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Interim Decision Document
SWMU 14 - Fire Training Pit Area

Naval Station Roosevelt Roads

RCRA/HSWA Permit No. PR2170027203

Ceiba, Puerto Rico

Prepared For

Department of the Navy

Atlantic Division
Naval Facilities Engineering Command

Norfolk, Virginia

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LIST OF ACRONYMS AND ABBREVIATIONS

AF	Adherence Factor
AT	Averaging Time
ATSDR	Agency for Toxic Substances and Disease Registry
Baker	Baker Environmental, Inc.
bgs	below ground surface
BW	Body Weight
CDI	Chronic Daily Intake
CLEAN	Comprehensive Long-Term Environmental Action Navy Program
COPC	Chemical of Potential Concern
CsF	Cancer Slope Factor
CTO	Contract Task Order
DAD	Dermal Absorbed Dose
DAF	Dermal Absorption Factor
ED	Exposure Duration
EF	Exposure Frequency
EFH	Exposure Factor Handbook
USEPA	Environmental Protection Agency
EPC	Exposure Point Concentrations
ET	Exposure Time
HEAST	Health Effects Assessment Summary Tables
HHRAP	Human Health Risk Assessment Protocol
HHRA	Human Health Risk Assessment
HI	Hazard Index
HQ	Hazard Quotient
ICR	Increased Cancer Risk
IR	Ingestion Rate
IRH	Inhalation Rate
IRIS	Integrated Risk Information System
LANTDIV	Atlantic Division Naval Facilities Engineering Control
Kp	Dermal Permeability
MET	Meteorological Factor
mg/kg-d	milligrams per kilogram day
NCEA	National Center for Exposure Assessment
NSRR	Navy Station Roosevelt Roads
PAH	Polynuclear aromatic hydrocarbons
PCB	Polychlorinated biphenyl
PEF	Particulate Emission Factor
PID	Photoionization detector
PM^{10}	respirable particulates
ppm	parts per million

LIST OF ACRONYMS AND ABBREVIATIONS
(Continued)

RAGS	Risk Assessment Guidance for Superfund
RBC	Region III Risk Based Concentration
RCRA	Resource Conservation and Recovery Act
RfD	Reference Dose
RFI	RCRA Facility Investigation Phase I Report
SA	Surface Area
SVOCs	Semivolatile Organic Compounds
SWMU	Solid Waste Management Unit
TPH	Total Petroleum Hydrocarbons
UCL	Upper Confidence Limit
Fg/kg	micrograms per kilogram
USEPA	United States Environmental Protection Agency
VF	Volatilization Factor
VOCs	Volatile Organic Compounds
WOE	Weight of Evidence

EXECUTIVE SUMMARY

Introduction

This Interim Decision Document was prepared for Solid Waste Management Unit (SWMU) 14 - Fire Training Pit located at Naval Station Roosevelt Roads (NSRR), Cieba, Puerto Rico. The purpose of this document is to provide information to support the Navy's recommendation to postpone final site disposition (whether or not corrective action will be required) until the site is no longer utilized for training activities. The interim decision document generation was agreed to by both the EPA Region II and the Navy during a conference call on July 5, 2000.

The Navy recommends not performing additional site characterization of SWMU 14 at this time. Fire training activities are still conducted at this pit, and the Human Health Risk Assessment (HHRA) for SWMU 14 did not identify risks to the current commercial utility worker in excess of the United States Environmental Protection Agency (USEPA) acceptable range of 10^{-4} to 10^{-6}. Therefore, the Navy's recommendation will not expose current receptors to an unacceptable risk, and it will provide the Navy with the opportunity to fully evaluate this SWMU upon completion of the fire training operations.

This document was prepared by Baker Environmental, Inc. (Baker), for the Atlantic Division (LANTDIV), Naval Facilities Engineering Command. The project was performed under Contract Task Order (CTO) 099 under the LANTDIV Comprehensive Long-Term Environmental Action Navy (CLEAN) Program, Contract Number N62470-95-D-6007.

Site History

SWMU 14, the Fire Training Pit Area (approximately 40 feet in diameter) was operated by the Air Operations Department from the early 1960s through 1983. An estimated 120,000 gallons of waste solvents, fuels and oils were burned during fire training exercises. Additional items burned in this area included wood, trash, plastic, fuel filter elements, oily rags and other debris.

The fires were extinguished using aqueous film-forming foam and potassium bicarbonate (Purple K). Aerial photographs indicated drainage from the pit to the ditch located along the adjacent runway shoulder. Prior to 1983, two unlined fire training pits were used as described in

the Navy's 1984 initial assessment study. Visibly contaminated soils were removed from these two unlined pits during construction of the new pit in 1983.

Current Site Conditions

The fire training pit is a concrete structure, constructed below grade, with a concrete apron. The pit prevents seepage of contaminants to the soils beneath the fire training area. A drainage system encircling the apron intercepts any overtopping, which is directed to an oil/water separator.

Current Site Usage

Currently, the fire training pit is utilized for training purposes. The training consists of Navy personnel simulating an aircraft crash by igniting pieces of aircraft with two to three (55 gallon containers) of JP-5 fuel per training session. The facility is currently undergoing repair work. When it is utilized again for fire training activities, Tech-flame may be used to ignite the fires. Personnel are required to use water (only) to extinguish the training fires developed at this pit. On average, fire training activities last from three to four hours and are conducted two to four times a month.

Previous Investigations

Previous assessments conducted at SWMU 14 include: the 1995 RFI Work Plan, the 1996 RCRA Facility Investigation (RFI), the Draft HHRA Report for SWMU 14, comment letters between the USEPA and the Navy, and issues discussed during the conference call on October 10, 2000.

As part of the 1996 RFI, a total of five surface soil samples (14SS04 through 14SS08) were collected at the locations which exhibited the highest PID readings which ranged from 21.1 parts per million (ppm) to 79.2 ppm. Fourteen SVOCs, twelve being Polynuclear Aromatic Hydrocarbons (PAHs), one congener of PCB (Aroclor-1260), and TPH diesel and gasoline range organics were detected in surface soil samples.

Human Health Risk Assessment

This Baseline Human Health Risk Assessment (HHRA) was designed to support the Interim Decision Document for SWMU 14 located at the Naval Station Roosevelt Roads (NSRR), Cieba, Puerto Rico. The objective of this HHRA is to assess the human health risks associated with exposures to surface soil contamination identified in the RCRA Facility Investigation Report for Phase I Investigations at Operable Units 1, 6, and 7 prepared by Baker Environmental, Inc. (Baker) for the Atlantic Division (LANTDIV), Naval Facilities Engineering Command (Baker 1996).

This HHRA was conducted in accordance with United States Environmental Protection Agency (USEPA) Regulations and is consistent with the following risk assessment guidance documents:

- Risk Assessment Guidance for Superfund (RAGS) Volume I Human Health Evaluation Manual Part A. USEPA 1989.
- RAGS for Superfund Volume I Human Health Evaluation Manual Part B. USEPA 1991a.
- Supplemental Guidance to RAGS Standard Default Exposure Factors OSWER Directive 9285.6-03. USEPA 1991b.
- Supplemental Guidance to RAGS: Calculating the Concentration Term. USEPA 1992.
- Exposure Factors Handbook Volume 1 and II. USEPA 1997a.
- Assessing Dermal Exposures to Soil. USEPA 1995.
- Region III Risk Based Concentrations (RBC). USEPA 2000a.
- Integrated Risk Information System. USEPA 2000b.

This HHRA is organized in the following manner (National Academy of Sciences National Research Council, 1983):

Hazard Identification - This section provides a summary of the analytical data for surface soil, and identifies Chemicals of Potential Concern (COPC).

Dose-Response Assessment - In this section reference doses, and slope factors for each COPC are presented, and methods for assessing cancer and noncancer dose-response relationships are discussed.

Exposure Assessment - This section identifies potentially exposed populations, exposure pathways, and exposure parameters used for estimating site-specific risk.

Risk Characterization - This section presents the estimated risks for each scenario, and provides a qualitative uncertainty analysis.

For this HHRA, COPCs were identified by comparing the chemicals in surface soil samples to Residential USEPA Region III Risk Based Concentrations (RBCs) (USEPA 2000a). RBCs are derived using conservative USEPA promulgated default values and the most recent toxicological criteria available. RBCs for potentially carcinogenic and noncarcinogenic chemicals are individually derived based on a target incremental lifetime cancer risk (ICR) of 1×10^{-6} and a target hazard quotient (HQ) of 1.0, respectively. For potential carcinogens, the toxicity criteria applicable to the derivation of the RBCs are chronic oral cancer slope factors; for noncarcinogens they are oral reference doses.

The following chemicals exceeded the residential RBCs values, and were therefore retained as COPCs for further analysis: benzo(a)anthracene, benzo(b)fluoranthene, benzo(a)pyrene, dibenz(a,h)anthracene and indeno(1,2,3-c,d)pyrene.

The results of the HHRA did not indicate an unacceptable cancer risk for any of the exposure scenarios or pathways. Current land use scenarios that were evaluated include commercial/utility worker, and trespasser. Future land use scenarios evaluated include commercial/utility worker, trespasser, military residential (adult and child), and construction worker. Each scenario evaluated the risk associated with ingestion of contaminants in surface soil, inhalation of particulates from surface soil, and dermal contact with surface soil. Based on the results of this HHRA it is not likely that the COPCs detected in surface soils at SWMU 14 will pose a significant health risk to current or future receptors.

Conclusions and Recommendations

There are currently engineering controls in place to prevent subsurface and groundwater exposure to activities conducted at the fire training pit. The engineering controls include a concrete pad with a concrete apron, and an oil and water separator which will be maintained as long as the fire training pit is utilized. SWMU 14 will not be utilized for any other function except for fire

training activities. Once fire training operations cease, additional site characterization of the site will be conducted. This characterization will be determined once the site is no longer in operation. If it is determined that an unacceptable risk is present, the area would be remediated as appropriate. Therefore, it is recommended that additional site characterization and a final decision of whether or not corrective action is necessary should be determined once the usage of the area is terminated.

1.0 INTRODUCTION

This Interim Decision Document was prepared for Solid Waste Management Unit (SWMU) 14 - Fire Training Pit located at Naval Station Roosevelt Roads (NSRR), Cieba, Puerto Rico. The purpose of this document is to provide information to support the recommendation to postpone final site disposition (whether or not corrective action will be required) until the site is no longer utilized for training activities.

The Navy recommends not performing additional site characterization of SWMU 14 at this time. Fire training activities are still conducted at this pit, and the Human Health Risk Assessment (HHRA) for SWMU 14 did not identify risks to the current commercial utility worker in excess of the United States Environmental Protection Agency (USEPA) Acceptable Range of 10^{-4} to 10^{-6}. Therefore, the Navy's recommendation will not expose current receptors to an unacceptable risk, and it will provide the Navy with the opportunity to fully evaluate this SWMU upon completion of the fire training operations.

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1.1 Site History

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The fires were extinguished using aqueous film-forming foam and potassium bicarbonate (Purple K). Aerial photographs indicated drainage from the pit to the ditch located along the adjacent runway shoulder. Prior to 1983, two unlined fire training pits were used as described in the Navy's 1984 initial assessment study. Visibly contaminated soils were removed from these two unlined pits during construction of the new pit in 1983.

1.2 Current Site Usage

Currently, the fire training pit is utilized for training purposes. The training consists of Navy personnel simulating an aircraft crash by igniting pieces of aircraft with two to three (55 gallon containers) of JP-5 fuel per training session. The facility is currently undergoing repair work. When it is utilized again for fire training activities, Tech-flame may be used to ignite the fires. Personnel are required to use water (only) to extinguish the training fires developed at this pit. On average, fire training activities last from three to four hours and are conducted two to four times a month.

1.3 Current Site Conditions

The fire training pit is a concrete structure, constructed below grade, with a concrete apron. The pit prevents seepage of contaminants to the soils beneath the fire training area. A drainage system encircling the apron intercepts any overtopping, which is directed to an oil/water separator.

The remainder of this section will review previous assessments conducted at SWMU 14 including: the 1995 RFI Work Plan, the 1996 RCRA Facility Investigation (RFI), the Draft HHRA Report for SWMU 14, comment letters between the USEPA and the Navy, and issues discussed during the conference call on October 10, 2000.

1.4 RCRA Permit and 1995 Work Plan

The Resource Conservation and Recovery Act (RCRA) permit for NSRR indicated that SWMU 14 would require a Phase I RFI. Therefore, it was included in the 1995 RFI Work Plan (Baker, 1995). The Work Plan specified that a limited soil gas survey would be conducted and surface soil samples would be collected.

1.5 Phase I RFI Investigation

SWMU 14 was included in the Phase I RFI investigation for OU 1, 6 and 7 in 1996. Five surface soil samples were collected at SWMU 14 for the investigation. Analysis performed and results of the Phase I RFI are discussed in Section 2.0. It was recommended in the original document that no further action was warranted at SWMU 14. The USEPA had agreed with the initial request for

No Further Action. However, during the development of the No Further Action Required documentation it was determined that the USEPA approved original RFI document (Baker, 1996) contained omissions associated with the SWMU 14 data set. A HHRA was not performed in the original RFI since it was believed that none of the constituents analyzed for were detected in the surface soil samples obtained from SWMU 14. The Navy identified the discrepancy and brought it to the attention of the USEPA.

1.6 Human Health Risk Assessment

The Navy voluntarily developed the HHRA for SWMU 14 following the recognition that data was inadvertently omitted in the presentation of SWMU 14 analytical data. On February 4, 2000, the Navy submitted a baseline HHRA to the USEPA to evaluate constituents detected in surface soil samples collected during the Phase I RFI in 1996.

An exposure assessment was conducted to determine human receptors, exposure pathways and exposure assumptions. Current land use scenarios that were evaluated included commercial/utility worker, and trespasser. Future land use scenarios evaluated included commercial/utility worker, trespasser, military residential (adult and child), and construction worker. Each scenario evaluated the risk associated with ingestion of contaminants in surface soil, inhalation of particulates from surface soil, and dermal contact with surface soil.

The results of the HHRA did not indicate an unacceptable cancer risk for any of the exposure scenarios or pathways (i.e. ingestion, inhalation, dermal). Based on results of the HHRA it is not likely that the Chemicals of Potential Concern (COPCs) detected in surface soils at SWMU 14 would pose a significant health risk to current or future receptors.

1.7 Comment Letters Between the USEPA and the Navy

The Draft Baseline HHRA Report for SWMU 14 was submitted to the USEPA on February 4, 2000. The USEPA's comments on the HHRA were reflected in a letter to the Navy on July 5, 2000. The USEPA requested that the Navy provide either a supplemental site characterization work plan to investigate subsurface soil and groundwater, or to submit a revised HHRA. The Navy responded with a letter to address the USEPA comments on August 22, 2000. A conference call was scheduled between the Navy and the USEPA to discuss the Draft HHRA and other issues related to NSRR with respect to the EPA current letter dated July 5, 2000.

1.8 Conference Call

A conference call was held on October 10, 2000 between the USEPA and the Navy to discuss comments from the July 5, 2000 USEPA comment letter and the August 22, 2000 Navy response letter. During the conference call the USEPA and the Navy agreed that this Interim Decision Document would be prepared to support the recommendation that no additional site characterization should be completed at SWMU 14 until the site is no longer utilized. As discussed during the conference call on October 10, 2000, both parties agreed to use a value of 100 mg/day to represent the incidental soil IR for an adult construction worker. The Draft HHRA was revised to incorporate the IR value of 100 mg/day in the calculations as well as the Navy responses (see section 3.0).

1.9 Report Organization

Section 1.0 of this document includes the introduction, site history, current site usage and conditions, the RCRA Permit and 1995 Work Plan, the Phase I RFI Investigation, and the conference call. Section 2.0 discusses the field work, sample analysis and results from the Phase I RFI. Section 3.0 presents the HHRA evaluating the human health risk associated with exposures to contaminants in surface soil and incorporating the USEPA comments on the Draft HHRA. The conclusions and recommendations for this report are presented in Section 4.0. Section 5.0 provides references used in this report.

2.0 1996 RCRA FACILITY INVESTIGATION

The field investigation for the initial RFI was performed in March 1996 in accordance with the EPA approved RFI Work Plan (Baker, 1995) and resulted in the development of the Draft RCRA Facility Investigation Report for Phase I Investigations at Operable Units 1,6, and 7. The remainder of this section discusses data collection, analysis performed and analytical results of the 1996 RFI investigation.

2.1 Data Collection and Analysis

A limited soil gas survey was conducted along the immediate perimeter of the Fire Training Pit. A total of 50 sampling nodes were spaced along two concentric rings around the pit. The first ring was established three feet from the edge of the concrete apron while the second ring was established at a distance of 10 feet. Each ring contained 25 sampling nodes (Baker 1996).

Each sampling node (generated by driving a metal pin one to two feet into the ground) was screened by inserting the tip of the photoionization detector (PID) and recording the results in a field log book. A total of five surface soil samples (14SS04 through 14SS08) as depicted in Figure 2-1 were collected at the locations which exhibited the highest PID reading which ranged from 21.1 parts per million (ppm) to 79.2 ppm.

Surface soil samples were collected using decontaminated stainless steel spoons. All surface soil samples were collected to a depth of one foot below ground surface (bgs). Prior to sampling all vegetation was removed from the location. The soil samples collected for volatile organic compounds (VOCs) was placed directly into the laboratory prepared sample containers without homogenizing to prevent volatilization. The soil samples collected for semivolatile organic compounds (SVOCs), polychlorinated biphenyls (PCBs) and total petroleum hydrocarbons (TPH) analysis was placed in disposable aluminum pans and homogenized before being placed in the associated laboratory prepared sample and placed in associated containers. All samples were placed in coolers on ice and maintained under strict chain-of-custody until delivered to the laboratory.

2.2 Analytical Results

The analytical results for all constituents tested are presented in Appendix A. Fourteen SVOCs, twelve of which being polynuclear aromatic hydrocarbons (PAHs), one congener of PCB (Aroclor-1260), and TPH diesel and gasoline range organics were detected in surface soil samples. Table 2-1 presents those constituents detected along with a comparison of the maximum concentrations of chemicals detected to USEPA Region III Residential Risk Based Concentrations (RBC) (USEPA, 2000a). All constituents that resulted in exceedances above the residential RBCs were in two of the five samples, 14SS06 and 14SS07. Benz(a)anthracene was detected in three samples ranging from 45J micrograms/kilogram (µg/kg) to 3,400 µg/kg. The residential RBC value was exceeded in sample 14SS07 at 3,400 µg/kg. Benzo(b)flouranthene was detected in samples 14SS06 and 14SS07 at values of 2,800 µg/kg and 7,600 µg/kg respectively, both exceeding residential RBC values. Benzo(a)pyrene was detected in three samples and exceeded residential RBC values in two samples (14SS06 and 14SS07) at 1,800 µg/kg and 5,000 µg/kg respectively. Dibenz(a,h)anthracene exceeded residential RBCs in samples 14SS06 and 14SS07 at 210J µg/kg and 920 µg/kg, respectively. Indeno(1,2,3-c,d)pyrene was detected in three samples and exceeded residential RBC levels in two surface soil samples, 14SS06 and 14SS07 at 1,300 µg/kg and 3,800 µg/kg respectively.

