Group 2A: probably carcinogenic to humans.
Group 2B: possibly carcinogenic to humans.

Table 6: IARC Group 3 PAHs and PAH derivatives[1]

Chemical	Animal Evidence
Acridine orange	inadequate
5-Aminoacenaphthene	inadequate
2-Aminoanthraquinone	limited
Anthanthrene	limited
Anthracene	inadequate
Benz[*a*]acridine	inadequate
Benz[*c*]acridine	limited
Benzo[*g*,*h*,*i*]fluoranthene	inadequate
Benzo[*g*,*h*,*i*]perylene	inadequate
Benzo[*c*]phenanthrene	inadequate
Benzo[*e*]pyrene	inadequate
Carbazole	limited
Chrysene	limited
Cyclopenta[*c*,*d*]pyrene	limited
Dibenz[*a*,*c*]anthracene	limited
Dibenz[*a*,*j*]anthracene	limited
Dibenz[*a*,*e*]fluoranthene	limited
Dibenzo[*h*,*rst*]pentaphene	limited
3,7-Dinitrofluoroanthene	limited
3,9-Dinitrofluoroanthene	limited
1,3-Dinitropyrene	limited
Fluoranthene	inadequate
Fluorene	inadequate
1-Methylchrysene	inadequate
2-Methylchrysene	limited
3-Methylchrysene	limited
4-Methylchrysene	limited
6-Methylchrysene	limited
2-Methylfluoranthene	limited
1-Methylphenanthrene	inadequate
1,5-Naphthalenediamine	limited
9-Nitroacenaphthene	limited
9-Nitroanthracene	no adequate data
7-Nitrobenz[*a*]anthracene	limited
6-Nitrobenzo[*a*]pyrene	limited
3-Nitrofluoranthene	inadequate
1-Nitronaphthalene	inadequate
2-Nitronaphthalene	inadequate
3-Nitroperylene	inadequate
2-Nitropyrene	inadequate
Perylene	inadequate
Phenanthrene	inadequate
N-Phenyl-2-naphthylamine	limited
Pyrene	inadequate
Triphenylene	inadequate

Table 6 (continued): IARC Group 3 PAHs and PAH derivatives[1].

[1]Source: OEHHA (1993). Abstracted from IARC Supplement 7 (1987) and IARC Volume 46. (1989). Group 3 have either limited or inadequate evidence in animals and are not classifiable as to their carcinogenicity in humans due to no adequate data.

Table 7: US EPA groupings of PAHs[1]

Group B2	Group D
Benz[*a*]anthracene Benzo[*a*]pyrene Benzo[*b*]fluoranthene Benzo[*j*]fluoranthene Benzo[*k*]fluoranthene Chrysene Dibenz[*a,h*]anthracene Indeno[1,2,3-*cd*]pyrene```	Acenaphthylene Anthracene Benzo[*e*]pyrene Benzo[*g,h,i*]perylene Fluorene Naphthalene Phenanthrene

[1]Source: OEHHA (1993). Abstracted from US EPA (1993a). Group B2: possibly carcinogenic to humans. Group D is unclassifiable as to carcinogenicity.

Due to the variety of data available on the carcinogenicity and mutagenicity of PAHs, an order of preference for the use of available data in assessing relative potency was developed. If a health effects evaluation and quantitative risk assessment leading to a cancer potency value had been conducted on a specific PAH, then those values were given the highest preference.

If potency values have not been developed for specific compounds, a carcinogenic activity relative to BaP, rather than a true potency, can be developed. These relative activity values are referred to by OEHHA as PEFs. For air contaminants, relative potency to BaP based on data from inhalation studies would be optimal. Otherwise, intrapulmonary or intratracheal administration, such as those published by Deutsch-Wenzel *et al.* (1983), would be most relevant, since such studies are in the target organ of interest. Next in order of preference is information on activity by the oral route and skin painting. Intraperitoneal and subcutaneous administration rank at the bottom of the *in vivo* tests considered useful for PEF development because of their lack of relevance to environmental exposures. Next in decreasing order of preference are genotoxicity data which exist for a large number of compounds. In many cases genotoxicity information is restricted to mutagenicity data. Finally, there are data on structure-activity relationships among PAH compounds. Structure-activity considerations may help identify a PAH as carcinogenic, but at this time have not been established as predictors of carcinogenic potency.

