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Environmental Hygiene  
Agency



FINAL REPORT  
KUWAIT OIL FIRE HEALTH RISK ASSESSMENT  
- No. 39-26-1192-91  
6 MAY - 3 DECEMBER 1991



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DEPARTMENT OF THE ARMY  
U. S. ARMY ENVIRONMENTAL HYGIENE AGENCY  
ABERDEEN PROVING GROUND. MARYLAND 21010-5422



REPLY TO  
ATTENTION OF

18 FEB 1994

HSHB-ME-S (40)

MEMORANDUM FOR HQDA(SGPS-PSP), 5109 Leesburg Pike, Falls Church,  
VA 22041-3258

SUBJECT: Final Report, Kuwait Oil Fire Health Risk Assessment  
No. 39-26-L192-91, 5 May - 3 December 1991

Copies of subject report with Executive Summary are enclosed.

Encl

*James R. Wiles*  
JAMES R. WILES  
Colonel, MS  
Commanding





DEPARTMENT OF THE ARMY  
U. S. ARMY ENVIRONMENTAL HYGIENE AGENCY  
ABERDEEN PROVING GROUND, MARYLAND 21010-5422



REPLY TO  
ATTENTION OF

**EXECUTIVE SUMMARY**  
FINAL REPORT  
KUWAIT OIL **FIRE** HEALTH RISK ASSESSMENT  
NO. 39-26-L192-91  
5 MAY - 3 DECEMBER 1991

I. PURPOSE. This health risk assessment (**HRA**) was conducted to characterize both the carcinogenic and noncarcinogenic health risks to Department of Defense (**DD**) troops and DD civilian employees exposed to the environment affected by the oil fires during and after Operation Desert Storm. The U.S. Army Environmental Hygiene Agency (**USAEHA**) deployed a team of physicians, scientists, and engineers to the Persian Gulf to establish monitoring stations in both Kuwait and Saudi Arabia where large U.S. military forces were located. Of the 605 oil wells that were initially ignited, 558 (92%) were still burning when the **USAEHA** sampling effort started. The HRA, employing U.S. Environmental Protection Agency (EPA) methodology, predicts population effects based on measured environmental concentrations. The methodology does not determine an individual's health outcome or include the use of reported health effects data. This **final HRA** includes data analyzed from 5 May 1991 through 3 December 1991.

**II. CONCLUSIONS .**

A. Health Risk Assessment.

1. The results of this **HRA** indicate the potential for **significant** long-term adverse health effects for the exposed DD troop or civilian employee populations is minimal.

2. The total predicted excess carcinogenic risk both in Kuwait and in Saudi Arabia did not exceed 3 excess cancers per 1,000,000 population exposed (3 per 1,000,000). The predicted carcinogenic risk levels are well within the EPA range of acceptable excess carcinogenic risk of 1 per 1,000,000 to 100 per 1,000,000 exposed population.

3. The predicted noncarcinogenic risk levels based upon **subchronic** exposure periods [i.e., hazard indices (**HI**s) per EPA methodology] ranged from 0.6 - 2.0 in Saudi Arabia and from 2.0 - 5.0 in Kuwait. For the protection of human health, EPA toxicity values used in the **HI**s are set far below levels known to cause health effects and include consideration of exposures of sensitive subpopulations such as children and the elderly. The EPA's criteria indicate that if the HI value exceeds 1 or unity, there may be concern for potential noncarcinogenic effects. The majority of noncarcinogenic risk (> 99 %) at all monitoring sites is predicted to be from the inhalation of volatile organic compounds (**VOCs**), in



particular, benzene, [see paragraph 2b(3)]. Due to the conservative nature of the risk assessment methodology and derivations of the HIs, the risks for potential adverse health effects among the DD population are considered low.

#### B. The Biologic Surveillance Initiative.

1. The Biologic Surveillance Initiative (BSI) was a companion study to quantify exposure to environmental contaminants by measuring biological markers of exposure and internal dose in DD troops [1 1<sup>th</sup> Armored Cavalry Regiment (1 1<sup>th</sup> ACR)] which deployed from Germany to Doha, Kuwait (within approximately 20 miles of the fires) soon after the war.

2. Metals assay results of biological specimens collected from troops of the 11<sup>th</sup> ACR, both in Germany and Kuwait, are consistent with extremely low-level exposures to metals. These results are characteristic of dietary and smoking exposures, and are consistent with the low levels of metals measured in the environmental characterization phase of the HRA.

3. Assay results of blood specimens for VOCs are consistent with the low levels of VOCs found in the environmental characterization phase of the HRA. Some variations in values were observed during study of the troops in Germany, in Kuwait, and later in Germany; but no clinical significance can be attached to these differences at this time. In particular, blood levels of benzene were lower in Kuwait during the deployed period than the pre or postdeployment periods. This finding supports the conclusion that the noncarcinogenic risk levels indicate minimal potential adverse health effects.

4. Assay results of blood for sister chromatid exchange (SCE) frequency are consistent with an increase in genic stress for soldiers both during and after deployment to Kuwait. Increases in SCE frequency have been associated with many different chemical and drug exposures, but they have never been determined to be predictive of or associated with any adverse health effects. Further study of genotoxic changes associated with deployments in general may be needed.