Other SVOCs that were detected but did not exceed RBC values include: Bis(2-ethylhexyl)phthalate, Diethylphthalate, Anthracene, Benzo(k)flouranthene, Benzo(g,h,i)perylene, Chrysene, Flouranthene, Phenanthrene and Pyrene as presented in Table 2-1.

Aroclor-1260 was detected in all 5 samples ranging from 6J to 28 µg/kg, none of which exceeded the residential RBC criteria of 319 µg/kg.

TPH Diesel Range Organics were detected in four samples ranging from 120 milligrams/ kilogram (mg/kg) to 560 mg/kg and TPH Gasoline Range Organics were detected in all five samples ranging from 0.032 mg/kg to 3.8 mg/kg.

3.0 HUMAN HEALTH RISK ASSESSMENT

This Baseline Human Health Risk Assessment (HHRA) was designed to support the Interim Decision Document for SWMU 14 located at the Naval Station Roosevelt Roads (NSRR), Cieba, Puerto Rico. The objective of this HHRA is to assess the human health risks associated with exposures to surface soil contamination identified in the RCRA Facility Investigation Report for Phase I Investigations at Operable Units 1, 6, and 7 prepared by Baker Environmental, Inc. (Baker) for the Atlantic Division (LANTDIV), Naval Facilities Engineering Command (Baker 1996).

This HHRA was conducted in accordance with United States Environmental Protection Agency (USEPA) Regulations and is consistent with the following risk assessment guidance documents:

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- Supplemental Guidance to RAGS Standard Default Exposure Factors OSWER Directive 9285.6-03. USEPA 1991b.
- Supplemental Guidance to RAGS: Calculating the Concentration Term. USEPA 1992.
- Exposure Factors Handbook Volume 1 and II. USEPA 1997a.
- Assessing Dermal Exposures from Soil. USEPA 1995.
- Region III Risk Based Concentrations (RBC). USEPA 2000a.
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This HHRA is organized in the following manner (National Academy of Sciences National Research Council, 1983):

Hazard Identification - This section provides a summary of the analytical data for surface soil, and identifies Chemicals of Potential Concern (COPC).

Dose-Response Assessment - In this section reference doses, and slope factors for each COPC are presented, and methods for assessing cancer and noncancer dose-response relationships are discussed.

Exposure Assessment - This section identifies potentially exposed populations, exposure pathways, and exposure parameters used for estimating site-specific risk.

Risk Characterization - This section presents the estimated risks for each scenario, and provides a qualitative uncertainty analysis.

3.1 Hazard Identification

This section provides a summary of the analytical data for surface soil that was collected in the RFI investigation, and identifies those chemicals that may pose the most significant risk associated with SWMU 14.

Data Collection

As discussed in Section 2.1, a total of five surface soil samples were collected (14SS04 through 14SS08) and were analyzed for VOCs, SVOCs, PCBs, and TPH (gas and diesel fractions) (Baker 1996). Results of these analyses are located in Appendix A of this interim decision document HRA. Fourteen SVOCs, twelve of which being PAHS, Aroclor-1260, TPH Diesel and Gasoline Range Organics were detected in surface soil.

COPC Identification

USEPA Region III suggests that; "The baseline risk assessment process can be made more efficient by focusing on dominant contaminants and routes of exposure at the earliest feasible stage. The mechanisms recommended for this are (1) a re-ordering of the process of eliminating contaminants and routes of exposure, and (2) use of a risk-based concentration screen. Appropriately used, this process can dramatically reduce the effort of risk assessment, while not changing the result significantly (USEPA 1993)." Therefore, for this HHRA a risk based concentration screen was used to identify COPCs.

COPCs are those constituents having the greatest potential to affect human health and the environment. They are selected by comparing the maximum constituent concentrations detected in the environmental samples to regulatory criteria. Chemicals exceeding regulatory criteria are retained as COPCs for further evaluation; chemicals detected at concentrations below these

criteria are not evaluated unless other circumstances (frequency of exposure or documented usage) warrant the reinclusion and further evaluation of chemicals selected as COPCs.

For this HHRA, COPCs were identified by comparing the chemicals in surface soil samples to Residential USEPA Region III Risk Based Concentrations (RBCs) (USEPA 2000a). RBCs are derived using conservative USEPA promulgated default values and the most recent toxicological criteria available. RBCs for potentially carcinogenic and noncarcinogenic chemicals are individually derived based on a target incremental lifetime cancer risk (ICR) of 1×10^{-6} and a target hazard quotient (HQ) of 1.0, respectively. For potential carcinogens, the toxicity criteria applicable to the derivation of the RBCs are chronic oral cancer slope factors; for noncarcinogens they are oral reference doses.

Fourteen SVOCs, Aroclor-1260, and TPH Diesel and Gasoline Range Organics were detected in surface soil. The following chemicals exceeded the residential RBCs values, and were therefore retained as COPCs for further analysis:

- benzo(a)anthracene
- benzo(b)fluoranthene
- benzo(a)pyrene
- dibenz(a,h)anthracene
- indeno(1,2,3-c,d)pyrene

Table 2-1 presents the results of this sampling event and the identification of COPCs. Appropriate toxicological criteria for TPH does not exist, and therefore was not retained for further evaluation in this HHRA.

3.2 Dose-Response Assessment

In this section the relationship between a dose of a chemical agent and frequency of an adverse effect in an exposed population was characterized. Dose-response information and toxicity criteria were obtained from the Integrated Risk Information System (IRIS) (USEPA, 2000b) and the Health Effects Summary Tables (HEAST) (USEPA 1997b).

3.2.1 Noncarcinogenic Dose-Response Assessment

Potential noncarcinogenic health effects associated with exposures to COPCs identified in Section 3.1 are unknown (USEPA, 2000b). Noncarcinogenic effects for these COPCs have not been identified in IRIS or HEAST. Therefore, only the carcinogenic effects associated with exposure to the COPCs were addressed in this HHRA.

3.2.2 Carcinogenic Dose –Response Assessment

The potential health effects associated with exposures to carcinogenic COPCs were evaluated using cancer slope factors (CsF) established by USEPA (2000b). The CsF is an estimate of an upper-bound probability of an individual developing cancer as a result of a lifetime of exposure to a particular level or dose of a potential carcinogen. The cancer slope factor is expressed as milligram per kilogram-day $(mg/kg\text{-}d)^{-1}$. CsFs for oral and inhalation pathways have been developed. The CsFs developed by USEPA for the COPCs and the USEPA Weight of Evidence (WOE) Classification for each COPC are presented in Table 3-1 of this HHRA. The WOE classifications are explained below:

Group A	Human Carcinogen (sufficient evidence of carcinogenicity in humans)
Group B1	Probable Human Carcinogen (limited evidence of carcinogenicity in humans)
Group B2	Probable Human Carcinogen (sufficient evidence of carcinogenicity in animals with inadequate or lack of evidence in humans)
Group C	Possible Human Carcinogen (limited evidence of carcinogenicity in animal or lack of human data)
Group D	Not Classifiable as to Human Carcinogenicity (inadequate or no evidence)
Group E	Evidence of Noncarcinogenicity for Humans (no evidence of carcinogenicity in adequate studies)

According to USEPA guidance (1989), only those chemicals with Group A, B (B1 or B2), or C rankings need to be addressed for possible carcinogenic effects. All COPCs identified in Section 3.1 are considered B2 carcinogens, and were therefore retained for further analysis. Toxicological profiles per each COPC are located in Appendix C.

The oral CsFs were applied to the dermal assessment in this evaluation. RAGs Part A, Appendix A recommends that the oral CsF be adjusted by an oral absorption efficiency to account for the

difference in toxicity between an administered dose and an absorbed dose (USEPA 1989). An appropriate oral absorption efficiency for PAHs could not be identified. Therefore, a default value of 1.0 was applied for this HHRA.

3.3 Exposure Assessment

The Exposure Assessment estimates the magnitude of actual and/or potential human exposure, the frequency and duration of those exposures, and the pathways (i.e., inhalation, ingestion, and dermal contact) by which people are potentially exposed (National Academy of Sciences National Research Council, 1983). In order for an exposure to occur, a complete pathway must exist with the following conditions:

- a source and mechanism of chemical release into the environment
- an environmental transport medium
- a point of potential human contact with the medium; and
- a human exposure route at the contact point.

Presented in Section 3.3.1 and 3.3.2 and Figure 3-1 of this HHRA are the potential exposure pathways and scenarios, and the exposure parameters that were used to estimate human health risk associated with these pathways.

3.3.1 Conceptual Site Exposure Model

Current and potential future exposure scenarios are summarized in Table 3-2 of this HHRA. Current potential exposures to COPCs in soil at SWMU 14 exist for commercial/utility workers, and trespassers. Due to limited access to Roosevelt Roads, it is unlikely that trespassers would be able to enter the site; regardless it was evaluated. Future exposures to surface soil on this site may consist of military residents, construction workers, trespassers, and commercial/utility workers.

The current/future potential land use scenarios evaluated adult exposures. In addition a residential child between the ages of 1-6 years old, and a youth trespasser between the ages of 7-17 years old was evaluated. Exposure routes (i.e. ingestion, dermal contact, and inhalation) for each exposure scenario are summarized in Table 3-2.

3.3.2 Exposure Parameters

The parameters used to quantify potential chemical uptake from each complete exposure pathway can be found in Table 3-3. Each parameter was selected from values provided by USEPA guidance documents (1989, 1991a, 1991b, 1995, 1996, 1997a). The USEPA 1997 Exposure Factors Handbook (EFH) serves as the primary source for the exposure parameters identified in this HHRA.

Exposure Duration

An exposure duration (ED) of 4 years for both the future military residential adult and child was used for this HHRA (Rivera, 1999). The future construction worker ED was 1 year based on professional judgement. The current commercial/utility worker ED was 25 years (USEPA, 1991b). Current/Future trespasser EDs were set at 30 years for adults and 9 years for youth (USEPA, 1991b).

Exposure Frequency

An exposure frequency (EF) of 350 days was used for the future military residential adult and child (USEPA 1989). 250 days per year was used for the commercial utility worker, and 180 days was used for the construction worker (USEPA, 1991b). An EF of 52 days per year was used for the adult and youth trespasser. This EF assumes that some type of trespassing activity occurs once a week (professional judgement).

Exposure Time

The future construction worker and current commercial/utility worker exposure time (ET) was 8 hours; this is assuming that both will be working outdoors the entire day (professional judgement). The EFH recommends that 2 hours per day be used for time spent outdoors at one's residence, therefore 2 hours per day was used for exposures to surface soil for the future military residential scenario (USEPA 1997a). An ET of 2 hours per day was likewise used for a trespasser. The base is secured and not accessible to trespassers, therefore assuming an ET of 2 hours per day is a conservative assumption.

Averaging Time

An averaging time (AT) of 25,550 days for exposures to carcinogenic chemicals in the environment was used for all scenarios (USEPA, 1989). Non-carcinogens were not evaluated in this HHRA.

Body Weight

The USEPA standard body weight (BW) of 15 kg for children and 70 kg for adults was used in this HHRA (USEPA, 1991a). A body weight of 45 kg was used for the youth trespasser (USEPA, 1997a). This BW represents the mean body weight of both males and females between the ages of 7 and 17 years.

Ingestion Rate

A soil ingestion rate (IR) of 50 mg/day for adult trespassers, adult commercial/utility workers, and youth trespassers; and 200 mg/day for children was used this HHRA (USEPA 1997a). For the construction worker and adult resident, Region II USEPA recommends using 100 mg/day (Personal communication with USEPA Region II Tim Gordon October 2000).

Dermal Absorption Factor (DAF)

For SVOCs (including PAHs) a DAF of 10% was used in accordance with USEPA Assessing Dermal Exposures to Soil (USEPA 1995).

Surface Area (SA)

A SA of 5,000 cm^2 for adult residents, commercial/utility workers, construction workers, and trespassers for contact with soil was used (USEPA 1997a). The EFH does not list body specific SAs for children, only total. Therefore, for military residential children it is assumed that 25% of the total body surface area will be exposed to surface soils. The total body surface area for a male child between the ages of 2-6 years old (50% percentile) is 7,000 cm^2, thus a SA of 1,700 cm^2 was used for children's exposure to surface soil (professional judgement). Similarly, the youth (ages 7-17 years old) trespasser total SA is 12,900 cm^2; 25% is equal to 3,200 cm^2 (USEPA 1997a).

Adherence Factor

The 1999 Dermal Risk Assessment Guidance recommends an adherence factor (AF) of 0.2 for the adult commercial/utility worker, and child, and 0.07 for the adult resident (USEPA 1999). These values were verified by USEPA Toxicologist Stanford Smucker in a recent correspondence with Melissa Fredrick at Baker Environmental (Dr. Smucker 415-744-2311). An AF of 0.43 mg/cm^2 was used for the construction worker (USEPA, 1997a). The 0.43 mg/cm^2 is based on the Kissel et al 1996 work as cited in the EFH (USEPA, 1997a).

Inhalation Rate

An inhalation rate (IR_h) of 1.27 m^3/hr was used for the military adult residents, adult and youth trespassers, and commercial/utility workers. This value represents long-term inhalation rates for a male (15.2 m^3/day) for a 12 hour day (USEPA, 1997a). Similarly an IR_h of 0.69 m^3/hr was derived for a child (USEPA 1997a). An IR_h for construction workers assumes an average 4 hours of moderate work per day(1.5 m^3/hr*4 hours) and 4 hours of heavy work per day (2.5m^3/hr*4 hours/day) which is 16m^3/8-hour day or 2 m^3/hr.

Particulate Emission Factor

A particulate emission factor (PEF) of 1.32 x 10^9 m^3/kg was used in this HHRA (USEPA 1996). The PEF relates the contaminant concentration in soil with the concentration of respirable particulates (PM_{10}) in the air due to fugitive dust emissions (USEPA, 1996). This value is a default PEF cited in USEPA Soil Screening Level Guidance Document, and assumes 50% vegetation and a mean annual wind speed of 4.69 m/s (USEPA 1996).

3.3.3 Quantification of Exposure

Calculating the EPC

Exposure Point Concentrations (EPC) are used to estimate chronic daily intakes (CDIs) and dermally absorbed doses (DADs) for each medium and are representative of the types of potential exposures encountered by each receptor. Exposure can occur discretely or at a number of sampling locations depending on the type of scenario considered for a given receptor. USEPA

risk assessment guidance (USEPA, 1989) recommends that an upper bound estimate of the arithmetic mean concentration be used to calculate the EPC. "Because of the uncertainty associated with estimating the true average concentration at a site, the 95 percent upper confidence limit (UCL) of the arithmetic mean should be used for this variable. The 95 percent UCL provides reasonable confidence that the true site average will not be underestimated (USEPA 1992)".

The EPC was calculated using the USEPA 1992 Calculating the Concentration Term Guidance and was based on its distribution. The Shapiro Wilkes test for samples less than 50 was used to determine if the data is distributed normally or lognormally. Table 3-4 presents the results of this test. Each sample set for each COPC was identified as "lognormal", therefore the appropriate method as provided in the 1992 USEPA Guidance was used to calculate the EPC based on a lognormal distribution. However, the lognormal 95% UCL was greater than the maximum detected concentration. In cases such as this, USEPA recommends using the maximum concentration as an EPC (USEPA, 1992). Therefore in this HHRA the maximum concentration for each COPC was used as the EPC.

Calculating the CDI

The following equations were derived from USEPA Risk Assessment Guidelines Part A and B (USEPA 1989, USEPA 1995, USEPA 1991b), and were used for this HHRA.

Ingestion of Chemicals in Soil:

CDI= Cs*CF*IR*EF*ED/BW*AT

CDI	Chronic daily intake (mg/kg-d)
Cs	Concentration of chemical in soil (mg/kg)
CF	Conversion Factor (10^{-6} kg/mg)
IR	Ingestion Rate soil (mg/d)
EF	Exposure Frequency (d/y)
ED	Exposure Duration (y)
BW	Body weight (kg)
AT	Averaging time (d)

Dermal Contact with Chemicals in Soil:

DAD= Cs*CF*SA*AF*EF*ED/BW*AT

DAD	Dermally Absorbed Dose (mg/kg-d)
Cs	Concentration of chemical in soil (mg/kg)
CF	Conversion Factor (10^{-6} kg/mg)
SA	Skin Surface Area (cm^2/d)
AF	Adherence Factor (mg/cm^2)
EF	Exposure Frequency (d/y)
ED	Exposure Duration (years)
BW	Body weight (kg)
AT	Averaging time (d)

Inhalation of Particulates:

CDI= Cs*ED*EF*ET*IR_h/BW*AT*PEF

CDI	Chronic daily intake (mg/kg-d)
Cs	Concentration of chemical in soil (mg/kg)
IR_h	Respiration Rate (m^3/hr)
EF	Exposure Frequency (d/y)
ED	Exposure Duration (y)
ET	Exposure Time (hrs/d)
PEF	Particulate Emission Factor (m^3/kg)
BW	Body weight (kg)
AT	Averaging time (d)

3.4 Risk Characterization

This section provides numerical estimates of human health and environmental risks posed by the presence of the COPCs at SWMU 14.

3.4.1 Quantification and Characterization of Carcinogenic Risks

Quantitative risk calculations for potentially carcinogenic COPCs estimate inferentially (versus probabilistically) the potential increased cancer risk (ICR) for an individual in a specified population. This unit of risk refers to a potential cancer risk that is above the background cancer risk in unexposed individuals. For example, an ICR of 1 x 10^{-6} indicates that an exposed individual has an increased probability of one in one million of developing cancer subsequent to exposure, over the course of his lifetime. The following equation was used in estimating the ICR:

ICR = Cancer Slope Factor (CsF) X Chronic Daily Intake (CDI)

The CsF is expressed as $(mg/kg\text{-}d)^{-1}$ and the chronic daily intake (CDI) is expressed as mg/kg-d. The aforementioned equation was derived assuming that cancer is a non-threshold process and that the potential excess risk level is proportional to the cumulative intake over a lifetime.

For quantitative estimation of risk, it is assumed that cancer risks from various exposure routes are additive. Estimated ICR values will be compared to 1×10^{-6} to 1×10^{-4} which represents the target risk range of ICR values considered by the USEPA to represent an acceptable (i.e., de minimus) risk (USEPA, 1990).

3.4.2 Quantification and Characterization of Noncarcinogenic Risks

Potential noncarcinogenic health effects associated with exposures to noncarcinogenic COPCs were not identified in IRIS or HEAST. Therefore only the carcinogenic effects associated with exposure to the COPCs were addressed in this HHRA. Calculation of the CDIs, DADs, and carcinogenic risk estimates are presented in Appendix B of this interim decision document and are summarized in Tables 3-5 through 3-8.

3.4.3 Results of Risk Characterization

The carcinogenic risk for future military adult residents was 4.2×10^{-6} for ingestion of surface soil, 1.5×10^{-6} for dermal contact with surface soil, and 2.3×10^{-11} for inhalation of particulates as stated in Table 3-5. A total risk across all three pathways was 5.7×10^{-6}, which falls within USEPA acceptable risk range of 1×10^{-4} and 1×10^{-6}.

The carcinogenic risk for future military child residents was 3.9×10^{-5} for ingestion of surface soil, 6.7×10^{-6} for dermal contact with surface soil, and 5.9×10^{-11} for inhalation of particulates as stated in Table 3-5. A total risk across all three pathways was 4.6×10^{-5}, which falls within USEPA acceptable risk range of 1×10^{-4} and 1×10^{-6}.