Using this order of preference, PEFs were derived for 21 PAHs and are presented in Table 8. The cancer potencies of four other PAH compounds are given in Table 9. Explanation of the derivation of each PEF, type of data used in the derivation, and the relevant references are given below.

Table 8: OEHHA PEF weighting scheme for PAHs[1]

PAH or derivative	PEF
benzo[*a*]pyrene	1.0 (index compound)
benz[*a*]anthracene	0.1
benzo[b]fluoranthene	0.1
benzo[*j*]fluoranthene	0.1
benzo[k]fluoranthene	0.1
dibenz[*a*,*j*]acridine	0.1
dibenz[*a*,*h*]acridine	0.1
7H-dibenzo[c,*g*]carbazole	1.0
dibenzo[*a*,*e*]pyrene	1.0
dibenzo[*a*,*h*]pyrene	10
dibenzo[*a*,*i*]pyrene	10
dibenzo[*a*,*l*]pyrene	10
indeno[1,2,3-*cd*]pyrene	0.1
5-methylchrysene	1.0
1-nitropyrene	0.1
4-nitropyrene	0.1
1,6-dinitropyrene	10
1,8-dinitropyrene	1.0
6-nitrochrysene	10
2-nitrofluorene	0.01
chrysene	0.01

[1]Source: OEHHA (1993)

Table 9: Potencies of PAHs and derivatives[1]

Chemicals	Cancer potency factors $(mg/kg\text{-}day)^{-1}$	Unit risks $(\mu g/m^3)^{-1}$
benzo[*a*]pyrene	11.5	1.1×10^{-3}
dibenz[*a*,*h*]anthracene	4.1	1.2×10^{-3}
7,12-dimethylbenzanthracene	250	7.1×10^{-2}
3-methylcholanthrene	22	6.3×10^{-3}
5-nitroacenaphthene	0.13	3.7×10^{-5}

[1]Source: OEHHA (1993). It is assumed that unit risks for inhalation have the same relative activities as cancer potencies for oral intake.

Potency and Potency Equivalency Factors (PEFs) for Selected PAHs

1. Benzo[*a*]pyrene. Benzo[*a*]pyrene (BaP) was the index compound for relative potency and for Potency Equivalency Factors (PEF) for PAHs and derivatives. It has a cancer potency of 11.5 $(mg/kg\text{-}day)^{-1}$ and inhalation unit risk of 1.1×10^3 $(\mu g/m^3)^{-1}$. For the potency equivalency scheme, it was assigned a PEF of 1.

2. Dibenz[*a*,*h*,]anthracene. An expedited potency of 4.1 $(mg/kg\text{-}day)^{-1}$ was derived using the linearized multistage model with the only dose-response data set available - a drinking water study (Snell and Stewart, 1962) which reported alveolar carcinomas of the lung in male DBA/2 mice due to dibenz[*a*,*h*]anthracene (incidence = 14/21 at 28.3 mg/kg-day versus 0/25 in controls). An inhalation unit risk can be obtained from a potency under the assumption that the chemicals are equally absorbed and are equally potent by oral and inhalation routes and that a 70 kg person inhales 20 cubic meters of air per day. When the potency in units of $(mg/kg\text{-}day)^{-1}$ is divided by 3500 (70 kg * 1000 $\mu g/mg/20\ m^3$), an inhalation unit risk is obtained in units of $(\mu g/m^3)^{-1}$.

3. 7,12-Dimethylbenzanthracene. An expedited potency of 250 $(mg/kg\text{-}day)^{-1}$ was derived. The only study listed in the Gold *et al.* cancer potency (TD50) database (Gold *et al.*, 1984; 1986; 1987; 1989; 1990) is the feeding study by Chourolinkov *et al.* (1967) in female albino mice. Significant increases in malignant angioendotheliomas of the mesenteric intestine and papillomas of the forestomach were observed in animals treated with 0.39 mg/kg-day of 7,12-dimethylbenzanthracene. Cancer potency is based on mesenteric intestine angioendothelioma incidence (incidence = 49/75 versus 0/40 in controls).