The carcinogenic risk for future construction worker was 5.4×10^{-7} for ingestion of surface soil, 5.4×10^{-8} for dermal contact with surface soil, and 1.9×10^{-11} for inhalation of particulates as stated in Table 3-6. A total risk across all three pathways was 6.0×10^{-7}, which is less than the USEPA acceptable risk range of 1×10^{-4} and 1×10^{-6}.

The carcinogenic risk for current adult trespasser was 2.4×10^{-6} for ingestion of surface soil, 4.7×10^{-6} for dermal contact with surface soil, and 2.6×10^{-11} for inhalation of particulates as stated in Table 3-7. A total risk across all three pathways was 7.1×10^{-6}, which falls within USEPA acceptable risk range of 1×10^{-4} and 1×10^{-6}.

The carcinogenic risk for current youth trespasser was 1.1×10^{-6} for ingestion of surface soil, 1.4×10^{-6} for dermal contact with surface soil, and 1.2×10^{-11} for inhalation of particulates as stated in Table 3-7. A total risk across all three pathways was 2.5×10^{-6}, which falls within USEPA acceptable risk range of 1×10^{-4} and 1×10^{-6}.

The carcinogenic risk for current/future commercial/utility workers was 9.4×10^{-6} for ingestion of surface soil, 1.9×10^{-5} for dermal contact with surface soil, and 4.2×10^{-10} for inhalation of particulates as stated in Table 3-8. A total risk across all pathways of exposure was 2.8×10^{-5}, which falls within USEPA acceptable risk range of 1×10^{-4} and 1×10^{-6}.

3.4.4 Uncertainty Analysis

Uncertainties are encountered throughout the process of performing a risk assessment. This section discusses the sources of uncertainty inherent in the following elements of the baseline HHRA prepared for SWMU 14.

- Sampling and analysis
- Selection of COPCs
- Exposure assessment
- Toxicological assessment
- Risk characterization

Uncertainties associated with this HHRA are discussed in the following paragraphs.

<u>Sampling and Analysis</u>

The development of a risk assessment depends on the reliability of, and uncertainties associated with, the analytical data available to the risk assessor. These, in turn, are dependent on the operating procedures and techniques applied to the collection of environmental samples in the field and their subsequent analyses in the laboratory. To minimize the uncertainties associated with sampling and analysis at SWMU 14, USEPA approved sampling and analytical methods

were employed. Data were generated following RCRA methods of analysis for organics and inorganics, and were validated in accordance with USEPA Region II procedures. Samples were taken from locations specified in the USEPA approved Work Plan along with the necessary QA/QC samples.

Analytical data are limited by the precision and accuracy of the methods of analysis which are reflected by the Relative Percent Difference (RPD) of duplicate analyses and the percent recovery of spikes, respectively. In addition, the statistical methods used to compile and analyze the data (mean concentrations, detection frequencies) are subject to the overall uncertainty in data measurement. Furthermore, chemical concentrations in environmental media fluctuate over time and with respect to sampling location. Analytical data must be sufficient to consider the temporal and spatial characteristics of contamination at the site with respect to exposure.

Selection of COPCs

Analytical data also must be comprehensive in order to address the COPCs associated with SWMU 14. Region III RBC values are based on exposure assumptions and equations that are intended to introduce conservatism in the risk assessment process by changing the COPC screening method from a relative toxicity screen as presented in RAGS, to an absolute comparison of risk. However, the use of the Region III RBC values which incorporate a set of non-site-specific assumptions in the selection of COPCs at SWMU 14, adds conservatism to the baseline HHRA.

Currently, there are no closure plans for NSRR and future residential development of the land is not expected. The application of the residential criteria in the selection of soil COPCs would, therefore, tend to result in a list of COPCs that could be considered conservative for a military base. The use of conservative COPC selections in the baseline HHRA ensures the protection of public health.

Exposure Assessment

In performing exposure assessments, uncertainties arise from two main sources. First, uncertainties arise in estimating the fate of a compound in the environment, including estimating release and transport in a particular environmental medium. Second, uncertainties arise in the estimation of chemical intakes resulting from contact by a receptor with a particular medium. To estimate an intake, certain assumptions must be made about exposure events, exposure durations,

and the corresponding assimilation of constituents by the receptor. Exposure factors are generally derived from a range of values generated by studies of limited numbers of individuals. In all instances, values used in this HHRA, scientific judgments, and conservative assumptions agree with those of the USEPA.

Toxicological Assessment

In making quantitative estimates of the toxicity for varying dosages of compounds to human receptors, uncertainties arise from two sources. First, data on human exposure and the subsequent effects are usually insufficient, if they are at all available. Human exposure data usually lack adequate concentration estimations and suffer from inherent temporal variability. Therefore, animal studies are often used, and new uncertainties arise from the process of extrapolating animal results to humans. Second, to obtain observable effects with a manageable number of experimental subjects, high doses of a compound are often used. In this situation, a high dose means that high exposures are used in the experiment with respect to most environmental exposures. Therefore, when applying the results of the animal experiment to the human condition, the effects at the high doses must be extrapolated to approximate effects at lower doses. In extrapolating effects from high doses in animals to low doses in humans, scientific judgment and conservative assumptions are employed. In selecting animal studies for use in dose-response calculations, the following factors are considered:

- Studies are preferred where the animal closely mimics human toxicokinetics.
- Studies are preferred where dose intake most closely mimics the intake route and duration for humans.
- Studies are preferred which demonstrate the most sensitive response to the compound in question.

For compounds believed to cause threshold effects (i.e., noncarcinogens) safety factors are employed in the extrapolation of effects from animals to humans and from high doses to low doses. In deriving carcinogenic potency factors, the 95% UCL value is promulgated by the USEPA to prevent underestimation of potential risk.

Further conservatism in the baseline HHRA is introduced through the use of experimentally-derived oral absorption efficiencies to account for a difference in the degree of toxicity between an administered dose and an absorbed dose. Equating the absorption efficiency of the dermal bi-

phasic barrier to the absorption efficiency of the gastrointestinal lining is a very conservative approach that tends to overestimate the potential risk to human health.

In summary, the use of conservative assumptions results in quantitative indices of toxicity that are not expected to underestimate potential toxic effects, but may overestimate these effects by an order of magnitude or more.

Risk Characterization

The risk characterization bridges the gap between potential exposure and the possibility of systemic or carcinogenic human health effects, ultimately providing impetus for the remediation of the site or providing a basis for no remedial action.

Uncertainties associated with risk characterization include the assumption of chemical additivity and the inability to predict synergistic or antagonistic interactions between COPCs. These uncertainties are inherent in any inferential risk assessment. To account for this, USEPA-promulgated inputs to the quantitative risk assessment and toxicological indices are calculated to be protective of the human receptor and to err conservatively, so as to not underestimate the potential human health risks.

4.0 CONCLUSIONS AND RECOMMENDATIONS

The results of the HHRA did not indicate an unacceptable cancer risk for any of the exposure scenarios or pathways. Current land use scenarios that were evaluated include commercial/utility worker, and trespasser. Future land use scenarios evaluated include commercial/utility worker, trespasser, military residential (adult and child), and construction worker. Each scenario evaluated the risk associated with ingestion of contaminants in surface soil, inhalation of particulates from surface soil, and dermal contact with surface soil. Based on the results of this HHRA it is not likely that the COPCs detected in surface soils at SWMU 14 will pose a unacceptable health risk to current or future receptors.

There are currently engineering controls in place to prevent subsurface and groundwater exposure to activities conducted at the fire training pit. The engineering controls include a concrete pad with a concrete apron, and an oil and water separator which will be maintained as long as the fire training pit is utilized. SWMU 14 will not be utilized for any other function except for fire training activities. Once fire training operations cease, additional site characterization of the site will be conducted. This characterization will be determined once the site is no longer in operation. If it is determined that an unacceptable risk is present, the area would be remediated as appropriate. Therefore, it is recommended that additional site characterization and a final decision of whether or not corrective action is necessary should be determined once the usage of the area is terminated.

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TABLES

TABLE 2-1

COMPARING THE MAXIMUM CONCENTRATIONS OF CHEMICALS DETECTED IN SURFACE SOIL TO REGION III RISK BASED CONCENTRATIONS
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

Constituent	Sample ID / Sample Date	14SS04 / 3/22/96	14SS05 / 3/22/96	14SS06 / 3/22/96	14SS07 / 3/22/96	14SS08 / 3/22/96	Maximum Detected Concentration	Residential RBC[a]	Will this chemical be retained as a COPC?
SVOCs (ug/kg)									
Bis(2-ethylhexyl)phthalate		400 U	410 U	97 J	390 U	360 U	97	45,623 C	no
Diethylphthalate		400 U	410 U	54 J	390 U	360 U	54	6,257,143 N	no
Anthracene		400 U	410 U	390 U	110 J	360 U	110	2,346,429 N	no
Benz(a)anthracene		400 U	45 J	300 J	**3400**	360 U	3,400	875 C	**YES**
Benzo(b)flouranthene		400 U	410 U	**2800**	**7600**	360 U	7,600	875 C	**YES**
Benzo(k)flouranthene		400 U	410 U	640	2400	360 U	2,400	8,750 C	no
Benzo(a)pyrene		400 U	45 J	**1800**	**5000**	360 U	5,000	87 C	**YES**
Benzo(g,h,i)perylene[c]		66 J	91 J	1200	3600	360 U	3,600	234,643 N	no
Chrysene		400 U	50 J	690	3800	360 U	3,800	87,497 C	no
Dibenz(a,h)anthracene		400 U	410 U	**210 J**	**920**	360 U	920	87 C	**YES**
Flouranthene		88 J	110 J	230 J	67 J	360 U	230	312,857 N	no
Indeno(1,2,3-c,d)pyrene		400 U	58 J	**1300**	**3800**	360 U	3,800	875 C	**YES**
Phenanthrene[b]		400 U	410 U	39 J	58 J	360 U	58	2,346,429 N	no
Pyrene		170 J	270 J	650	100 J	360 U	650	234,643 N	no
PCBs (ug/kg)									
Aroclor-1260		28	17	12	19	6 J	28	319 C	no
TPH (mg/kg)									
Diesel Range Organics		560	360	120	490	4.5 U	560	NA	no
Gasoline Range Organics		3.7	3.8	1.8	1.8	0.032	3.8	NA	no

a Region III Risk Based Concentrations 10/5/00 non carcinogenic RBCs were adjusted downward to correspond to a target HQ of 0.1 rather than 1 to ensure that chemicals with additive effects are not prematurely eliminated during screening (USEPA 2000a).
b anthracene used as a surrogate value for phenanthrene
c pyrene used as a surrogate for benzo(g,h,i)perylene
N= noncarcinogenic
C=carcinogenic
U= Non-detect
J= Estimated
NA= RBCs not derived for TPHs
Bolding indicates exceedances of USEPA Region III Residential RBC's

TABLE 3-1

CARCINOGENIC TOXICITY VALUES
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

Constituents	Oral Cancer Slope Factor (CSFo) $(mg/kg\text{-}day)^{-1}$	Inhalation Cancer Slope Factor (CSFi) $(mg/kg\text{-}day)^{-1}$	Weight of Evidence	Reference
Benz(a)anthracene	0.73	NA	B2	USEPA 2000a,b
Benzo(b)flouranthene	0.73	NA	B2	USEPA 2000a,b
Benzo(a)pyrene	7.3	3.1	B2	USEPA 2000a,b
Dibenz(a,h)anthracene	7.3	NA	B2	USEPA 2000a,b
Indeno(1,2,3-cd)pyrene	0.73	NA	B2	USEPA 2000a,b

TABLE 3-2

POTENTIAL FUTURE AND CURRENT EXPOSURES TO SURFACE SOIL
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

		Future			Current/Future		
		Military Residential		Construction Worker	Trespasser-Recreator		Commercial/ Utility Worker
		Adult	Child (1-6)	Adult	Adult	Youth (7-17)	Adult
Surface Soil							
	incidental ingestion	X	X	X	X	X	X
	dermal contact	X	X	X	X	X	X
	inhalation of particulates	X	X	X	X	X	X

TABLE 3-3

EXPOSURE PARAMETERS FOR HUMAN HEALTH RISK ASSESSMENT
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

			Future			Current/Future		
			Military	Residential	Construction Worker	Tresspasser-Recreator		Commercial/Utility Worker
			Adult	Child	Adult	Adult	Youth	Adult
Exposure Duration	ED	year	4	4	1	30	9	25
Exposure Frequency	EF	days/year	350	350	180	52	52	250
Exposure Time-Soil	ET_s	hours/day	2	2	8	2	2	8
Averaging Time	AT	days						
carcinogenic			25,550	25,550	25,550	25,550	25,550	25,550
Body Weight	BW	kg	70	15	70	70	45	70
Ingestion Rate-soil	IR_s	mg/day	100	200	100	50	50	50
Dermal Absorption Factor	DAF	unitless						
SVOCs			0.10	0.10	0.10	0.10	0.10	0.10
Oral Absorption Factor			1.00	1.00	1.00	1.00	1.00	1.00
Surface Area-soil	SA_s	cm^2	5,000	1,700	5,000	5,000	3,200	5,000
Adherence Factor	AF	mg/cm^2	0.07	0.2	0.43	0.2	0.2	0.2
Inhalation Rate	IR_h	m^3/hr	1.27	0.69	2	1.27	1.27	1.27
Particulate Emmission Factor	PEF	m^3/kg	1.32×10^9	1.32×10^9	1.32×10^9	1.32×10^9	1.32×10^9	1.32×10^9

TABLE 3-4

SHAPIRO WILKES TEST RESULTS AND IDENTIFICATION OF THE EXPOSURE POINT CONCENTRATION
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

	W-Value	Quantile	Normal Distribution?	Lognormal 95th UCL (mg/kg)	Maximum Concentration (mg/kg)	Is the Lognormal 95th UCL Greater than the Maximum Detected Concentration?	Exposure Point Concentration (mg/kg)
Benz(a)anthracene	0.61	0.76	No	975.39	3.40	Yes	3.40
Benzo(b)flouranthene	0.74	0.76	No	1,222,086.06	7.60	Yes	7.60
Benzo(a)pyrene	0.75	0.76	No	539,138.14	5.00	Yes	5.00
Dibenz(a,h)anthracene	0.65	0.76	No	3.16	0.92	Yes	0.92
Indeno(1,2,3-c,d)pyrene	0.74	0.76	No	11,138.32	3.80	Yes	3.80

TABLE 3-5

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICIES (HIs)
FOR FUTURE MILITARY ADULT AND MILITARY YOUNG CHILD RESIDENTS
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

	Future Military Adult Residents		Future Military Young Child Residents	
Pathway	ILCR	HI	ILCR	HI
Surface Soil				
Ingestion	4.2E-06	0.00	3.9E-05	0.00
Dermal Contact	1.5E-06	0.00	6.7E-06	0.00
Inhalation	2.3E-11	0.00	5.9E-11	0.00
Total	5.7E-06	0.0E+00	4.6E-05	0.0E+00

Notes:

Bolding indicates exceedances of USEPA acceptable target risk criteria by pathway exposures
Shading indicates exceedances of USEPA acceptable target risk criteria by subtotal and total risk value

TABLE 3-6

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICIES (HIs)
FOR FUTURE CONSTRUCTION WORKERS
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

Pathway	Future Construction Workers	
	ILCR	HI
Surface Soil		
Ingestion	5.4E-07	0.00
Dermal Contact	5.4E-08	0.00
Inhalation	1.9E-11	0.00
Total	6.0E-07	0.0E+00

Notes:

Bolding indicates exceedances of USEPA acceptable target risk criteria by pathway exposures
Shading indicates exceedances of USEPA acceptable target risk criteria by subtotal and total risk value

TABLE 3-7

INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICIES (HIs)
CURRENT ADULT AND ADOLESCENT TRESPASSERS
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

	Current and Future Adult Trespassers		Current and Future Adolescent Trespassers	
Pathway	ILCR	HI	ILCR	HI
Surface Soil Ingestion	2.4E-06	0.00	1.1E-06	0.00
Dermal Contact	4.7E-06	0.00	1.4E-06	0.00
Inhalation	2.6E-11	0.00	1.2E-11	0.00
Total	7.1E-06	0.0E+00	2.5E-06	0.0E+00

Notes:

Bolding indicates exceedances of USEPA acceptable target risk criteria by pathway exposures
Shading indicates exceedances of USEPA acceptable target risk criteria by subtotal and total risk value

TABLE 3-8

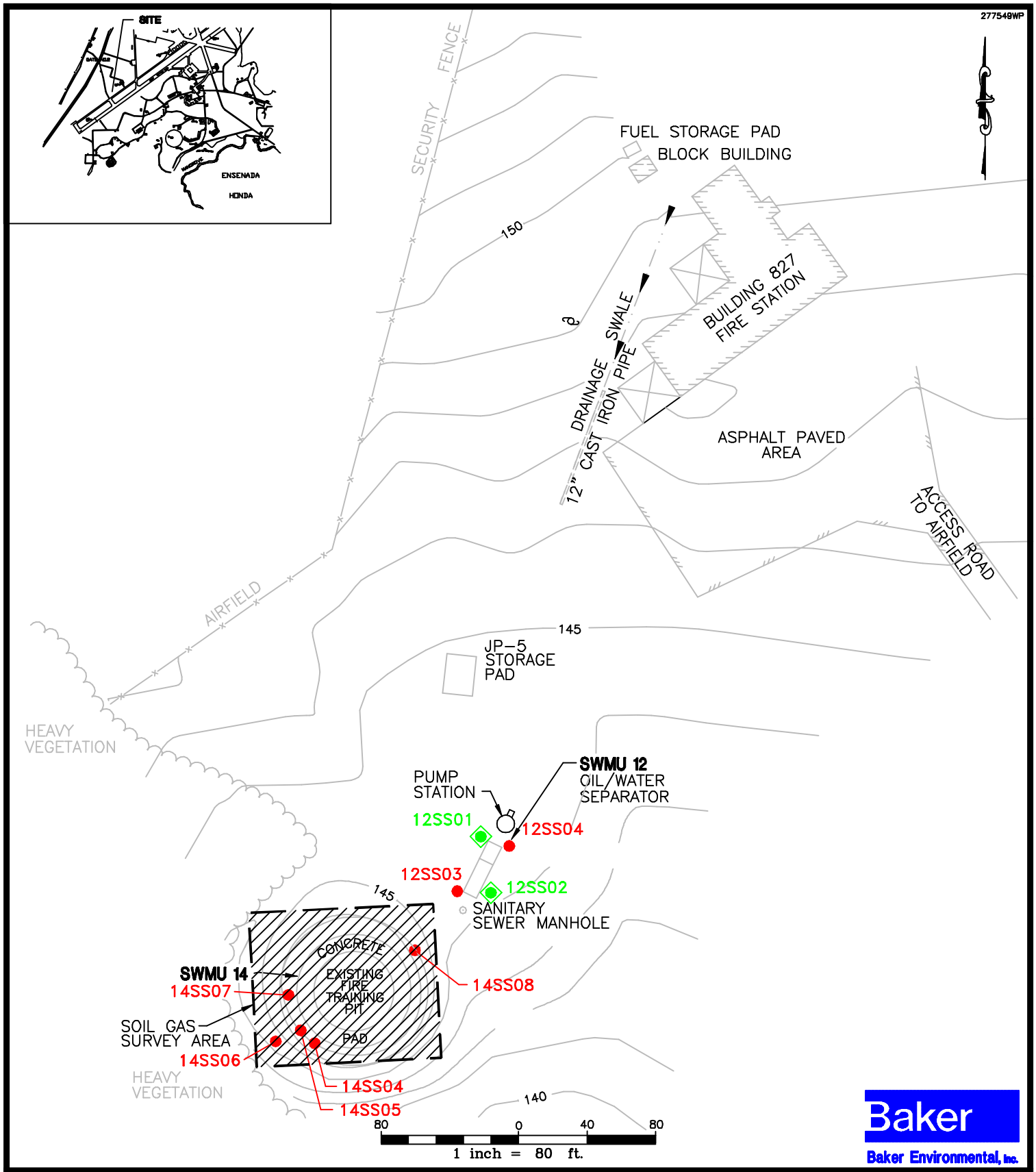
INCREMENTAL LIFETIME CANCER RISKS (ILCRs) AND HAZARD INDICIES (HIs)
FOR CURRENT COMMERCIAL/UTILITY WORKERS
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

Pathway	Current Commercial/Utility Workers	
	ILCR	HI
Surface Soil		
Ingestion	9.4E-06	0.00
Dermal Contact	1.9E-05	0.00
Inhalation	4.2E-10	0.00
TotaL	2.8E-05	0.0E+00

Notes:

Bolding indicates exceedances of USEPA acceptable target risk criteria by pathway exposures
Shading indicates exceedances of USEPA acceptable target risk criteria by subtotal and total risk value

FIGURES



LEGEND

- 145 SURFACE ELEVATION CONTOUR
- SURFACE WATER DRAINAGE DIRECTION
- SURFACE SOIL SAMPLE COLLECTED (9-95)
- SURFACE SOIL SAMPLE COLLECTED (3-22-96)
- AREA OF SOIL GAS SURVEY

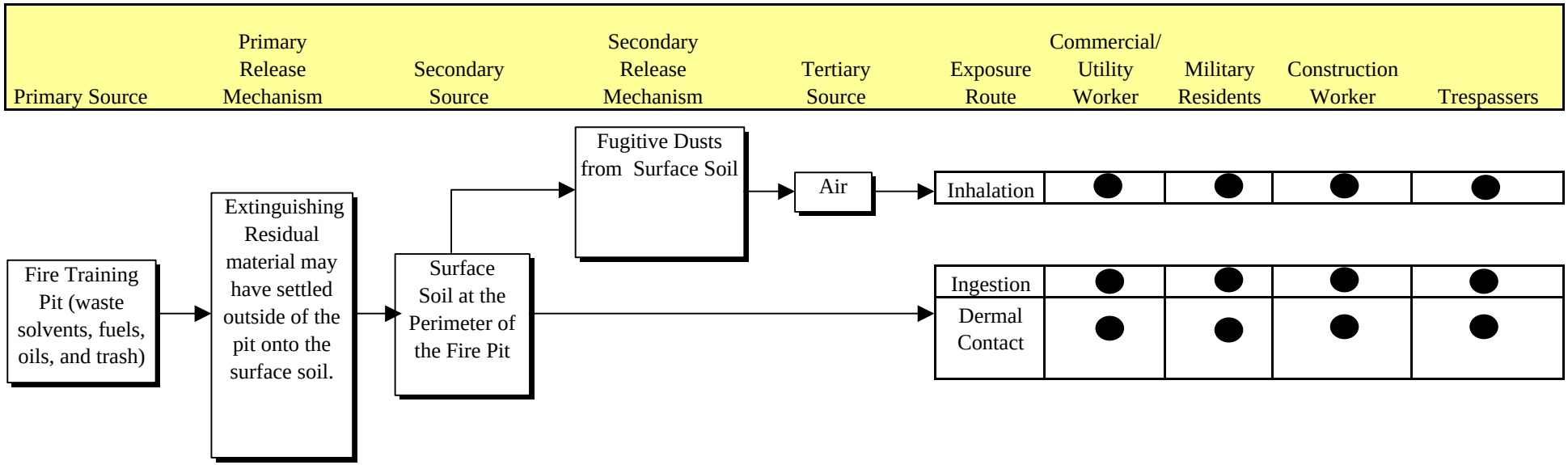
SOURCE: LANTDIV, FEB. 1992.

FIGURE 2-1
SAMPLING LOCATIONS
SWMU 14 – FIRE TRAINING PIT AREA
OPERABLE UNIT NO. 1

NAVAL STATION ROOSEVELT ROADS
PUERTO RICO

FIGURE 3-1

EXPOSURE PATHWAYS TO SURFACE SOIL
SWMU 14
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO



Legend

● Complete Exposure Pathway

○ Incomplete Exposure Pathway

APPENDIX A
RCRA FACILITY INVESTIGATION DATA (BAKER 1996)

APPENDIX A

ANALYTICAL SUMMARY OF ORGANIC COMPOUNDS IN SWMU 14 SURFACE SOIL
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

SAMPLE ID	14SS04	14SS05	14SS06	14SS07	14SS08
SAMPLE DATE	03/22/96	03/22/96	03/22/96	03/22/96	03/22/96
VOLATILES (ug/kg)					
CARBON TETRACHLORIDE	6 U	6 UJ	6 U	6 U	5 U
ACETONE	12 UJ	12 UJ	12 UJ	12 UJ	11 UJ
CHLOROFORM	6 U	6 UJ	6 U	6 U	5 U
BENZENE	6 U	6 UJ	6 U	6 U	5 U
1,1,1-TRICHLOROETHANE	6 U	6 UJ	6 U	6 U	5 U
BROMOMETHANE	12 U	12 UJ	12 U	12 U	11 U
CHLOROMETHANE	12 U	12 UJ	12 U	12 U	11 U
IODOMETHANE	12 U	12 UJ	12 U	12 U	11 U
DIBROMOMETHANE	12 U	12 UJ	12 U	12 UJ	11 UJ
CHLOROETHANE	12 U	12 UJ	12 U	12 U	11 U
VINYL CHLORIDE	12 U	12 UJ	12 U	12 U	11 U
ACETONITRILE	120 U	120 UJ	120 U	120 U	110 U
METHYLENE CHLORIDE	6 UJ	6 UJ	6 UJ	6 U	5 U
CARBON DISULFIDE	6 U	6 UJ	6 U	6 U	5 U
BROMOFORM	6 U	6 UJ	6 U	6 U	5 U
BROMODICHLOROMETHANE	6 U	6 UJ	6 U	6 U	5 U
1,1-DICHLOROETHANE	6 U	6 UJ	6 U	6 U	5 U
1,1-DICHLOROETHENE	6 U	6 UJ	6 U	6 U	5 U
TRICHLOROFLUOROMETHANE	12 UJ	12 UJ	12 UJ	12 UJ	11 UJ
DICHLORODIFLUOROMETHANE	25 U	25 UJ	23 U	24 UJ	22 UJ
PENTACHLOROETHANE	25 U	25 UJ	23 U	24 U	22 U
ISOBUTYL ALCOHOL	2500 R	2500 R	2300 R	2400 R	2200 R
1,2-DICHLOROPROPANE	6 U	6 UJ	6 U	6 U	5 U
2-BUTANONE	12 U	12 UJ	12 U	12 U	11 U
1,1,2-TRICHLOROETHANE	6 U	6 UJ	6 U	6 U	5 U
TRICHLOROETHENE	6 U	6 UJ	6 U	6 U	5 U
1,1,2,2-TETRACHLOROETHANE	6 U	6 UJ	6 U	6 U	5 U
METHYLMETHACRYLATE	25 U	25 UJ	23 U	24 U	22 U
1,2-DIBROMO-3-CHLOROPROPANE	25 UJ	25 UJ	23 UJ	24 U	22 U
1,2,3-TRICHLOROPROPANE	12 U	12 UJ	12 U	12 U	11 U
ETHYLMETHACRYLATE	25 U	25 UJ	23 U	24 U	22 U
ETHYLBENZENE	6 U	6 UJ	6 U	6 UJ	5 UJ
STYRENE	6 U	6 UJ	6 U	6 U	5 U
1,2-DIBROMOETHANE	25 U	25 UJ	23 U	24 U	22 U
ACROLEIN	620 U	630 UJ	590 U	590 U	540 U
3-CHLOROPROPENE	25 U	25 UJ	23 U	24 U	22 U
1,2-DICHLOROETHANE	6 UJ	6 UJ	6 UJ	6 U	5 U
PROPIONITRILE (ETHYL CYANIDE)	62 R	63 R	59 R	59 R	54 R
ACRYLONITRILE	120 U	120 UJ	120 U	120 U	110 U
VINYL ACETATE	12 U	12 UJ	12 U	12 U	11 U
4-METHYL-2-PENTANONE	12 U	12 UJ	12 U	12 U	11 U
TOLUENE	6 U	6 UJ	6 U	6 U	5 U
CHLOROBENZENE	6 U	6 UJ	6 U	6 U	5 U
TRANS-1,4-DICHLORO-2-BUTENE	25 U	25 UJ	23 U	24 U	22 U
DIBROMOCHLOROMETHANE	6 U	6 UJ	6 U	6 U	5 U
METHACRYLONITRILE	25 U	25 UJ	23 U	24 U	22 U
2-CHLORO-1,3-BUTADIENE	120 U	120 UJ	120 U	120 U	110 U
TETRACHLOROETHENE	6 U	6 UJ	6 U	6 U	5 U
1,2-DICHLOROETHENE (TOTAL)	6 U	6 UJ	6 U	6 U	5 U
2-HEXANONE	12 UJ	12 UJ	12 UJ	12 U	11 U
1,1,1,2-TETRACHLOROETHANE	12 U	12 UJ	12 U	12 U	11 U
XYLENE (TOTAL)	6 U	6 UJ	6 U	6 U	5 U
CIS-1,3-DICHLOROPROPENE	6 U	6 UJ	6 U	6 U	5 U
TRANS-1,3-DICHLOROPROPENE	6 U	6 UJ	6 U	6 U	5 U

APPENDIX A

ANALYTICAL SUMMARY OF ORGANIC COMPOUNDS IN SWMU 14 SURFACE SOIL
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

SAMPLE ID	14SS04	14SS05	14SS06	14SS07	14SS08
SAMPLE DATE	03/22/96	03/22/96	03/22/96	03/22/96	03/22/96
SEMIVOLATILES (ug/kg)					
BENZO(A)PYRENE	400 U	45 J	1800	5000	360 U
2,4-DINITROPHENOL	2000 UJ	2100 UJ	1900 UJ	1900 UJ	1800 UJ
DIBENZO(A,H)ANTHRACENE	400 U	410 U	210 J	920	360 U
2-ACETYLAMINOFLUORENE	800 U	820 U	770 U	770 U	710 U
N-NITROSODIETHYLAMINE	400 UJ	410 U	390 U	390 U	360 U
3-METHYLCHOLANTHRENE	400 U	410 U	390 U	390 U	360 U
BENZO(A)ANTHRACENE	400 U	45 J	300 J	3400	360 U
4-NITROQUINOLINE-1-OXIDE	2000 UJ	2100 UJ	1900 UJ	1900 UJ	1800 UJ
7,12-DIMETHYLBENZ(A)ANTHRACENE	800 U	820 U	770 U	770 U	710 U
2,3,4,6-TETRACHLOROPHENOL	400 UJ	410 UJ	390 UJ	390 UJ	360 UJ
4-CHLORO-3-METHYLPHENOL	800 U	820 U	770 U	770 U	710 U
N-NITROSOMORPHOLINE	800 UJ	820 UJ	770 UJ	770 UJ	710 UJ
P-DIMETHYLAMINOAZOBENZENE	800 U	820 U	770 U	770 U	710 U
PHENACETIN	400 U	410 U	390 U	390 U	360 U
ETHYL METHANESULFONATE	400 U	410 U	390 U	390 U	360 U
ANILINE	2000 UJ	2100 UJ	1900 UJ	1900 UJ	1800 UJ
N-NITROSODIMETHYLAMINE	400 U	410 UJ	390 U	390 U	360 U
BENZOIC ACID	2000 U	2100 U	1900 U	1900 U	1800 U
METHYL METHANESULFONATE	400 U	410 U	390 U	390 U	360 U
HEXACHLOROETHANE	400 U	410 U	390 U	390 U	360 U
HEXACHLOROPHENE	4000 R	4100 R	3900 R	3900 R	3600 R
HEXACHLOROCYCLOPENTADIENE	400 U	410 U	390 U	390 U	360 U
ISOPHORONE	400 U	410 U	390 U	390 U	360 U
PENTACHLORONITROBENZENE	400 U	410 U	390 U	390 U	360 U
ACENAPHTHENE	400 U	410 U	390 U	390 U	360 U
DIETHYLPHTHALATE	400 U	410 U	54 J	390 U	360 U
DI-N-BUTYLPHTHALATE	400 U	410 U	390 U	390 U	360 U
PHENANTHRENE	400 U	410 U	39 J	58 J	360 U
BUTYLBENZYLPHTHALATE	400 U	410 U	390 U	390 U	360 U
N-NITROSODIPHENYLAMINE (1)	400 U	410 U	390 U	390 U	360 U
FLUORENE	400 U	410 U	390 U	390 U	360 U
CARBAZOLE	400 U	410 U	390 U	390 U	360 U
2,6-DICHLOROPHENOL	400 U	410 U	390 U	390 U	360 U
HEXACHLOROBUTADIENE	400 U	410 U	390 U	390 U	360 U
PENTACHLOROPHENOL	2000 U	2100 U	1900 U	1900 U	1800 U
2,4,6-TRICHLOROPHENOL	400 U	410 U	390 U	390 U	360 U
2-NITROANILINE	2000 U	2100 U	1900 U	1900 U	1800 U
2-NITROPHENOL	400 U	410 U	390 U	390 U	360 U
2-SEC-BUTYL-4,6-DINITROPHENOL	800 UJ	820 UJ	770 UJ	770 UJ	710 UJ
NAPHTHALENE	400 U	410 U	390 U	390 U	360 U
2-METHYLNAPHTHALENE	400 UJ	410 UJ	390 UJ	390 UJ	360 UJ
2-CHLORONAPHTHALENE	400 U	410 U	390 U	390 U	360 U
2-NAPHTHYLAMINE	1000 U	1000 U	970 U	970 U	890 U
METHAPYRILENE	1000 UJ	1000 UJ	970 UJ	970 UJ	890 UJ
3,3'-DICHLOROBENZIDINE	800 U	820 U	770 U	770 U	710 U
4-AMINOBIPHENYL	800 U	820 U	770 U	770 U	710 U
BENZIDINE	4000 UJ	4100 UJ	3900 UJ	3900 UJ	3600 UJ
SAFROLE	400 U	410 U	390 U	390 U	360 U
O-CRESOL	400 U	410 U	390 U	390 U	360 U
1,2-DICHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
O-TOLUIDINE	400 R	410 R	390 R	390 R	360 R
2-CHLOROPHENOL	400 U	410 U	390 U	390 U	360 U
1,2,4,5-TETRACHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
2,4,5-TRICHLOROPHENOL	2000 U	2100 U	1900 U	1900 U	1800 U

APPENDIX A

ANALYTICAL SUMMARY OF ORGANIC COMPOUNDS IN SWMU 14 SURFACE SOIL
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

SAMPLE ID	14SS04	14SS05	14SS06	14SS07	14SS08
SAMPLE DATE	03/22/96	03/22/96	03/22/96	03/22/96	03/22/96
ACETOPHENONE	400 U	410 U	390 U	390 U	360 U
NITROBENZENE	400 U	410 U	390 U	390 U	360 U
3-NITROANILINE	2000 U	2100 U	1900 U	1900 U	1800 U
1,3,5-TRINITROBENZENE	4000 U	4100 U	3900 U	3900 U	3600 U
5-NITRO-O-TOLUIDINE	800 U	820 U	770 U	770 U	710 U
1,3-DINITROBENZENE	800 U	820 U	770 U	770 U	710 U
4-NITROANILINE	2000 U	2100 U	1900 U	1900 U	1800 U
4-NITROPHENOL	2000 U	2100 U	1900 U	1900 U	1800 U
BENZYL ALCOHOL	400 UJ	410 UJ	390 UJ	390 UJ	360 UJ
N-NITROSOPIPERIDINE	400 U	410 U	390 U	390 U	360 U
4-BROMOPHENYL-PHENYLETHER	400 U	410 U	390 U	390 U	360 U
2,4-DIMETHYLPHENOL	400 U	410 U	390 U	390 U	360 U
1,4-DICHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
4-CHLOROANILINE	800 U	820 U	770 U	770 U	710 U
P-PHENYLENEDIAMINE	800 UJ	820 UJ	770 UJ	770 UJ	710 UJ
2,2'-OXYBIS(1-CHLOROPROPANE)	400 U	410 U	390 U	390 U	360 U
PHENOL	400 U	410 U	390 U	390 U	360 U
2-PICOLINE	400 R	410 R	390 R	390 R	360 R
PYRIDINE	800 U	820 U	770 U	770 U	710 U
BIS(2-CHLOROETHYL)ETHER	400 U	410 U	390 U	390 U	360 U
BIS(2-CHLOROETHOXY)METHANE	400 U	410 U	390 U	390 U	360 U
BIS(2-ETHYLHEXYL)PHTHALATE	400 U	410 U	97 J	390 U	360 U
DI-N-OCTYL PHTHALATE	400 U	410 U	390 U	390 U	360 U
HEXACHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
3,3'-DIMETHYLBENZIDINE	2000 U	2100 U	1900 U	1900 U	1800 U
ANTHRACENE	400 U	410 U	390 U	110 J	360 U
ISOSAFROLE	400 UJ	410 UJ	390 UJ	390 UJ	360 UJ
1,2,4-TRICHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
2,4-DICHLOROPHENOL	400 U	410 U	390 U	390 U	360 U
2,4-DINITROTOLUENE	400 U	410 U	390 U	390 U	360 U
A,A-DIMETHYLPHENETHYLAMINE	2000 U	2100 U	1900 U	1900 U	1800 U
DIPHENYLAMINE	400 U	410 U	390 U	390 U	360 U
1,2-DIPHENYLHYDRAZINE	400 U	410 U	390 U	390 U	360 U
1,4-DIOXANE	800 R	820 R	770 R	770 R	710 R
PYRENE	170 J	270 J	650	100 J	360 U
1,4-NAPHTHOQUINONE	2000 UJ	2100 UJ	1900 UJ	1900 UJ	1800 UJ
DIMETHYLPHTHALATE	400 U	410 U	390 U	390 U	360 U
DIBENZOFURAN	400 U	410 U	390 U	390 U	360 U
1-NAPHTHYLAMINE	800 U	820 U	770 U	770 U	710 U
ARAMITE	800 UJ	820 UJ	770 UJ	770 UJ	710 UJ
BENZO(G,H,I)PERYLENE	66 J	91 J	1200	3600	360 U
INDENO(1,2,3-CD)PYRENE	400 U	58 J	1300	3800	360 U
BENZO(B)FLUORANTHENE	400 U	410 U	2800	7600	360 U
FLUORANTHENE	88 J	110 J	230 J	67 J	360 U
BENZO(K)FLUORANTHENE	400 U	410 U	640	2400	360 U
ACENAPHTHYLENE	400 U	410 U	390 U	390 U	360 U
CHRYSENE	400 U	50 J	690	3800	360 U
CHLOROBENZILATE	400 UJ	410 UJ	390 UJ	390 UJ	360 UJ
4,6-DINITRO-2-METHYLPHENOL	2000 UJ	2100 UJ	1900 UJ	1900 UJ	1800 UJ
1,3-DICHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
2,6-DINITROTOLUENE	400 U	410 U	390 U	390 U	360 U
PENTACHLOROBENZENE	400 U	410 U	390 U	390 U	360 U
N-NITROSO-DI-N-PROPYLAMINE	400 U	410 U	390 U	390 U	360 U
N-NITROSO-DI-N-BUTYLAMINE	400 UJ	410 UJ	390 UJ	390 UJ	360 UJ
N-NITROSOPYRROLIDINE	2000 UJ	2100 UJ	1900 UJ	1900 UJ	1800 UJ