4. 3-Methylcholanthrene. An expedited potency of 22 $(mg/kg\text{-}day)^{-1}$ was derived. Results of 3 studies in male Long Evans rats, one study in an unspecified strain of female rats, and 10 studies in female Wistar rats were included in the Gold *et al.* database. All studies in female rats found highly significant increases in tumors of the mammary gland. The cancer potency for 3-methylcholanthrene was taken as the geometric mean of cancer potencies estimated from 9 of the 10 studies in female rats (Shay *et al.*, 1962; Gruenstein *et al.*, 1964; Shay *et al.*, 1961). The upper bound on potency could not be estimated from one of the studies by Shay *et al.* (1961), because 100% of the treated animals developed mammary gland tumors.

5. 5-Nitroacenaphthene. An expedited potency of 0.13 $(mg/kg\text{-}day)^{-1}$ was derived based on the combined incidence of benign and malignant tumors of the ear canal in female rats. Usable studies were feeding studies by Takemura *et al.* (1974) in female Syrian golden hamsters and by the National Cancer Institute (1978) in male and female $B6C3F_1$ mice and F344 rats. The compound 5-nitroacenaphthene induced increases in tumor incidences at multiple sites in rats and female mice. Rats were the most sensitive species; the sensitivity of males were similar to that of females.

6. Benzo[*b*]fluoranthene. Benzo[*b*]fluoranthene was assigned a PEF of 0.1. Clement Associates (1988) applied both a two stage model and the multistage model to various data sets for several PAHs. The two models generally gave similar results for relative potency. In order to verify the results, OEHHA staff (OEHHA, 1993) used GLOBAL86 to fit the multistage model to the tumor data used by Clement Associates and obtained relative cancer potencies similar to those obtained by Clement Associates. Clement Associates (1988) used the mouse skin carcinogenesis data obtained by Habs *et al.* (1980) and the intrapulmonary administration to rats by Deutsch-Wenzel *et al.* (1983) to estimate a cancer potency for benzo[*b*]fluoranthene relative to BaP. As an example of

the type of data used, Deutsch-Wenzel *et al.* obtained pulmonary tumor incidences of 0, 2.9, and 25.7% after intrapulmonary administration of 0.1, 0.3, and 1 mg benzo[*b*]fluoranthene, respectively, whereas they obtained 11.8, 60.0, and 94.3% tumor incidence after the same doses of benzo[*a*]pyrene. Clement Associates estimated a relative cancer potency for benzo[*b*]fluoranthene of 0.140 after fitting the two stage model to the data and 0.105 after fitting the multistage model. Using the data of Habs *et al.* a relative cancer potency of 0.167 was obtained with the two stage model and 0.201 with the multistage model. The results from the multistage model were averaged, then rounded (down) to 0.1 to obtain the PEF. OEHHA obtained a relative potency of 0.208 for benzo[*b*]fluoranthene fitting the multistage model to the data from Habs *et al.* OEHHA staff were also able to reproduce the calculations for the two stage model in the accepted model for cancer risk assessment in California; results from the multistage model have been used to obtain PEFs although the two models usually gave the same PEF.

7. Benzo[*j*]fluoranthene. Benzo[*j*]fluoranthene was assigned a PEF of 0.1. Clement Associates (1988) used the mouse skin carcinogenesis data obtained by Habs *et al.* (1980) to estimate a cancer potency relative to BaP of 0.0648. OEHHA staff estimated 0.065 using the same data. This was rounded to 0.1 to obtain the PEF. Clement Associates did not use the data of Deutsch-Wenzel *et al.* (1983) on benzo[*j*]fluoranthene to calculate a relative potency but Deutsch-Wenzel *et al.* found that it was very similar in tumorigenic activity to benzo[*k*]fluoranthene.

8. Benzo[*k*]fluoranthene. Benzo[*k*]fluoranthene was assigned a PEF of 0.1. Clement Associates (1988) used mouse skin carcinogenesis data obtained by Habs *et al.* (1980) to obtain a cancer potency relative to BaP of 0.0235 and the intrapulmonary administration to rats by Deutsch-Wenzel *et al.* (1983) to estimate a PEF of 0.085. Because the latter was obtained by the pulmonary route it was chosen to be the basis of the PEF. The value was rounded to 0.1 to obtain the PEF.