APPENDIX A

ANALYTICAL SUMMARY OF ORGANIC COMPOUNDS IN SWMU 14 SURFACE SOIL
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

SAMPLE ID	14SS04	14SS05	14SS06	14SS07	14SS08
SAMPLE DATE	03/22/96	03/22/96	03/22/96	03/22/96	03/22/96
HEXACHLOROPROPENE	2000 U	2100 U	1900 U	1900 U	1800 U
DIALLATE	400 U	410 U	390 U	390 U	360 U
4-CHLOROPHENYL-PHENYLETHER	400 U	410 U	390 U	390 U	360 U
N-NITROSOMETHYLETHYLAMINE	400 U	410 U	390 UJ	390 UJ	360 UJ
PRONAMIDE	400 U	410 U	390 U	390 U	360 U
META & PARA-CRESOL	400 U	410 U	390 U	390 U	360 U
PCB (ug/kg)					
AROCLOR-1260	28	17	12	19	6 NJ
AROCLOR-1254	96 U	98 U	93 U	93 U	85 U
AROCLOR-1221	48 U	49 U	46 U	46 U	42 U
AROCLOR-1232	48 U	49 U	46 U	46 U	42 U
AROCLOR-1248	48 U	49 U	46 U	46 U	42 U
AROCLOR-1016	48 U	49 U	46 U	46 U	42 U
AROCLOR-1242	48 U	49 U	46 U	46 U	42 U
TPH (mg/kg)					
DIESEL FUEL	560	360	120	490	4.5 U
GASOLINE	3.7	3.8	1.8	1.8	0.032

APPENDIX B
HHRA RISK CALCULATIONS

MILITARY ADULT AND YOUNG CHILD RESIDENTS (AGES 1 TO 6 YEARS) - FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS EMANATING FROM SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

CDI (mg/kg-d)= $(Ca*IR_h*ET*EF*ED)/(BW*AT)$
Where: $Ca = Cs * (1/PEF)$

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Adult	Young Child	
CDI	Chronic daily intake (mg/kg-d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg-d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Inhalation reference dose (mg/kg-d)	CS	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m^3)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
PEF	Particulate emission factor (m^3/kg)	1.32E+09	1.32E+09	USEPA 1996 assumes 50% vegetation
IR_h	Respiration rate (m^3/hr)	1.27	0.69	
ET	Exposure time (hrs/d)	2	2	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	4	4	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25,550	25,550	
ATn	Averaging time, noncarcinogens (d)	1,460	1,460	

					Adult						Young Child					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFi 1/(mg/kg-d)	RfDi (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	2.576E-09	NA	NA	5.1E-12	--	--	9.0E-11	--	--	1.3E-11	--	--	2.3E-10	--	--
Benzo(b)fluoranthene	7.60	5.758E-09	NA	NA	1.1E-11	--	--	2.0E-10	--	--	2.9E-11	--	--	5.1E-10	--	--
Benzo(a)pyrene	5.00	3.788E-09	3.1	NA	7.5E-12	2.3E-11	100.0%	1.3E-10	--	--	1.9E-11	5.9E-11	100.0%	3.3E-10	--	--
Dibenz(a,h)anthracene	0.92	6.97E-10	NA	NA	1.4E-12	--	--	2.4E-11	--	--	3.5E-12	--	--	6.1E-11	--	--
Indeno(1,2,3 c,d)pyrene	3.80	2.879E-09	NA	NA	5.7E-12	--	--	1.0E-10	--	--	1.5E-11	--	--	2.5E-10	--	--
					Total ILCR:	2.3E-11	100.0%	HI:	0.0E+00	0.0%	Total ILCR:	5.9E-11	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

MILITARY ADULT AND YOUNG CHILD RESIDENTS (AGES 1 TO 6 YEARS) - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

CDI (mg/kg-d)= (Cs*IR*CF*ABS*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult	Young Child	
CDI	Chronic daily intake (mg/kg-d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	100	200	
CF	Conversion factor (kg/mg)	0.000001	0.000001	
ABS	Amount available for Absorption	1	1	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	4	4	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25,550	25,550	
ATn	Averaging time, noncarcinogens (d)	1,460	1,460	

				Adult						Young Child					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg-d)	RfDo (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	7.30E-01	NA	2.7E-07	1.9E-07	4.6%	4.7E-06	--	--	2.5E-06	1.8E-06	4.6%	4.3E-05	--	--
Benzo(b)fluoranthene	7.60	7.30E-01	NA	5.9E-07	4.3E-07	10.3%	1.0E-05	--	--	5.6E-06	4.1E-06	10.3%	9.7E-05	--	--
Benzo(a)pyrene	5.00	7.30E+00	NA	3.9E-07	2.9E-06	67.6%	6.8E-06	--	--	3.7E-06	2.7E-05	67.6%	6.4E-05	--	--
Dibenz(a,h)anthracene	0.92	7.30E+00	NA	7.2E-08	5.3E-07	12.4%	1.3E-06	--	--	6.7E-07	4.9E-06	12.4%	1.2E-05	--	--
Indeno(1,2,3 c,d)pyrene	3.80	7.30E-01	NA	3.0E-07	2.2E-07	5.1%	5.2E-06	--	--	2.8E-06	2.0E-06	5.1%	4.9E-05	--	--
				Total ILCR:	4.2E-06	100.0%	HI:	0.0E+00	0.0%	Total ILCR:	3.9E-05	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

MILITARY ADULT AND YOUNG CHILD RESIDENTS (AGES 1 TO 6 YEARS) - FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

DAD (mg/kg-d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFd
HQ = CDI/RfDd

Parameter	Description	Adult	Young Child	
DAD	Dermally absorbed dose (mg/kg-d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1.00E-06	1.00E-06	
AF	Soil to skin adherence factor (mg/cm^2-event)	0.07	0.2	
ABS	Absorption fraction	CS	CS	
A	Skin surface area available for contact (cm^2)	5,000	1,700	
EF	Exposure Frequency (d/yr)	350	350	
ED	Exposure Duration (yrs)	4	4	
BW	Body weight (kg)	70	15	
ATc	Averaging time, carcinogens (d)	25,550	25,550	
ATn	Averaging time, noncarcinogens (d)	1,460	1,460	

					Adult						Young Child					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFd 1/(mg/kg-d)	RfDd (mg/kg-d)	DAD (mg/kg-d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg-d)	HQ	% Contrib. HI	DAD (mg/kg-d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthrancene	3.40	0.10	7.30E-01	NA	9.3E-08	6.8E-08	4.6%	1.6E-06	--	--	4.2E-07	3.1E-07	4.6%	7.4E-06	--	--
Benzo(b)fluoranthene	7.60	0.10	7.30E-01	NA	2.1E-07	1.5E-07	10.3%	3.6E-06	--	--	9.4E-07	6.9E-07	10.3%	1.7E-05	--	--
Benzo(a)pyrene	5.00	0.10	7.30E+00	NA	1.4E-07	1.0E-06	67.6%	2.4E-06	--	--	6.2E-07	4.5E-06	67.6%	1.1E-05	--	--
Dibenz(a,h)anthracene	0.92	0.10	7.30E+00	NA	2.5E-08	1.8E-07	12.4%	4.4E-07	--	--	1.1E-07	8.3E-07	12.4%	2.0E-06	--	--
Indeno(1,2,3 c,d)pyrene	3.80	0.10	7.30E-01	NA	1.0E-07	7.6E-08	5.1%	1.8E-06	--	--	4.7E-07	3.4E-07	5.1%	8.3E-06	--	--
					Total ILCR:	1.5E-06	100.0%	HI:	0.0E+00	0.0%	Total ILCR:	6.7E-06	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

CONSTRUCTION WORKERS - FUTURE SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE- SWMU 14
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

CDI (mg/kg-d)= (Cs*IR*CF*ABS*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult	
CDI	Chronic daily intake (mg/kg-d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	100	
CF	Conversion factor (kg/mg)	1.00E-06	
ABS	Amount available for Absorption	1	
EF	Exposure Frequency (d/yr)	180	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25,550	
ATn	Averaging time, noncarcinogens (d)	365	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg-d)	RfDo (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	7.30E-01	NA	3.4E-08	2.5E-08	4.6%	2.4E-06	--	--
Benzo(b)fluoranthene	7.60	7.30E-01	NA	7.6E-08	5.6E-08	10.3%	5.4E-06	--	--
Benzo(a)pyrene	5.00	7.30E+00	NA	5.0E-08	3.7E-07	67.6%	3.5E-06	--	--
Dibenz(a,h)anthracene	0.92	7.30E+00	NA	9.3E-09	6.8E-08	12.4%	6.5E-07	--	--
Indeno(1,2,3 c,d)pyrene	3.80	7.30E-01	NA	3.8E-08	2.8E-08	5.1%	2.7E-06	--	--
				Total ILCR:	5.4E-07	100.0%	Total HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

CONSTRUCTION WORKERS - FUTURE SCENARIO
DERMAL CONTACT WITH SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE -SWMU 14
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

DAD (mg/kg-d)= (Cs*CF*AF*ABS*SA*EF*ED)/(BW*AT)
ILCR = CDI*CSFd
HQ = CDI/RfDd

Parameter	Description	Adult	
DAD	Dermally absorbed dose (mg/kg-d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1.00E-06	
AF	Soil to skin adherence factor (mg/cm^2-event)	0.43	
ABS	Absorption fraction	CS	
SA	Skin surface area available for contact (cm^2)	5,000	
EF	Exposure Frequency (d/yr)	180	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25,550	
ATn	Averaging time, noncarcinogens (d)	365	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFd 1/(mg/kg-d)	RfDd (mg/kg-d)	DAD (mg/kg-d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	0.10	7.30E-01	NA	7.4E-08	5.4E-08	100.0%	5.1E-06	--	--
Benzo(b)fluoranthene	7.60	0.10	7.30E-01	NA	1.6E-07	1.2E-07	223.5%	1.2E-05	--	--
Benzo(a)pyrene	5.00	0.10	7.30E+00	NA	1.1E-07	7.9E-07	1470.6%	7.6E-06	--	--
Dibenz(a,h)anthracene	0.92	0.10	7.30E+00	NA	2.0E-08	1.5E-07	270.6%	1.4E-06	--	--
Indeno(1,2,3 c,d)pyrene	3.80	0.10	7.30E-01	NA	8.2E-08	6.0E-08	111.8%	5.8E-06	--	--
					Total ILCR:	5.4E-08	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

CONSTRUCTION WORKERS - FUTURE SCENARIO
INHALATION OF FUGITIVE DUSTS EMANATING FROM SURFACE SOIL
REASONABLE MAXIMUM EXPOSURE- SWMU
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

CDI (mg/kg-d)= $(Ca*IR_h*ET*EF*ED)/(BW*AT)$
Where: $Ca = Cs * (1/PEF)$

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Adult	
CDI	Chronic daily intake (mg/kg-d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg-d))	CS	
HQ	Hazard quotient	CS	
RfDi	Inhalation reference dose (mg/kg-d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m^3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor (m^3/kg)	1.32E+09	
IR_h	Respiration rate (m^3/hr)	2.00	
ET	Exposure time (hrs/d)	8	
EF	Exposure Frequency (d/yr)	180	
ED	Exposure Duration (yrs)	1	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25,550	
ATn	Averaging time, noncarcinogens (d)	365	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m^3)	CSFi 1/(mg/kg-d)	RfDi (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	2.576E-09	NA	NA	4.1E-12	--	--	2.9E-10	--	--
Benzo(b)fluoranthene	7.60	5.758E-09	NA	NA	9.3E-12	--	--	6.5E-10	--	--
Benzo(a)pyrene	5.00	3.788E-09	3.1	NA	6.1E-12	1.9E-11	100.0%	4.3E-10	--	--
Dibenz(a,h)anthracene	0.92	6.97E-10	NA	NA	1.1E-12	--	--	7.9E-11	--	--
Indeno(1,2,3 c,d)pyrene	3.80	2.879E-09	NA	NA	4.6E-12	--	--	3.2E-10	--	--
					Total ILCR:	1.9E-11	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TRESPASSERS - CURRENT AND FUTURE SCENARIOS
ACCIDENTAL INGESTION OF SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

CDI (mg/kg-d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	Adult	Adolescent	
CDI	Chronic daily intake (mg/kg-d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
IR	Ingestion Rate (mg/d)	50	50	
CF	Conversion factor (kg/mg)	1.00E-06	0.000001	
FI	Fraction of soil ingested from site	1	1	
EF	Exposure Frequency (d/yr)	52	52	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	45	
ATc	Averaging time, carcinogens (d)	25,550	25,550	
ATn	Averaging time, noncarcinogens (d)	10,950	3,285	

				Adult						Adolescent					
				Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg-d)	RfDo (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	7.30E-01	NA	1.5E-07	1.1E-07	4.6%	3.5E-07	--	--	6.9E-08	5.1E-08	4.6%	5.4E-07	--	--
Benzo(b)fluoranthene	7.60	7.30E-01	NA	3.3E-07	2.4E-07	10.3%	7.7E-07	--	--	1.5E-07	1.1E-07	10.3%	1.2E-06	--	--
Benzo(a)pyrene	5.00	7.30E+00	NA	2.2E-07	1.6E-06	67.6%	5.1E-07	--	--	1.0E-07	7.4E-07	67.6%	7.9E-07	--	--
Dibenz(a,h)anthracene	0.92	7.30E+00	NA	4.0E-08	2.9E-07	12.4%	9.4E-08	--	--	1.9E-08	1.4E-07	12.4%	1.5E-07	--	--
Indeno(1,2,3-cd)pyrene	3.80	7.30E-01	NA	1.7E-07	1.2E-07	5.1%	3.9E-07	--	--	7.7E-08	5.6E-08	5.1%	6.0E-07	--	--
				Total ILCR:	2.4E-06	100.0%	Total HI:	0.0E+00	0.0%	Total ILCR:	1.1E-06	100.0%	Total HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TRESPASSERS - CURRENT AND FUTURE SCENARIOS
DERMAL CONTACT WITH SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

DAD (mg/kg-d)= (Cs*CF*AF*ABS*SA*EF*ED)/(BW*AT)
ILCR = CDI*CSFd
HQ = CDI/RfDd

Parameter	Description	Adult	Adolescent	
DAD	Dermally absorbed dose (mg/kg-d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
CF	Conversion factor (kg/mg)	1.00E-06	1.00E-06	
AF	Soil to skin adherence factor (mg/cm^2-event)	0.2	0.2	
ABS	Absorption fraction	CS	CS	
SA	Skin surface area available for contact (cm^2)	5,000	3,200	
EF	Exposure Frequency (d/yr)	52	52	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	45	
ATc	Averaging time, carcinogens (d)	25,550	25,550	
ATn	Averaging time, noncarcinogens (d)	10,950	3,285	

					Adult						Adolescent					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFd 1/(mg/kg-d)	RfDd (mg/kg-d)	DAD (mg/kg-d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg-d)	HQ	% Contrib. HI	DAD (mg/kg-d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	0.10	7.30E-01	NA	3.0E-07	2.2E-07	4.6%	6.9E-07	--	--	8.9E-08	6.5E-08	4.6%	6.9E-07	--	--
Benzo(b)fluoranthene	7.60	0.10	7.30E-01	NA	6.6E-07	4.8E-07	10.3%	1.5E-06	--	--	2.0E-07	1.4E-07	10.3%	1.5E-06	--	--
Benzo(a)pyrene	5.00	0.10	7.30E+00	NA	4.4E-07	3.2E-06	67.6%	1.0E-06	--	--	1.3E-07	9.5E-07	67.6%	1.0E-06	--	--
Dibenz(a,h)anthracene	0.92	0.10	7.30E+00	NA	8.0E-08	5.9E-07	12.4%	1.9E-07	--	--	2.4E-08	1.7E-07	12.4%	1.9E-07	--	--
Indeno(1,2,3-cd)pyrene	3.80	0.10	7.30E-01	NA	3.3E-07	2.4E-07	5.1%	7.7E-07	--	--	9.9E-08	7.2E-08	5.1%	7.7E-07	--	--
					Total ILCR:	4.7E-06	100.0%	HI:	0.0E+00	0.0%	Total ILCR:	1.4E-06	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

TRESPASSERS - CURRENT AND FUTURE SCENARIOS
INHALATION OF FUGITIVE DUSTS EMANATING FROM SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS

CDI (mg/kg-d)= $(Ca*IR_h*ET*EF*ED)/(BW*AT)$
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	Adult	Adolescent	
CDI	Chronic daily intake (mg/kg-d)	CS	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg-d))	CS	CS	
HQ	Hazard quotient	CS	CS	
RfDi	Inhalation reference dose (mg/kg-d)	CS	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m^3)	CS	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	CS	
PEF	Particulate emission factor (m^3/kg)	1.32E+09	1.32E+09	
IR_h	Respiration rate (m^3/hr)	1.27	1.27	
ET	Exposure time (hrs/d)	2	2	
EF	Exposure Frequency (d/yr)	52	52	
ED	Exposure Duration (yrs)	30	9	
BW	Body weight (kg)	70	45	
ATc	Averaging time, carcinogens (d)	25,550	25,550	
ATn	Averaging time, noncarcinogens (d)	10,950	3,285	

					Adult						Adolescent					
					Carcinogens			Noncarcinogens			Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	Ca (mg/m3)	CSFi 1/(mg/kg-d)	RfDi (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.40	2.58E-09	NA	NA	5.7E-12	--	--	1.3E-11	--	--	2.7E-12	--	--	2.1E-11	--	--
Benzo(b)fluoranthene	7.60	5.76E-09	NA	NA	1.3E-11	--	--	3.0E-11	--	--	6.0E-12	--	--	4.6E-11	--	--
Benzo(a)pyrene	5.00	3.79E-09	3.1	NA	8.4E-12	2.6E-11	100.0%	2.0E-11	--	--	3.9E-12	1.2E-11	100.0%	3.0E-11	--	--
Dibenz(a,h)anthracene	0.92	6.97E-10	NA	NA	1.5E-12	--	--	3.6E-12	--	--	7.2E-13	--	--	5.6E-12	--	--
Indeno(1,2,3-cd)pyrene	3.80	2.88E-09	NA	NA	6.4E-12	--	--	1.5E-11	--	--	3.0E-12	--	--	2.3E-11	--	--
					Total ILCR:	2.6E-11	100.0%	HI:	0.0E+00	0.0%	Total ILCR:	1.2E-11	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

COMMERCIAL/UTILITY WORKERS - CURRENT SCENARIO
ACCIDENTAL INGESTION OF SURFACE SOIL -SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

CDI (mg/kg-d)= (Cs*IR*CF*FI*EF*ED)/(BW*AT)
ILCR = CDI*CSFo
HQ = CDI/RfDo

Parameter	Description	On-site Worker	
CDI	Chronic daily intake (mg/kg-d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
IR	Ingestion Rate (mg/d)	50	
CF	Conversion factor (kg/mg)	1.00E-06	
FI	Fraction of soil ingested from site	1	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25,550	
ATn	Averaging time, noncarcinogens (d)	9,125	

				Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	CSFo 1/(mg/kg-d)	RfDo (mg/kg-d)	CDI (mg/kg-d)	ILCR	% Contrib. Total ILCR	CDI (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.4	7.30E-01	NA	5.9E-07	4.3E-07	4.6%	1.7E-06	--	--
Benzo(b)fluoranthene	7.6	7.30E-01	NA	1.3E-06	9.7E-07	10.3%	3.7E-06	--	--
Benzo(a)pyrene	5	7.30E+00	NA	8.7E-07	6.4E-06	67.6%	2.4E-06	--	--
Dibenz(a,h)anthracene	0.92	7.30E+00	NA	1.6E-07	1.2E-06	12.4%	4.5E-07	--	--
Indeno(1,2,3 c,d)pyrene	3.8	7.30E-01	NA	6.6E-07	4.8E-07	5.1%	1.9E-06	--	--
				Total ILCR:	9.4E-06	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

COMMERCIAL/UTILITY WORKERS - CURRENT SCENARIO
DERMAL CONTACT WITH SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

DAD (mg/kg-d)= (Cs*CF*AF*ABS*A*EF*ED)/(BW*AT)
ILCR = CDI*CSFd
HQ = CDI/RfDd

Parameter	Description	On-site Worker	
DAD	Dermally absorbed dose (mg/kg-d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFo	Oral cancer slope factor (1/(mg/kg-d))	CS	
HQ	Hazard quotient	CS	
RfDo	Oral reference dose (mg/kg-d)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
CF	Conversion factor (kg/mg)	1.00E-06	
AF	Soil to skin adherence factor (mg/cm^2-event)	0.2	
ABS	Absorption fraction	CS	
A	Skin surface area available for contact (cm^2)	5,000	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25,550	
ATn	Averaging time, noncarcinogens (d)	9,125	

					Carcinogens			Noncarcinogens		
Parameter	Cs (mg/kg)	ABS	CSFd 1/(mg/kg-d)	RfDd (mg/kg-d)	DAD (mg/kg-d)	ILCR	% Contrib. Total ILCR	DAD (mg/kg-d)	HQ	% Contrib. HI
Benz(a)anthracene	3.4	0.10	7.30E-01	NA	1.2E-06	8.7E-07	4.6%	3.3E-06	--	--
Benzo(b)fluoranthene	7.6	0.10	7.30E-01	NA	2.7E-06	1.9E-06	10.3%	7.4E-06	--	--
Benzo(a)pyrene	5	0.10	7.30E+00	NA	1.7E-06	1.3E-05	67.6%	4.9E-06	--	--
Dibenz(a,h)anthracene	0.92	0.10	7.30E+00	NA	3.2E-07	2.3E-06	12.4%	9.0E-07	--	--
Indeno(1,2,3 c,d)pyrene	3.8	0.10	7.30E-01	NA	1.3E-06	9.7E-07	5.1%	3.7E-06	--	--
					Total ILCR:	1.9E-05	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

COMMERCIAL/UTILITY WORKERS - CURRENT SCENARIO
INHALATION OF FUGITIVE DUSTS EMANATING FROM SURFACE SOIL - SWMU 14
REASONABLE MAXIMUM EXPOSURE
POTENTIAL CARCINOGENIC AND NONCARCINOGENIC RISKS
NAVAL STATION ROOSEVELT ROADS, PUERTO RICO

CDI (mg/kg-d)= $(Ca*IR_h*ET*EF*ED)/(BW*AT)$
Where: Ca = Cs * (1/PEF)

ILCR = CDI*CSFi
HQ = CDI/RfDi

Parameter	Description	On-site Worker	
CDI	Chronic daily intake (mg/kg-d)	CS	(Chemical Specific)
ILCR	Incremental lifetime cancer risk	CS	
CSFi	Inhalation cancer slope factor (1/(mg/kg-d))	CS	
HQ	Hazard quotient	CS	
RfDi	Inhalation reference dose (mg/kg-d)	CS	
Ca	Concentration of chemical in air as fugitive dusts (mg/m^3)	CS	
Cs	Concentration of chemical in soil (mg/kg)	CS	
PEF	Particulate emission factor ($m^{3/}kg$)	1.32E+09	
IR_h	Respiration rate ($m^{3/}hr$)	1.27	
ET	Exposure time (hrs/d)	8	
EF	Exposure Frequency (d/yr)	250	
ED	Exposure Duration (yrs)	25	
BW	Body weight (kg)	70	
ATc	Averaging time, carcinogens (d)	25,550	
ATn	Averaging time, noncarcinogens (d)	9,125	

					Carcinogens			Noncarcinogens		
	Cs	Ca	CSFi	RfDi	CDI		% Contrib.	CDI		% Contrib.
Benz(a)anthracene	3.4	2.58E-09	NA	NA	9.1E-11	NA	--	2.6E-10	--	--
Benzo(b)fluoranthene	7.6	5.76E-09	NA	NA	2.0E-10	--	--	5.7E-10	--	--
Benzo(a)pyrene	5	3.79E-09	3.1	NA	1.3E-10	4.2E-10	100.0%	3.8E-10	--	--
Dibenz(a,h)anthracene	0.92	6.97E-10	NA	NA	2.5E-11	--	--	6.9E-11	--	--
Indeno(1,2,3 c,d)pyrene	3.8	2.88E-09	NA	NA	1.0E-10	--	--	2.9E-10	--	--
					Total ILCR:	4.2E-10	100.0%	HI:	0.0E+00	0.0%

NOTES:
NA - Toxicity criterion not available.
-- Not applicable.

APPENDIX C
TOXICOLOGICAL PROFILES

Benz[a]anthracene

CASRN 56-55-3

Contents

I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0454
Benz[a]anthracene; CASRN 56-55-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Benz[a]anthracene

File On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	09/01/1994

Carcinogenicity Assessment (II.) on-line 03/01/1994

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Benz[a]anthracene
CASRN -- 56-55-3

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Benz[a]anthracene
CASRN -- 56-55-3

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Benz[a]anthracene
CASRN -- 56-55-3
Last Revised -- 03/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m

air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays. Benz[a]anthracene produced tumors in mice exposed by gavage; intraperitoneal, subcutaneous or intramuscular injection; and topical application. Benz[a]anthracene produced mutations in bacteria and in mammalian cells, and transformed mammalian cells in culture.

___II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to benz[a]anthracene to human cancers, benz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984; Lee et al., 1976; Brockhaus and Tomingas, 1976).

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Benz[a]anthracene administration caused an increase in the incidence of tumors by gavage (Klein, 1963); dermal application (IARC, 1973); and both subcutaneous injection (Steiner and Faulk, 1951; Steiner and Edgecomb, 1952) and intraperitoneal injection (Wislocki et al., 1986) assays. A group of male B6AF1/J mice was exposed to gavage solutions containing 3% benz[a]anthracene in Methocel-Aerosol O.T. (dioctyl ester of sodium sulfo-succinic acid), 3 doses/week for 5 weeks (total dose of approximately 225 mg/mouse, 500 mg/kg/day) or the vehicle (Klein, 1963). Mice were evaluated for tumors on days 437-444 and 547 after treatment was initiated. A statistical analysis was not reported. Increased incidences of pulmonary adenoma and hepatoma in treated vs. control mice were reported by the authors at both observation times. The incidence of pulmonary adenoma at 437-444 days was 37/39 (95%) in treated animals vs. 10/38 (26%) in controls; whereas at 547 days, 19/20 (95%) treated animals and 7/20 (35%) controls had pulmonary

adenomas. The incidence of hepatomas at 437 to 440 days was 18/39 (46%) in treated animals compared with 0/38 among the vehicle controls. After 547 days, the hepatoma incidences increased to 20/20 for the treated animals versus 2/20 (10%) for vehicle controls.

Mice (strain and sex not specified) were exposed to a single gavage dose of 0.5 mg benz[a]anthracene in mineral oil (approximately 17 mg/kg). No tumors were reported in 13 mice examined 16 months after exposure. In another part of the study, multiple gavage treatments, 8 or 16 treatments at 3-7 day intervals over a 16-month period, resulted in forestomach papillomas in 2/27 treated mice compared with 0/16 in vehicle controls (Bock and King, 1959).

Groups of male and female CD-1 mice (n=90-100) received intraperitoneal injections of benz[a]anthracene in DMSO on days 1, 8, and 15 of age (total dose = 638 ug/mouse) (Wislocki et al., 1986). Tumors were evaluated in animals that died spontaneously after weaning and in all remaining animals at 1 year after exposure. In treated male mice, a statistically significant increase in the incidence of liver adenomas or carcinomas (31/39 treated vs. 2/28 controls) occurred; 25/39 had carcinomas. Female mice did not develop liver tumors. The incidence of pulmonary adenomas or carcinomas in benz[a]anthracene-treated males (6/39, with a majority of adenomas) was increased but not statistically significantly relative to the vehicle controls (1/28). In the female mice, however, the incidence of pulmonary adenomas was significantly elevated in the treated group (6/32) when compared with vehicle controls (0/31).

Benz[a]anthracene yielded positive results in tests for complete carcinogenicity and initiating activity in skin painting assays in C3H/He, CAF1 and ICR/Ha mouse strains. These studies are reviewed in IARC (1973).

Subcutaneous injection of benz[a]anthracene in tricaprylin into C57Bl mice (40-50/group) produced injection site sarcomas 9 months after treatment (Steiner and Falk, 1951; Steiner and Edgecomb, 1952). The sarcoma incidences were: uninjected controls, 0/76; tricaprylin controls, 3/28 (11%); 0.05 mg, 5/43 (12%); 0.2 mg, 11/43 (26%); 1.0 mg, 15/31 (48%); 5.0 mg, 49/145 (34%); and 10 mg, 5/16 (31%). The results of similar experiments in this series were combined (Steiner and Edgecomb, 1952). A statistical analysis of the results was not reported. Survival was roughly equivalent in all groups (70%).

Klein (1952) showed that an intramuscular injection of benz[a]anthracene in combination with 1 or 3% croton oil produced injection site fibrosarcomas and hemangioendotheliomas in Strain A-derived albino mice; 3/24 mice injected with benz[a]anthracene and 1% croton oil and 1/26 mice injected with benz[a]anthracene and 3% croton oil developed tumors. None of the 30 mice injected with benz[a]anthracene and 0.1% croton oil and none of the 30 mice injected with benz[a]anthracene and 5% croton oil developed tumors. In the control groups none of the 35 mice injected only with 1% croton oil and none of the 32 mice injected only with benz[a]anthracene developed tumors. The survival rate for all groups was roughly equivalent (74%).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

The results of tests for DNA damage in Escherichia coli have not been positive at concentrations of benz[a]anthracene up to 250 ug/mL and 1000 ug/well (Rosenkrantz and Poirier, 1979; DeFlora et al., 1984). Positive results were obtained in tests for reverse mutation in five different strains of Salmonella typhimurium and for forward mutation in one strain (McCann et al., 1975; Coombs et al., 1976; Simmon, 1979; Salamone et al., 1979; Bartsch

et al., 1980; DeFlora et al., 1984; Norpoth et al., 1984; Utesch et al., 1987; Bos et al., 1988; Kaden et al. 1979).

Benz[a]anthracene produced positive results in an assay for mutations in Drosophila melongaster (Fahmy and Fahmy, 1973).

Tests for DNA damage, mutation, chromosomal effects and cell transformation in a variety of eukaryotic cell preparations have yielded mostly positive results. Benz[a]anthracene tested positive for DNA damage in primary rat hepatocytes and HeLa cells (Probst et al., 1981; Martin et al., 1978). It also tested positive for forward mutation in Chinese hamster cells, V79 cells, mouse lymphoma L5178Y cells and rat liver epithelial cells (Slaga et al., 1978; Krahn and Heidelberger, 1977; Amacher et al., 1980; Amacher and Turner, 1980; Tong et al., 1981). Benz[a]anthracene tested positive for chromosomal affects in Chinese hamster ovary cells (Pal, 1981). Tests for cell transformation (cell morphology) have yielded positive results in Syrian hamster embryo cells and mouse prostate C3HG23 cells (Pienta et al., 1977; DiPaolo et al., 1969, 1971; Marquardt and Heidelberger, 1972).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for benz[a]anthracene. Benz[a]anthracene has a "bay-region" structure (Jerina et al., 1978). It is metabolized by mixed function oxidases to reactive "bay-region" diol epoxides that are mutagenic in bacteria and tumorigenic in mouse skin painting assays (Booth and Sims, 1974; Wood et al., 1977a,b).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSUR

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 02/07/1990, 08/05/1993, 09/21/1993, 02/02/1994

Verification Date -- 02/07/1990

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Benz[a]anthracene
CASRN -- 56-55-3
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Benz[a]anthracene

CASRN -- 56-55-3

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
09/01/1993	II.	Carcinogenicity assessment noted as pending change
09/01/1993	II.D.2.	Work group review date added
11/01/1993	II.D.2.	Work group review date added
03/01/1994	II.	Pending change note removed; no change
03/01/1994	II.D.2.	Work group review date added
09/01/1994	I.B.	Inhalation RfC now under review

VIII. SYNONYMS

Substance Name -- Benz[a]anthracene
CASRN -- 56-55-3
Last Revised -- 12/01/1990

56-55-3
Benz(a)anthracene
benz(a)anthracene
Benzanthracene
Benzanthrene
BENZO(a)ANTHRACENE
BENZO(b)PHENANTHRENE
Benzoanthracene
HSDB 4003
NSC 30970
RCRA WASTE NUMBER U018
Tetraphene
1,2-BENZ(a)ANTHRACENE
1,2-Benzanthracene
1,2-BENZANTHRAZEN [German]
1,2-BENZANTHRENE
1,2-BENZOANTHRACENE
2,3-Benzophenanthrene
□

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Last updated: **5 May 1998**
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EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Benzo[b]fluoranthene

CASRN 205-99-2

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VIII. SYNONYMS

0453
Benzo[b]fluoranthene; CASRN 205-99-2

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Benzo[b]fluoranthene

File On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	

Carcinogenicity Assessment (II.) on-line 03/01/1994

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Benzo[b]fluoranthene
CASRN -- 205-99-2

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Benzo[b]fluoranthene
CASRN -- 205-99-2

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Benzo[b]fluoranthene
CASRN -- 205-99-2
Last Revised -- 03/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m

air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays. Benzo[b]fluoranthene produced tumors in mice after lung implantation, intraperitoneal (i.p.) or subcutaneous (s.c.) injection, and skin painting.

___II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to benzo[b]fluoranthene to human cancers, benzo[b]fluoranthene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. In a lifetime implant study, 3-month-old female Osborne-Mendel rats (35/group) received a single lung implant of either 0.1 mg (0.4 mg/kg), 0.3 mg (1.2 mg/kg) or 1 mg (4.1 mg/kg) benzo[b]fluoranthene in 0.05 mL of a 1:1 (v:v) mixture of beeswax and trioctanoin (Deutsch-Wenzel et al., 1983). Controls consisted of an untreated group and a group receiving an implant of the vehicle. The median survival times were: 118, 104, 110, 113 and 112 weeks, for the untreated, vehicle control, low-, mid- and high-dose groups, respectively. The incidences of epidermoid carcinomas and pleomorphic sarcomas in the lung and thorax (combined) were: untreated controls, 0/35; vehicle controls, 0/35; low-dose group, 1/35; mid-dose group, 3/35; and high-dose group, 13/35. These incidences showed a statistically significant dose-response relationship.

Groups of 15-17 male and 17-18 female CD-1 mice received i.p. injections of benzo[b]fluoranthene in DMSO on days 1, 8 and 15 after birth (total dose was approximately 126 ug/mouse) and were sacrificed at 52 weeks of age (LaVoie

et al., 1987). A statistically significant increase in the incidence of liver adenomas and hepatomas (combined) occurred in treated males (8/15) relative to vehicle controls (1/17), but not in females. Lung adenomas (2/15 males, 3/17 females) were reported in treated animals, whereas none were found in controls.

Injection site sarcomas occurred in 18/24 survivors of a total of 16 male and 14 female XVIInc/Z mice that received three s.c. injections of benzo[b]fluoranthene (total dose = 2.6 mg) over a period of 2 months (Lacassagne et al., 1963).

Benzo[b]fluoranthene has yielded positive results for complete carcinogenic activity and initiating activity in mouse skin-painting assays. In skin-painting assays groups of 20 female Swiss mice were treated 3 times/week with 0.01, 0.1 or 0.5% solutions of benzo[b]fluoranthene in acetone (Wynder and Hoffmann, 1959). The high dose produced papillomas in 100% of the mice and carcinomas in 90% of the mice within 8 months. The middle dose produced papillomas in 65% and carcinomas in 85% within 12 months, while the low dose produced a papilloma in only 1 animal among 10 survivors at 14 months. No concurrent controls were observed. LaVoie et al. (1982) applied solutions of 0, 10, 30 or 100 ug benzo[b]fluoranthene in 0.1 mL acetone (10 doses, one every other day) to the skins of groups of 20 Crl:CD-1 mice. This regimen was followed by treatment with 2.5 ug 12-0-tetradecanoyl-phorbol-13-acetone (TPA) (a tumor promoter), 3 times/week for 20 weeks. Increases in the percentage of tumor-bearing animals (0, 45, 60, 80) as well as the number of skin tumors/animal (0, 0.9, 2.3, 7.1) appeared to be dose-related. Similar studies by Amin et al. (1985a,b) resulted in comparable elevations of tumor incidence.

II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Positive results have been reported for a reverse mutation assay in Salmonella TA98 and the results for Salmonella TA100 have been positive and not positive (Mossanda et al., 1979; LaVoie et al., 1979; Hermann, 1981; Amin et al., 1985a,b).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for benzo[b]fluoranthene. Benzo[b]fluoranthene does not have a "classic bay-region" structure (Jerina et al., 1978). It is metabolized by mixed function oxidases to dihydrodiols (Amin et al., 1982). The 9,10-dihydrodiol is tumorigenic in mouse skin-painting assays, suggesting the possible formation of a reactive diol-epoxide (LaVoie et al., 1982).

II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSUR

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984, 1990

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 02/07/1990, 08/05/1993, 09/21/1993, 02/02/1994

Verification Date -- 02/07/1990

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Benzo[b]fluoranthene
CASRN -- 205-99-2
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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LaVoie, E.J., J. Braley, J.E. Rice and A. Rivenson. 1987. Tumorigenic activity for non-alternant polynuclear aromatic hydrocarbons in newborn mice. Cancer Lett. 34: 15-20.

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Wynder, E.L. and D. Hoffmann. 1959. A study of tobacco carcinogenesis. VII. The role of higher polycyclic hydrocarbons. Cancer. 12: 1079-1086.