9. Benz[*a*]anthracene. Benz[*a*]anthracene was assigned a PEF of 0.1. In the case of benz[*a*]anthracene, mouse skin carcinogenesis data obtained by Bingham and Falk (1969) were used by Clement Associates (1988) to calculate potencies for benz[*a*]anthracene. For this chemical the multistage model gave a relative potency of 0.0137. Using the two stage model a higher cancer potency of 0.145 relative to BaP was obtained. In the Wislocki *et al.* (1986) report, in which lung adenomas were induced in newborn mice, benz[*a*]anthracene (2.8 micromoles) was less carcinogenic (12/71 or 17% versus 7/138 or 5% in controls) relative to 0.56 micromoles BaP (24/64 or 38% versus 7/138 in controls). The relative potency was 0.08, which rounds to 0.1. Since the US EPA was using a PEF of 0.1 for this PAH (US EPA, 1993b) and the data from the Wislocki study were consistent with a PEF of 0.1, a value of 0.1 was selected by OEHHA.

10. Dibenz[*a*,*j*]acridine. Dibenz[*a*,*j*]acridine was assigned a PEF of 0.1. Warshawsky *et al.* (1992) compared the tumor-initiating ability of dibenz[*a*,*j*]acridine to BaP in mouse skin. Two hundred nanomoles of each compound were applied to groups of 30 mice, then the skin lesion was promoted with a phorbol ester for 24 weeks. Twenty-seven out

of 30 BaP mice (90%) had skin papillomas, while 17 of 30 (57%) of the dibenz[*a*,*j*]acridine mice had skin papillomas. The multistage model was fit to both sets of data and the ratio of upper 95% confidence limits on the linear coefficient was 0.36. This was rounded to a PEF of 0.1.

11. Dibenz[*a*,*h*]acridine. Dibenz[*a*,*h*]acridine was also assigned a PEF of 0.1. Its carcinogenic classification by IARC was based on studies published in 1940 and earlier and the studies did not appear appropriate for estimation of a PEF. Since its structure is similar to dibenz[*a*,*j*]acridine, it was assigned the same PEF as dibenz[*a*,*j*]acridine until usable compound-specific bioassay data becomes available.

12. 7H-Dibenzo[*c*,*g*]carbazole. 7H-dibenzo[*c*,*g*]carbazole was assigned a PEF of 1.0. Warshawsky *et al.* (1992) compared the tumor-initiating ability of 7H-dibenzo[*c*,*g*]carbazole to BaP in mouse skin. Two hundred nanomoles of each compound were applied to 30 mice, then promoted with a phorbol ester for 24 weeks. Twenty-seven out of 30 BaP-treated mice (90%) had skin papillomas, while 26 of 30 (87%) of the dibenzo[*a*,*j*]acridine-treated mice had skin papillomas for a relative tumorigenic activity of 0.97. This was rounded to a PEF of 1.

13. Dibenzo[*a*,*l*]pyrene. Dibenzo[*a*,*l*]pyrene was assigned a PEF of 10. Cavalieri *et al.* (1989; 1991) studied the tumor-initiating and dose-response tumorigenicity of dibenzo[*a*,*l*]pyrene in mouse skin and rat mammary gland. BaP was used as a reference compound in some experiments. Dibenzo[*a*,*l*]pyrene was the most potent member of the group. Several levels of PAHs were tested. When results from 33.3 nanomoles of dibenzo[*a*,*l*]pyrene as a skin tumor initiator (with promotion by a phorbol ester) were compared to results using the same amount of BaP, dibenzo[*a*,*l*]pyrene induced skin tumors in 23/24 (96%) of the animals while BaP induced tumors in 10/23 (43%) which resulted in a relative potency of 5.8. Dibenzo[*a*,*l*]pyrene induced approximately 5 times as many tumors per tumor-bearing animal. In a second experiment 4 nanomoles of each chemical were compared. Ninety-two percent (22/24) of the dibenzo[*a*,*l*]pyrene-treated mice had tumors but only 4% (1/24) of the BaP animals, which yielded a relative potency of 25.1. In a third experiment 100 nM were compared without promotion. Twenty-nine percent (7/24) of the dibenzo[*a*,*l*]pyrene-treated mice had tumors but only 4% (1/24) of the BaP animals, for a relative potency of 4. Finally, with direct application to the mammary gland, 0.25 and 1.0 nanomoles dibenzo[*a*,*l*]pyrene led to tumors in all the rats treated (19 and 20 per group, respectively) whereas only one animal in the 0.25 micromoles BaP group showed a tumor for a relative potency greater than 100. Based on its much greater tumorigenic activity than BaP in the above tests, dibenzo[*a*,*l*]pyrene was assigned a PEF of 10.

14. Dibenzo[*a*,*h*]pyrene. Dibenzo[*a*,*h*]pyrene was assigned a PEF of 10 since, in the experiments by Cavalieri *et al.* (1989) in which all four dibenzo[a]pyrenes were studied, its tumor causing activity was similar to dibenzo[*a*,*l*]pyrene. For example, when used to initiate tumors in mouse skin, 18 of 24 (75%) of mice treated with dibenzo[*a*,*h*]pyrene had tumors compared to 22 of 24 (92%) with dibenzo [*a*,*l*]pyrene. Controls showed skin tumors in 2 of 23 mice (9%).

15. Dibenzo[*a*,*i*]pyrene. Dibenzo[*a*,*i*] pyrene was assigned a PEF of 10 since, in the experiments by Cavalieri *et al.* (1989) in which all four dibenzo[*a*]pyrenes were studied, its tumor-causing activity was similar to dibenzo[a,l]pyrene. For example, when used to initiate tumors in mouse skin, 15 of 24 (63%) of mice treated with dibenzo[a,i]pyrene had tumors compared to 22 of 24 (92%) with dibenzo-[*a*,*l*]pyrene. Controls showed skin tumors in 2 of 23 mice (9%).

16. Dibenzo[*a*,*e*]pyrene. Dibenzo[*a*,*e*]pyrene was assigned a PEF of 1.0. Dibenzo[*a*,*e*]pyrene was the least potent of the four dibenzo[*a*]pyrenes studied by Cavalieri *et al.* (1989; 1991). In the experiments in which all four dibenzo[*a*]pyrenes were compared (Cavalieri *et al.*, 1989), its tumor-causing activity was approximately one-tenth to one-twentieth that of dibenzo[*a*,*l*]pyrene.

17. Indeno[1,2,3-*cd*]pyrene. Indeno[1,2,3-*cd*]pyrene was assigned a PEF of 0.1. Clement Associates (1988) used the mouse skin carcinogenesis data obtained by Habs *et al.* (1980) and by Hoffman and Wynder (1966) and the lung tumor data obtained by Deutsch-Wenzel *et al.* (1983) after intrapulmonary administration to estimate cancer potencies relative to BaP of 0.0302, 0.0292, and 0.246, respectively. These were averaged and rounded to obtain a PEF of 0.1.

18. 5-Methylchrysene. 5-Methylchrysene was assigned a PEF of 1.0. The activity of 5-methylchrysene relative to BaP has been studied by Hecht *et al.* (1976) using skin tumor initiation with phorbol ester (tetradecanoy1 phorbol acetate) promotion as well as skin tumor induction in mice. In the skin tumor induction test the tumorigenic activities of 5-methylchrysene and BaP were comparable enough so that a PEF of 1.0 was selected for 5 methylchrysene. Weekly application of 0.01% 5-methylchrysene led to skin carcinomas in 10 of 15 mice treated for up to 62 weeks, while 0.01% BaP led to skin carcinomas in 14 of 18 mice. The results for 0.005% of the 2 chemicals were 6 of 9 and 7 of 10, respectively.

19. 1-Nitropyrene. 1 Nitropyrene has been assigned a PEF of 0.1. In the Wislocki *et al.* (1986) report, in which lung tumors were induced in newborn mice, 1-nitropyrene (0.7 micromoles) was weakly carcinogenic in males (6/34 or 18% versus 4/45 or 9% in controls) and not carcinogenic in females (3/50 or 6% versus 2/34 or 6% in controls) relative to 0.56 micromoles BaP (13/37 or 35% in males versus 1/28 or 4% in control males and 13/27 or 48% in females versus 0/31 in control females). The relative potency was 0.348 in males and 0.076 in females. A PEF of 0.1 was assigned based on the experiment.

20. 4-Nitropyrene. 4-Nitropyrene was assigned a PEF of 0.1. Wislocki *et al.* (1986) compared the lung tumorigenicity of nitrated derivatives of pyrene to BaP in a newborn mouse assay. The background incidences were 4% in males and 0% in females. The administration of 2.8 micromoles of 4-nitropyrene gave a net incidence of 34% tumors in males and 31% in females, while 0.56 micromoles BaP gave 31% tumors in males and

48% in females. The potency of 4-nitropyrene relative to BaP was 0.23 in males and 0.12 in females. These were averaged and rounded to a PEF of 0.1.