_VII. REVISION HISTORY

Substance Name -- Benzo[b]fluoranthene
CASRN -- 205-99-2

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
09/01/1993	II.	Carcinogenicity assessment noted as pending change
09/01/1993	II.D.2.	Work group review date added
11/01/1993	II.D.2.	Work group review date added
03/01/1994	II.	Pending change note removed; no change
03/01/1994	II.D.2.	Work group review date added

VIII. SYNONYMS

Substance Name -- Benzo[b]fluoranthene
CASRN -- 205-99-2
Last Revised -- 12/01/1990

205-99-2
Benz(e)acephenanthrylene
B(b)F
BENZ(e)ACEPHENANTHRYLENE
Benzo(b)fluoranthene
Benzo(e)fluoranthene
HSDB 4035
NSC 89265
2,3-BENZFLUORANTHENE
2,3-BENZOFLUORANTHENE
2,3-BENZOFLUORANTHRENE
3,4-BENZ(e)ACEPHENANTHRYLENE
3,4-BENZFLUORANTHENE
3,4-Benzofluoranthene
□

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0453.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Benzo[a]pyrene (BaP)

CASRN 50-32-8

Contents

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VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0136
Benzo[a]pyrene (BaP); CASRN 50-32-8

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR BaP

File On-Line 03/31/1987

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	

Carcinogenicity Assessment (II.) on-line 11/01/1994

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Benzo[a]pyrene (BaP)
CASRN -- 50-32-8

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Benzo[a]pyrene (BaP)
CASRN -- 50-32-8

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Benzo[a]pyrene (BaP)
CASRN -- 50-32-8
Last Revised -- 11/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m

air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

NOTE: At the June 1992 CRAVE Work Group meeting, a revised risk estimate for benzo[a]pyrene was verified (see Additional Comments for Oral Exposure). This section provides information on three aspects of the carcinogenic risk assessment for the agent in question; the U.S. EPA classification, and quantitative estimates of risk from oral exposure and from inhalation exposure. The classification reflects a weight-of-evidence judgment of the likelihood that the agent is a human carcinogen. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000 or 1 in 1,000,000. The Carcinogenicity Background Document provides details on the rationale and methods used to derive the carcinogenicity values found in IRIS. Users are referred to the Oral Reference Dose (RfD) and Reference Concentration (RfC) sections for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Human data specifically linking benzo[a]pyrene (BAP) to a carcinogenic effect are lacking. There are, however, multiple animal studies in many species demonstrating BAP to be carcinogenic following administration by numerous routes. BAP has produced positive results in numerous genotoxicity assays.

___II.A.2. HUMAN CARCINOGENICITY DATA

Inadequate. Lung cancer has been shown to be induced in humans by various mixtures of polycyclic aromatic hydrocarbons known to contain BAP including cigarette smoke, roofing tar and coke oven emissions. It is not possible, however, to conclude from this information that BAP is the responsible agent.

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. The animal data consist of dietary, gavage, inhalation, intratracheal instillation, dermal and subcutaneous studies in numerous strains of at least four species of rodents and several primates. Repeated BAP administration has been associated with increased incidences of total tumors and of tumors at the site of exposure. Distant site tumors have also been observed after BAP administration by various routes. BAP is frequently used as a positive control in carcinogenicity bioassays.

BAP administered in the diet or by gavage to mice, rats and hamsters has produced increased incidences of stomach tumors. Neal and Rigdon (1967) fed BAP (purity not reported) at concentrations of 0, 1, 10, 20, 30, 40, 45, 50, 100 and 250 ppm in the diets of male and female CFW-Swiss mice. The age of the mice ranged from 17-180 days old and the treatment time from 1-197 days; the size of the treated groups ranged from 9 to 73. There were 289 mice (number of mice/sex not stated) in the control group. No forestomach tumors were reported in the 0-, 1- and 10-ppm dose groups. The incidence of forestomach tumors in the 20-, 30-, 40-, 45-, 50-, 100- and 250-ppm dose groups were 1/23, 0/37, 1/40, 4/40, 23/34, 19/23 and 66/73, respectively. The authors felt that the increasing tumor incidences were related to both the concentration and the number of doses administered. Historical control forestomach tumor data are not available for CFW-Swiss strain mice. In historical control data from a related mouse strain, SWR/J Swill, the forestomach tumor incidence rate was 2/268 and 1/402 for males and females, respectively (Rabstein et al., 1973).

Brune et al., (1981) fed 0.15 mg/kg BAP (reported to be "highly pure") in the diet of 32 Sprague-Dawley rats/sex/group either every 9th day or 5 times/week. These treatments resulted in annual average doses of 6 or 39 mg/kg, respectively. An untreated group of 32 rats/sex served as the control. Rats were treated until moribund or dead; survival was similar in all groups. Histologic examinations were performed on each rat. The combined incidence of tumors of the forestomach, esophagus and larynx was 3/64, 3/64 and 10/64 in the control group, the group fed BAP every 9th day and the group fed BAP 5 times/week, respectively. A trend analysis showed a statistically significant tendancy for the proportion of animals with tumors of the forestomach, esophagus or larynx to increase steadily with dose (Knauf and Rice, 1992).

As part of the same study, Brune et al. (1981) administered BAP ("highly pure") orally to Sprague-Dawley rats by caffeine gavage. The rats were treated until moribund or dead; all rats were subjected to terminal histopathologic examination. Gavaged rats were divided into 3 dose groups of 32 rats/sex/group; the groups received 0.15 mg/kg per gavage either every 9th day (Group A), every 3rd day (Group B) or 5 times per week (Group C); these treatments resulted in annual average doses of 6, 18 or 39 mg/kg, respectively. Untreated and gavage (5 times/week) controls (32 rats/sex/group) were included. The median survival times for the untreated control group; the gavage control group; and groups A, B and C were 129, 102, 112, 113 and 87 weeks, respectively. The survival time of Group C was short compared with controls and may have precluded tumor formation (Knauf and Rice, 1992). The combined tumor incidence in the forestomach, esophagus and larynx was 3/64, 6/64, 13/64, 26/64 and 14/64 for the untreated control group, gavage control group, group A, group B and group C, respectively. There was a statistically significant association between the dose and the proportions of rats with tumors of the forestomach, esophagus or larynx. This association is not characterized by a linear trend. The linearity was affected by the apparently reduced tumor incidence that is seen in the high-dose group (Knauf and Rice, 1992).

Intratracheal instillation and inhalation studies in guinea pigs, hamsters and rats have resulted in elevated incidences of respiratory tract and upper digestive tract tumors (U.S. EPA, 1991a). Male Syrian golden hamsters (24/group) were exposed by inhalation to 0, 2.2, 9.5 or 46.5 mg BAP/cu.m in a sodium chloride aerosol (Thyssen et al., 1981). (Greater than 99% of the particles had diameters between 0.2 and 0.5 um.) For the first 10 weeks of the study, the hamsters were exposed to BAP daily for 4.5 hours/day; thereafter, daily for 3 hours/day. Animals dying within the first year of the study were replaced; the effective number of hamsters in the control, low-, mid- and high-dose groups was 27, 27, 26 and 25, respectively. (The total time of treatment, although over 60 weeks, was not stated.) During the first 10 weeks, animals in the 3 dose groups reportedly lost weight. After week 10, however, the body weights in all groups were similar until week 60 when the body weights of hamsters in the high-dose group decreased and the mortality increased significantly. The incidence of respiratory tract tumors (including tumors of the nasal cavity, larynx and trachea) in the control, low-, mid- and high-dose groups was 0/27, 0/27, 9/26 and 13/25, respectively; the incidences of upper digestive tract tumors (including tumors of the pharynx, esophagus and forestomach) were 0/27, 0/27, 7/26 and 14/25, respectively. Trend analysis for incidences of both respiratory tract tumors and upper gastrointestinal tract tumors showed a statistically significant tendancy for the proportion of animals with either tumor type to increase steadily with increased dose (Knauf and Rice, 1992).

Intraperitoneal BAP injections have caused increases in the number of injection site tumors in mice and rats (reviewed in U.S. EPA, 1991a). Subcutaneous BAP injections have caused increases in the number of injection site tumors in mice, rats, guinea pigs, hamsters and some primates (IARC, 1983; U.S. EPA, 1991a). BAP is commonly used as a positive control in many dermal application bioassays and has been shown to cause skin tumors in mice, rats, rabbits and guinea pigs. BAP is both an initiator and a complete carcinogen in mouse skin (IARC, 1983). Increased incidences of distant site tumors have also been reported in animals as a consequence of dermal BAP exposure (reviewed in U.S. EPA, 1991a).

BAP has also been reported to be carcinogenic in animals when administered by the following routes: i.v.; transplacentally; implantation in the stomach wall, lung, renal parenchyma and brain; injection into the renal pelvis; and vaginal painting (U.S. EPA, 1991a).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Benzo[a]pyrene has been shown to cause genotoxic effects in a broad range of prokaryotic and mammalian cell assay systems (U.S. EPA, 1991a). In prokaryotes, BAP tested positive in DNA damage assays and in both reverse and forward mutation assays. In mammalian cell culture assays, BAP tested positive in DNA damage assays, forward mutation assays, chromosomal effects assays and cell transformation assays.

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSUR

NOTE: The range of oral slope factors calculated was: 4.5E+0 to 11.7E+0 per (mg/kg)/day.

___II.B.1. SUMMARY OF RISK ESTIMATES

Oral Slope Factor -- 7.3E+0 per (mg/kg)/day

Drinking Water Unit Risk -- 2.1E-4 per (ug/L)

Extrapolation Method -- Risk estimate based on a geometric mean of four slope factors obtained by differing modeling procedures. Derived from the combination of multiple data sets from two different reports using more than one sex and species.

Drinking Water Concentrations at Specified Risk Levels:

Risk Level	Concentration
E-4 (1 in 10,000)	5E-1 ug/L
E-5 (1 in 100,000)	5E-2 ug/L
E-6 (1 in 1,000,000)	5E-3 ug/L

___II.B.2. DOSE-RESPONSE DATA (CARCINOGENICITY, ORAL EXPOSURE)

Tumor Type -- forestomach, squamous cell papillomas and carcinomas
Test Animals -- CFW mice, sex unknown
Route -- oral, diet
Reference -- Neal and Rigdon, 1967

a) Conditional upper bound two-stage model with terms for promotion (modification of Moolgavkar-Venson-Knudson, generalized forms of two-stage model)

Administered Dose (ppm)	Tumor Incidence
0	0/289
1	0/25
10	0/24
20	1/23
30	0/37
40	1/40
45	4/40
50	24/34
100	19/23
250	66/73

Tumor Type -- squamous cell carcinoma of the forestomach
Test Animals -- SWR/J Swill mice
Route -- oral, diet
Reference -- Rabstein et al., 1973

Administered Dose (ppm)	Tumor Incidence

0	2/268* male
0	1/402* female

*See additional comments concerning the use of control data from other studies that utilized similar mouse strains.

b) Same data as above. Upper bound estimate by extrapolation from 10% response point to background of empirically fitted dose-response curve. (Procedure using two-stage model described in (a)).

c) Same data as above except the additional 2 control groups (Rabstein et al., 1973) were excluded. Generalized Weibull-type dose-response model.

d) Tumor Type -- forestomach, larynx and esophagus, papillomas and carcinomas (combined). Linearized Multistage Model, Extra Risk.

Test Animals -- Sprague-Dawley rats, males and females
Route -- oral, diet
Reference -- Brune et al., 1981

Dose (mg/kg diet/year)	Tumor Incidence
0	3/64
6	3/64
39	10/64

___II.B.3. ADDITIONAL COMMENTS (CARCINOGENICITY, ORAL EXPOSURE)

At the June 1992 CRAVE Work Group meeting, it was noted that an error had been made in the 1991 document "Dose-Response Analysis of Ingested Benzo[a]pyrene" which is quoted in the Drinking Water Criteria Document for PAH. In the calculation of the doses in the Brune et al. (1981) study it was erroneously concluded that doses were given in units of mg/year, whereas it was in fact mg/kg/year. When the doses are corrected the slope factor is correctly calculated as 11.7 per (mg/kg)/day, as opposed to 4.7 per (mg/kg)/day as reported in the Drinking Water Criteria Document. The correct range of slope factors is 4.5 to 11.7 per (mg/kg)/day, with a geometric mean of 7.3 per (mg/kg)/day. A drinking water unit risk based on the revised slope factor is 2.1E-4 per (ug/L). Therefore, these values have been changed on IRIS and an Erratum to the Drinking Water Criteria Document is being prepared.

Risk estimates were calculated from two different studies in two species of outbred rodents (Neal and Rigdon, 1967; Brune et al., 1981). These studies have several commonalities including mode of administration, tumor sites, tumor types and the presumed mechanisms of action. The data sets were not combined prior to modeling (the preferred approach) because they employed significantly dissimilar protocols.

The geometric mean from several slope factors, each considered to be of equal merit, was used to calculate a single unit risk. These four slope factor estimates span less than a factor of three and each is based on an acceptable, but less-than-optimal, data set. Each estimate is based on a low-dose extrapolation procedure which entails the use of multiple assumptions and default procedures.

Clement Associates (1990) fit the Neal and Rigdon (1967) data to a two-

stage dose response model. In this model the transition rates and the growth rate of preneoplastic cells were both considered to be exposure-dependent. (The functional form for the dose-dependence of preneoplastic cell growth rate was simple saturation.) A term to permit the modeling of BAP as its own promoter was also included. Historical control stomach tumor data from a related, but not identical, mouse strain, SWR/J Swill (Rabstein et al., 1973) and the CFW Texas colony (Neal and Rigdon, 1967) were used in the modeling. In calculating the lifetime unit risk for humans several standard assumptions were made: mouse food consumption was 13% of its body weight/day; human body weight was assumed to be 70 kg and the assumed body weight of the mouse 0.034 kg. The standard assumption of surface area equivalence between mice and humans was the cube root of 70/0.034. A conditional upper bound estimate was calculated to be 5.9 per (mg/kg)/day (U.S. EPA, 1991a).

A U.S. EPA report (1991b) argued that the upper-bound estimate calculated in Clement Associates (1990) involved the use of unrealistic conditions placed on certain parameters of the equation. Other objections to this slope factor were also raised. The authors of this report used the Neal and Rigdon (1967) data to generate an upper-bound estimate extrapolated linearly from the 10% response point to the background of an empirically fitted dose-response curve (Clement Associates, 1990). Other results, from similar concepts and approaches used for other compounds, suggest that the potency slopes calculated in this manner are comparable to those obtained from a linearized multistage procedure for the majority of the other compounds. The upper bound estimate calculated in U.S. EPA (1991b) is 9.0 per (mg/kg)/day.

The authors of U.S. EPA (1991b) selected a model to reflect the partial lifetime exposure pattern over different parts of the animals' lifetimes. The authors thought that this approach more closely reflected the Neal and Rigdon (1967) regimen. A Weibull-type dose-response model was selected to accommodate the partial lifetime exposure; the upper-bound slope factor calculated from this method was 4.5 per (mg/kg)/day.

Using the dietary portion of the Brune et al. (1981) rat data, a linearized multistage procedure was used to calculate an upper bound slope factor for humans. In the interspecies conversion the assumed human body weight was 70 kg and the rat 0.4 kg. The slope factor calculated by this method was 11.7 per (mg/kg)/day.

___II.B.4. DISCUSSION OF CONFIDENCE (CARCINOGENICITY, ORAL EXPOSURE)

The data are considered to be less than optimal, but acceptable. There are precedents for using multiple data sets from different studies using more than one sex, strain and species; the use of the geometric mean of four slope factors is preferred because it makes use of more of the available data. The use of the geometric means was based on arguments presented in a personal communication (Stiteler, 1991).

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1991a,b

The 1991 Drinking Water Criteria Document for the polycyclic aromatic hydrocarbons has received agency review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 01/07/1987, 12/04/1991, 06/03/1992, 08/05/1993, 02/02 06/09/1994

Verification Date -- 12/04/1991

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Benzo[a]pyrene (BaP)
CASRN -- 50-32-8
Last Revised -- 12/01/1993

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfD REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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_VII. REVISION HISTORY

Substance Name -- Benzo[a]pyrene (BaP)
CASRN -- 50-32-8

Date	Section	Description
08/01/1989	VI.	Bibliography on-line
01/01/1992	II.	Carcinogen assessment noted as pending change
01/01/1992	IV.	Regulatory actions updated
04/01/1992	II.	Summary revised; oral quantitative section added
04/01/1992	VI.C.	Carcinogen assessment references revised
05/01/1992	II.D.2.	Work group review and verification date corrected
07/01/1992	II.	Text revised in NOTE
07/01/1992	II.B.	Range of slope factors corrected
07/01/1992	II.B.1.	Slope factor and risks corrected
07/01/1992	II.B.2.	Data table heading corrected
07/01/1992	II.B.3.	Slope factor corrected; last paragraph
07/01/1992	II.D.3.	Secondary contact changed
09/01/1993	II.	Carcinogenicity assessment noted as pending change
09/01/1993	II.D.2.	Work group review date added
12/01/1993	VI.C.	Reference revised - U.S. EPA, 1991b
02/01/1994	II.D.3.	Primary contact's phone number changed
03/01/1994	II.	Pending change note removed; no change
03/01/1994	II.D.2.	Work group review date added
07/01/1994	II.D.2.	Work group review date added
11/01/1994	II.B.1.	Slope factor clarified; changed O to "0"

VIII. SYNONYMS

Substance Name -- Benzo[a]pyrene (BaP)
CASRN -- 50-32-8
Last Revised -- 03/31/1987

50-32-8
BaP
Benzo[a]pyrene
BENZO(d,e,f)CHRYSENE
3,4-BENZOPIRENE
3,4-BENZOPYRENE
6,7-BENZOPYRENE
BENZO(a)PYRENE
3,4-BENZPYREN
3,4-BENZPYRENE
3,4-BENZ(a)PYRENE
BENZ(a)PYRENE
3,4-BENZYPYRENE

BP
3,4-BP
B(a)P
RCRA WASTE NUMBER U022
□

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Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0136.htm

Dibenz[a,h]anthracene

CASRN 53-70-3

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I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (RfC)

II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0456
Dibenz[a,h]anthracene; CASRN 53-70-3

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Dibenz[a,h]anthracene

File On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	

Carcinogenicity Assessment (II.) on-line 03/01/1994

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3
Last Revised -- 03/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m

air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2; probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays. Dibenz[a,h]anthracene produced carcinomas in mice following oral or dermal exposure and injection site tumors in several species following subcutaneous or intramuscular administration. Dibenz[a,h]anthracene has induced DNA damage and gene mutations in bacteria as well as gene mutations and transformation in several types of mammalian cell cultures.

___II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to dibenz[a,h]anthracene with human cancers, dibenz[a]anthracene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. Dibenz[a,h]anthracene has been shown to be carcinogenic when administered to mice by the oral route (Snell and Stewart, 1962, 1963). Instead of drinking water DBA/2 mice (21/sex) were given a water-olive oil emulsion containing 0.2 mg/mL dibenz[a,h]anthracene ad libitum. Average exposure was estimated to be 0.85 mg/day for males and 0.76 mg/day for females. The control groups (25 male and 10 female) received the water-olive oil emulsion in place of water. The mice did not tolerate the olive oil vehicle well and all 4 groups lost weight after a few weeks exposure and eventually became emaciated and dehydrated. Animals that died spontaneously or that became moribund were examined for tumors. The duration of the experiment was 279 and 237 days for males and females, respectively, in the dosed groups and 351 and 226 days for male and female controls. Mice developed pulmonary adenomas (treated males, 14/14; control males 1/23;

treated females, 13/13; control females, 0/6), pulmonary carcinomas (treated males, 14/14; control males, 0/23; treated females, 10/13; control females, 0/6), mammary carcinoma (treated females, 12/13; control females, 0/6) and hemangioendothelioma (treated males, 10/14; control males, 0/23; treated females, 6/13; control females, 0/6). No statistical analyses appear to have been performed.