21. 1,6-Dinitropyrene. 1,6-Dinitropyrene was assigned a PEF of 10. In the Wislocki *et al.* (1986) report, 1,6-dinitropyrene (0.2 micromoles) was weakly carcinogenic in inducing lung tumors in females (2/29 versus 0/31 in controls) and essentially not carcinogenic in males (1/25 versus 1/28 in controls) relative to 0.56 micromoles BaP (see 1-nitropyrene above for BaP data). The weak response combined with the low dose of 1,6-dinitropyrene (0.2 micromoles) relative to BaP (0.56 micromoles) resulted in a relative potency of 0.52 in females and 0.54 in males. In an intratracheal injection experiment (Takayama *et al.*, 1985) hamsters were given 26 weekly instillations of 0.5 mg BaP. All 10 males and 9 of 10 females developed respiratory tract tumors. A unit risk of 2.9×10^{-2} $(\mu g/m^3)^{-1}$ obtained from the female data which is 6.4 times the unit risks obtained from intratracheal studies using BaP and 26 times that using inhalation data. In a study by Iwagawa *et al.* (1989) using several doses of 1,6-dinitropyrene or BaP implanted directly into the lungs, a relative potency of 5.1 was obtained from the resulting lung cancer data. In light of the two experiments showing high relative potency and of 1,6-dinitropyrene's strong mutagenicity, a PEF of 10 appeared to be more appropriate than 1.0.

22. 1,8-Dinitropyrene. 1,8-Dinitropyrene was assigned a PEF of 1.0. In the Wislocki *et al.* (1986) report, 1,8-dinitropyrene (0.2 micromoles) was weakly carcinogenic in females (2/29 versus 0/31 in controls) and not carcinogenic in males (1/31 versus 1/28 in controls) relative to 0.56 micromoles BaP. However, due again to the low dose of 1,8-dinitropyrene chosen, the relative potency was 0.46 in females and 0.41 in males. In view of the high PEF of 1,6-dinitropyrene derived above and the very high mutagenicity of 1,8-dinitropyrene, the default PEF of 1.0 was assigned to 1,8-dinitropyrene until better *in vivo* data becomes available to derive a PEF.

23. 6-Nitrochrysene. 6-Nitrochrysene was assigned a PEF of 10. In the Wislocki *et al.* (1986) report, 0.7 micromoles of 6-nitrochrysene gave a net incidence of 76% lung tumors in males (28/33 versus 4/45 in controls) and 84% in females (36/40 versus 2/34 in controls). The potency of 6-nitrochrysene relative to BaP was 3.27 in males and 2.50 in females. In the newborn mouse assay of Busby *et al.* (1988), "(t)he ED50 for total lung tumors was 0.02 µmol for 6-NC and 0.2 µmol for BaP, thus showing a 10-fold higher potency for 6-NC compared with the 25-fold difference noted with tumor multiplicity." In a subsequent report (Busby *et al.*, 1989), 0.03 micromoles of 6-nitrochrysene caused lung adenomas and adenocarcinomas in 19/26 males and 13/22 females (versus controls of 13/91 in males and 7/101 in females) while 0.24 micromoles BaP caused lung adenomas and adenocarcinomas in 13/28 males and 19/27 females (against the same controls). The relative potencies were 17.51 for males and 6.17 for females. Based on the several experiments a PEF of 10 was selected.

24. 2-Nitrofluorene. 2-Nitrofluorene was assigned a PEF of 0.01. Miller *et al.* (1955) fed 2-nitrofluorene at a level of 1.62 mmol(215 mg)/kg diet to rats. This is estimated to give an animal dose of 33.1 mg/kg-day and a human equivalent dose of 4.7 mg/kg-day.

In one experiment 17 of 20 male rats (85%) developed forestomach tumors by 12 months. In another experiment 4 of 9 female rats (44%) developed mammary tumors by 10 months. These experiments yielded cancer potencies of 0.25 and 0.62 $(mg/kg\text{-}day)^{-1}$, approximately 0.02 and 0.05 that of BaP obtained in this risk assessment. The values of 0.02 and 0.05 were averaged and rounded down to obtain a PEF of 0.01.

25. Chrysene. Chrysene was assigned a PEF of 0.01. Clement Associates (1988) used the mouse skin carcinogenesis data obtained by Wynder and Hoffman (1959) to estimate a cancer potency relative to BaP of 0.0132. This was rounded to obtain a PEF of 0.01.

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