Mammary carcinomas were observed in two strains of female mice following gavage with dibenz[a,h]anthracene (Biancifiori and Caschera, 1962; Berenblum and Haran, 1955). Biancifiori and Caschera (1962) observed mammary carcinomas when female Balb/c (1/20) and pseudo-pregnant female (obtained by mating virgin females with vasectomized males) Balb/c (13/24) mice were treated for 15 weeks with a twice-weekly gavage containing 0.5% dibenz[a,h]anthracene (total dose was 15 mg/animal). Mammary carcinomas occurred in 2/30 pseudo-pregnant females not dosed with dibenz[a,h]anthracene. Previous studies indicated that mammary carcinomas did not occur in virgin Balb/c females (Biancifiori et al., 1959). A single 1.5-mg dose of dibenz[a,h]anthracene in polyethylene glycol [average molecular weight (a.m.u.) 400] (PEG-400) produced forestomach papillomas in 2/42 male Swiss mice after 30 weeks. In this short-term study no mice developed tumors when treated with PEG alone (1 time/week) for 30 weeks (0/20) (Berenblum and Haran, 1955).

Dibenz[a,h]anthracene has produced positive results in mouse skin painting assays for complete carcinogenicity. Swiss mice developed carcinomas following dermal exposure to dibenz[a,h]anthracene at concentrations of 0.001% or greater (Wynder and Hoffman, 1959; Van Duuren et al., 1967). Numerous studies that demonstrate complete carcinogenic activity and initiating activity are summarized in IARC (1973) and U.S. EPA (1990).

Subcutaneous injection of dibenz[a,h]anthracene induced sarcomas at the site of injection in several animal species. Groups (>19) of C3H mice received single subcutaneous injections of dibenz[a,h]anthracene in tricaprylin at doses ranging from 0.0019-8 mg (approximately 0.09-360 mg/kg). No controls appear to have been used in this experiment (Bryan and Shimkin, 1943). Tumor latency appeared to decrease and the incidence of injection site sarcomas appeared to increase with dose (>76% at doses >0.06 mg or 2.8 mg/kg). A single subcutaneous injection of 2.4, 4.7, 9.3, 18.7, 37.5, or 75 ug dibenz[a,h]anthracene into groups of 100 NMRI mice was reported to produce a dose-related increase in tumor incidence (37/100, 39/100, 44/100, 56/100, 65/100, and 69/100, respectively) by the 114th week after injection (Pfeiffer, 1977). No concurrent controls were reported; however, a spontaneous tumor rate for NMRI mice was previously reported to be 0-2% (Pfeiffer, 1973). The development of fibrosarcomas from a single subcutaneous injection of 150 ug dibenz[a,h]anthracene was shown to be higher in AHH+ strains of mice than in AHH- strains.

Lubet et al. (1983) found that subcutaneous injections of dibenz[a,h]anthracene were associated with fibrosarcoma development in mice, but only for some strains. Four strains of mice used included two, C3H/HeJ and C57B1/6J, that respond to 3-methylcholanthrene treatment with increased levels and types of hepatic enzymes, including AHH. Two strains, AKR/J and DBA/2J were nonresponders. Groups of 30 animals were injected with a single dose of 150 mg dibenz[a,h]anthracene in 0.05 mL trioctanoin and observed for 9 months. A control group for each strain, consisting of 10 animals each, received a subcutaneous injection of 0.05 mL trioctanoin alone. The tumor incidence in the treated animals varied between 0 and 80%, depending on the strain. Tumor incidences were higher in the C3H and C57B1 mice but not in AKR or DBA mice. Likewise, the average latency period (in days) for fibrosarcoma development varied with the strain and tended to be inversely correlated with the tumor incidence rate. Numerous earlier studies that demonstrate the carcinogenicity of parenterally injected dibenz[a,h]anthracene in a variety of species are summarized in IARC (1973) and U.S. EPA (1990).

__II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Dibenz[a,h]anthracene has produced positive results in bacterial DNA damage and mutagenicity assays and in mammalian cell DNA damage, mutagenicity and cell transformation assays. In bacterial DNA damage assays, positive results were obtained in Escherichia coli and Bacillus subtilis at exposure levels of 12-50 ug/well. Dibenz[a,h]anthracene tested positive for reverse mutation in Salmonella typhimurium strains TA100 and TA98 (3-5 ug/plate) and positive for forward mutation in strain TM677 (21 ug/mL) (McCann et al., 1975; Andrews et al., 1978; Baker et al., 1980; Hermann, 1981; Kaden et al., 1979). In mammalian cell DNA damage assays, positve results were obtained in human foreskin epithelial cells not activated with mixed-function oxidase (MFO) inducers (1-100 ug/mL) and in HeLa cells (28 ng/mL) activated with 3-methylcholanthrene (Lake et al., 1978; Martin et al., 1978). When Syrian hamster embryo cells and rat hepatocytes not activated with MFO inducers were exposed to 20-30 ug/mL the results were not positive (Casto, 1979; Probst et al., 1981). Dibenz[a,h]anthracene induced forward mutations in Chinese hamster embryo cells exposed to concentrations of 1 ug/mL or greater (Huberman and Sachs, 1976; Krahn and Heidelberger, 1977; Huberman, 1978). It transformed several types of mammalian cells exposed to concentrations of 10 ug/mL or greater; these cell types included: Syrian hamster embryo cells, mouse C3H10T 1/2 cells and mouse prostate C3H cells (DiPaolo et al., 1969; Chen and Heidelberger, 1969; Pienta et al., 1977; Casto et al., 1977; Casto, 1979; Reznikoff et al., 1973; Lubet et al., 1983).

Current theories on mechanisms of metabolic activation of polycyclic aromatic hydrocarbons are consistent with a carcinogenic potential for dibenz[a,h]anthracene. Dibenz[a,h]anthracene has a "bay-region" structure (Jerina et al., 1978). It is metabolized by mixed-function oxidases to dihydrodiols that are mutagenic in bacteria and tumorigenic in mouse skin painting assays and when injected into newborn mice (Wood et al., 1978; Nordqvist et al., 1979; Slaga et al., 1980; Buening et al., 1979).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSUR

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984, 1990

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 02/07/1990, 08/05/1993, 09/21/1993, 02/02/1994

Verification Date -- 02/07/1990

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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U.S. EPA. 1990. Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons (PAHs). Prepared by the Office of Health and Environmental Assessment, Environmental Criteria and Assessment Office, Cincinnati, OH for the Office of Drinking Water, Washington, DC. Final Draft. ECAO-CIN-D010, September, 1990.

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_VII. REVISION HISTORY

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3

Date	Section	Description
12/01/1990	II.	Carcinogen assessment on-line
12/01/1990	VI.	Bibliography on-line
01/01/1992	IV.	Regulatory Action section on-line
09/01/1993	II.	Carcinogenicity assessment noted as pending change
09/01/1993	II.D.2.	Work group review date added
11/01/1993	II.D.2.	Work group review date added
03/01/1994	II.	Pending change note removed; no change
03/01/1994	II.D.2.	Work group review date added

VIII. SYNONYMS

Substance Name -- Dibenz[a,h]anthracene
CASRN -- 53-70-3
Last Revised -- 12/01/1990

53-70-3
Dibenz(a,h)anthracene
DB(a,h)A
DBA
dibenz(a,h)anthracene
DIBENZO(a,h)ANTHRACENE
HSDB 5097
NSC 22433
RCRA WASTE NUMBER U063
1,2,5,6-DIBENZANTHRACEEN [Dutch]
1,2,5,6-dibenzanthracene
1,2:5,6-BENZANTHRACENE
1,2:5,6-DIBENZ(a)ANTHRACENE
1,2:5,6-Dibenzanthracene
1,2:5,6-DIBENZOANTHRACENE
□

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EPA Home Page

Last updated: **5 May 1998**
URL: http://www.epa.gov/iris/subst/0456.htm

EPA United States Environmental Protection Agency

IRIS Integrated Risk Information System

Indeno[1,2,3-cd]pyrene

CASRN 193-39-5

Contents

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VI. BIBLIOGRAPHY

VII. REVISION HISTORY

VIII. SYNONYMS

0457
Indeno[1,2,3-cd]pyrene; CASRN 193-39-5

Health assessment information on a chemical substance is included in IRIS only after a comprehensive review of chronic toxicity data by U.S. EPA health scientists from several Program Offices and the Office of Research and Development. The summaries presented in Sections I and II represent a consensus reached in the review process. Background information and explanations of the methods used to derive the values given in IRIS are provided in the Background Documents.

STATUS OF DATA FOR Indeno[1,2,3-cd]pyrene

File On-Line 12/01/1990

Category (section)	Status	Last Revised
Oral RfD Assessment (I.A.)	no data	
Inhalation RfC Assessment (I.B.)	no data	

Carcinogenicity Assessment (II.) on-line 03/01/1994

_I. CHRONIC HEALTH HAZARD ASSESSMENTS FOR NONCARCINOGENIC EFFECTS

__I.A. REFERENCE DOSE FOR CHRONIC ORAL EXPOSURE (RfD)

Substance Name -- Indeno[1,2,3-cd]pyrene
CASRN -- 193-39-5

Not available at this time.

__I.B. REFERENCE CONCENTRATION FOR CHRONIC INHALATION EXPOSURE (Rf

Substance Name -- Indeno[1,2,3-cd]pyrene
CASRN -- 193-39-5

Not available at this time.

_II. CARCINOGENICITY ASSESSMENT FOR LIFETIME EXPOSURE

Substance Name -- Indeno[1,2,3-cd]pyrene
CASRN -- 193-39-5
Last Revised -- 03/01/1994

Section II provides information on three aspects of the carcinogenic assessment for the substance in question; the weight-of-evidence judgment of the likelihood that the substance is a human carcinogen, and quantitative estimates of risk from oral exposure and from inhalation exposure. The quantitative risk estimates are presented in three ways. The slope factor is the result of application of a low-dose extrapolation procedure and is presented as the risk per (mg/kg)/day. The unit risk is the quantitative estimate in terms of either risk per ug/L drinking water or risk per ug/cu.m

air breathed. The third form in which risk is presented is a drinking water or air concentration providing cancer risks of 1 in 10,000, 1 in 100,000 or 1 in 1,000,000. The rationale and methods used to develop the carcinogenicity information in IRIS are described in The Risk Assessment Guidelines of 1986 (EPA/600/8-87/045) and in the IRIS Background Document. IRIS summaries developed since the publication of EPA's more recent Proposed Guidelines for Carcinogen Risk Assessment also utilize those Guidelines where indicated (Federal Register 61(79):17960-18011, April 23, 1996). Users are referred to Section I of this IRIS file for information on long-term toxic effects other than carcinogenicity.

__II.A. EVIDENCE FOR CLASSIFICATION AS TO HUMAN CARCINOGENICITY

___II.A.1. WEIGHT-OF-EVIDENCE CLASSIFICATION

Classification -- B2, probable human carcinogen

Basis -- Based on no human data and sufficient data from animal bioassays. Indeno[1,2,3-cd]pyrene produced tumors in mice following lung implants, subcutaneous injection and dermal exposure. Indeno[1,2,3-cd]pyrene tested positive in bacterial gene mutation assays.

___II.A.2. HUMAN CARCINOGENICITY DATA

None. Although there are no human data that specifically link exposure to indeno[1,2,3-cd]pyrene to human cancers, indeno[1,2,3-cd]pyrene is a component of mixtures that have been associated with human cancer. These include coal tar, soots, coke oven emissions and cigarette smoke (U.S. EPA, 1984, 1990; IARC, 1984).

___II.A.3. ANIMAL CARCINOGENICITY DATA

Sufficient. In carcinogen bioassays indeno[1,2,3-cd]pyrene exposure resulted in increased incidences of epidermoid carcinomas in a lung implantation study (Deutsch-Wenzel et al., 1983), injection site sarcomas in a subcutaneous injection assay (Lacassagne et al., 1963) and skin tumors in dermal application studies (Hoffman and Wynder, 1966; Rice et al., 1985a, 1986).

In a lifetime implant study, 3-month-old female Osborne-Mendel rats (35/group) received lung implants of indeno[1,2,3-cd]pyrene in 0.05 mL of a 1:1 (v:v) mixture of beeswax and trioctanoin (Deutsch-Wenzel et al., 1983). Rats received either 0.16 mg (0.65 mg/kg), 0.83 mg (3.4 mg/kg) or 4.15 mg (17 mg/kg) indeno[1,2,3-cd]pyrene. Controls consisted of an untreated group and a group receiving an implant of the vehicle. Median survival times in weeks were as follows: untreated controls, 118; vehicle controls, 104; low-dose, 116; mid-dose, 109; and high-dose, 92. Incidence of epidermoid carcinomas in

the lung and thorax (combined) showed a statistically significant dose-related increase. The incidences were: untreated controls, 0/35; vehicle controls, 0/35; low-dose, 4/35 (11%); mid-dose, 8/35 (23%); and high-dose, 21/35 (60%).

Groups of male and female CD-1 mice (n=32) received intraperitoneal injections of indeno[1,2,3-cd]pyrene in dimethyl sulfoxide (DMSO) on days 1, 8 and 15 after birth (total dose = 580 ug/mouse) and were evaluated for tumors upon sacrifice at 52 weeks of age (LaVoie et al., 1987). One male mouse (1/11) developed a lung adenoma, no tumors occurred in female mice. Tumor incidence was not significantly different from vehicle controls. This test is considered to be a short-term lung tumor assay.

In mouse skin painting assays, indeno[1,2,3-cd]pyrene tested positive for cancer-initiating activity in several mouse strains (Hoffmann and Wynder, 1966; Rice et al., 1985a, 1986). In the Hoffmann and Wynder (1966) study female Swiss albino Ha/ICR/Mil mice (20/group) were given topical applications of indeno[1,2,3-cd]pyrene prepared as dioxane (at 0.05 and 0.1%) or in acetone solutions (at 0.01, 0.05 and 0.1%). Dioxane preparations did not induce skin tumors. By contrast, acetone solutions of indeno[1,2,3-cd]pyrene produced skin tumors in a dose-related fashion. No tumors were observed in animals painted with 0.01 or 0.05% indeno[1,2,3-cd]pyrene in acetone; 0.1% induced six papillomas and three carcinomas beginning at 9 months; and 0.5% resulted in seven papillomas and five carcinomas with the first tumor appearing at 3 months. The authors also reported that a total dose of 250 mg indeno[1,2,3-cd]pyrene delivered in 10 applications in 2 days was a sufficient initiating dose when followed by promotion with croton oil.

To examine the initiating capability of the compound's major metabolites in mouse skin, indeno[1,2,3-cd]pyrene was applied to the shaved backs of 20 Crl:CD-1(ICR)BR female mice (Rice et al., 1986). Acetone solutions were applied every other day for 10 days for a total initiating dose of 1 mg indeno[1,2,3-cd]pyrene. This was followed 10 days later by applications of the promotor tetradecanoylphorbol (TPA) (0.0025% in 100 mL acetone) 3 times/week for 20 weeks. Tumor incidence was essentially 100%. Indeno[1,2,3-cd]pyrene-1,2-diol and -1,2-oxide treatment both resulted in 80% tumor incidence in contrast to 8-hydroxy- and acetone-treated controls (approximately 25 and 5%, respectively).

An earlier initiation-promotion bioassay performed by Rice et al. (1985a) showed a pronounced dose-response relationship for tumors. Following the same protocol described above, an 80% tumor incidence was observed in mice receiving a total initiating dose of 1 mg indeno[1,2,3-cd]pyrene with an average of about four tumors/mouse after 22 weeks of promotion. However, when the total initiating dose was decreased to 100 or 300 mg/mouse, the number of tumor-bearing mice was not significantly increased.

Injection site sarcomas were reported in 10/14 male and 1/14 female XVIIc/Z mice administered 3 injections at 1-month intervals of 0.6 mg indeno[1,2,3-cd]pyrene. No concurrent controls appear to have been run in this experiment; the authors report, however, that in this mouse strain no spontaneous subcutaneous tumors have been reported (Lacassagne et al., 1963).

___II.A.4. SUPPORTING DATA FOR CARCINOGENICITY

Indeno[1,2,3-cd]pyrene produced positive results in reverse mutation assays in Salmonella typhimurium strains TA100 and TA98 (2-3 ug/plate) (LaVoie et al., 1979; Hermann et al., 1980; Rice et al., 1985b).

__II.B. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM ORAL EXPOSUR

Not available.

__II.C. QUANTITATIVE ESTIMATE OF CARCINOGENIC RISK FROM INHALATION E

Not available.

__II.D. EPA DOCUMENTATION, REVIEW, AND CONTACTS (CARCINOGENICITY ASS

___II.D.1. EPA DOCUMENTATION

Source Document -- U.S. EPA, 1984, 1990

The 1990 Drinking Water Criteria Document for Polycyclic Aromatic Hydrocarbons has received Agency and external review.

___II.D.2. REVIEW (CARCINOGENICITY ASSESSMENT)

Agency Work Group Review -- 02/07/1990, 08/05/1993, 09/21/1993, 02/02/1994

Verification Date -- 02/07/1990

___II.D.3. U.S. EPA CONTACTS (CARCINOGENICITY ASSESSMENT)

Please contact the Risk Information Hotline for all questions concerning this assessment or IRIS, in general, at (513)569-7254 (phone), (513)569-7159 (FAX) or RIH.IRIS@EPAMAIL.EPA.GOV (internet address).

_VI. BIBLIOGRAPHY

Substance Name -- Indeno[1,2,3-cd]pyrene
CASRN -- 193-39-5
Last Revised -- 12/01/1990

__VI.A. ORAL RfD REFERENCES

None

__VI.B. INHALATION RfC REFERENCES

None

__VI.C. CARCINOGENICITY ASSESSMENT REFERENCES

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VII. REVISION HISTORY

Substance Name -- Indeno[1,2,3-cd]pyrene
CASRN -- 193-39-5

Date	Section	Description
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12/01/1990	VI.	Bibliography on-line
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09/01/1993	II.	Carcinogenicity assessment noted as pending change
09/01/1993	II.D.2.	Work group review date added
11/01/1993	II.D.2.	Work group review date added
03/01/1994	II.	Pending change note removed; no change
03/01/1994	II.D.2.	Work group review date added

VIII. SYNONYMS

Substance Name -- Indeno[1,2,3-cd]pyrene
CASRN -- 193-39-5
Last Revised -- 12/01/1990

193-39-5
Indeno(1,2,3-cd)pyrene
HSDB 5101
indeno(1,2,3-cd)pyrene
o-PHENYLENEPYRENE
RCRA WASTE NUMBER U137
1,10-(O-PHENYLENE)PYRENE
1,10-(1,2-Phenylene)pyrene
2,3-o-PHENYLENEPYRENE
2,3-PHENYLENEPYRENE
□

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