5. Assay results of blood specimens for polycyclic aromatic hydrocarbon (PAH) adducts to DNA are consistent with low PAH exposure levels. These results may be related to the low PAH levels measured in the environmental characterization phase of the HRA, or they could be related to other exposures, such as smoking and dietary. The results also indicate the possibility that exposures to these compounds were lower in Kuwait than in Germany.



C. Environmental.

1. Ground level pollutant concentrations were lower than anticipated, possibly due to a rapid rise of smoke into a consolidated plume high above the ground, preventing contaminants from reaching high concentrations at ground level.

2. The particulate levels measured in ambient air generally exceeded U.S. standards and may have presented the potential for acute respiratory health effects in the very young, the very old, and in personnel with pre-existing **airway** disease. Any adverse health effects would be expected to have been minimal in a healthy troop population. Measured particulate concentrations in the Southwest Asia area are normally the highest levels **observed** in the world, primarily due to windblown sand.

3. Concentrations of organic compounds of concern were less than expected and were found near or below analytical detection limits,

4. Differentiation of contaminant contributions from the oil well **fires**, the gushing wells, and the oil pools was confounded by the presence of high levels of these contaminants in the anthropogenic and natural backgrounds of Saudi Arabia and Kuwait.

5. There is no evidence of environmentally induced radiological health risk to the general DD population based on air sample analyses. No uranium particles were detected on a representative sample of air **filters** using electron microscopy techniques. Over 40,000,000 particles were scanned in this evaluation.

III. **RECOMMENDATION.** Establish an intergovernmental agency committee. Task it with the responsibility to develop a comprehensive environmental incident response program, based upon the experience gamed from the **Kuwaiti** oil well **fires**. This program must quickly identify the level of response needed, provide transport and logistical support integrated response teams of environmental and medical experts throughout the world.



TABLE OF CONTENTS

Paragraph	Page
I. REFERENCES .....	1
II. AUTHORITY .....	1
III. PURPOSE.. .....	1
Iv. GENERAL .....	1
A. Background .....	1
B. <b>Project Scope</b> .....	2
C. Methodology .....	4
V. <b>HEALTH RISK ASSESSMENT</b> .....	9
A. Data Collection and Evaluation .....	9
<b>B. Exposure</b> Assessment .....	9
<b>C. Toxicity</b> Assessment .....	21
D. Risk Characterization .....	22
<b>E. Uncertainty</b> Analysis .....	24
VI. DISCUSSION OF HEALTH RISK ASSESSMENT RESULTS .....	44
A. Cancer Risk Levels .....	44
B. Noncancer Risk <b>Levels</b> .....	47
<b>C. Incremental Versus Absolute Risk</b> .....	49
VII. <b>HEALTH RISK - RELATED STUDIES</b> .....	52
A. Incorporation of the Biologic <b>Surveillance</b> Initiative Results With the Health Risk Assessment Results .....	52
<b>B. Radiation Exposure</b> .....	54

Paragraph	Page
<b>VIII. CONCLUSIONS</b> . . . . .	<b>55</b>
A. <b>Health</b> Risk Assessment . . . . .	55
B. Specific Components . . . . .	56
<b>IX. RECOMMENDATIONS</b> . . . . .	<b>56</b>
Appendices	
A-1 REFERENCES . . . . .	A-1-1
A-2 <b>CARCINOGENIC/NONCARCINOGENIC EXPOSURE AND RISK SUMMARY TABLES</b> . . . . .	A-2- 1
A-3 <b>CARCINOGENIC SLOPE FACTORS, NONCARCINOGENIC REFERENCE DOSES/REFERENCE CONCENTRATIONS, TOXICITY PROFILES, AND HEALTH EFFECTS OF INHALED PARTICULATES</b> . . . . .	A-3-1
B <b>AMBIENT AIR SAMPLING</b> AND AIR PATHWAY ANALYSIS . . .	B-1
C <b>SOIL SAMPLING</b> AND SOIL PATHWAY ANALYSIS . . . . .	C-1
D <b>INDUSTRIAL HYGIENE AIR SAMPLING</b> . . . . .	D-1
E <b>PHASE 1 AND PHASE 2 METHODOLOGIES, ANALYTICAL RESULTS AND QUALITY ASSURANCE</b> . . . . .	E-1
F <b>BIOLOGICAL SURVEILLANCE INITIATIVE</b> . . . . .	F-1
G <b>SAND AND AMBIENT AIR SAMPLE ANALYSIS</b> . . . . .	G-1
H <b>RADIOLOGICAL ANALYSIS</b> . . . . .	H-1
I <b>RESPONSE TO COMMENTS ON INTERIM KUWAIT OIL FIRE HEALTH RISK ASSESSMENT</b> . . . . . * . . . *	I-1



REPLY TO  
ATTENTION OF

DEPARTMENT OF THE ARMY  
U. S. ARMY ENVIRONMENTAL HYGIENE AGENCY  
ABERDEEN PROVING GROUND, MARYLAND 21010-5422



HSFB-ME-SR

FINAL REPORT  
KUWAIT OIL FIRE **HEALTH** RISK ASSESSMENT  
NO. 39-26-L192-9 1  
5 MAY - 3 DECEMBER 1991

I. REFERENCES. See Appendix A-1 for a list of references that were used in the body of this assessment.

II. AUTHORITY. Memorandum, OTSG, SGPS-PSP, 16 April 1991, subject: Kuwait Oil Fire **Health** Risk Assessment.

III. PURPOSE. This health risk assessment (**HRA**) was conducted to characterize both the carcinogenic and **noncarcinogenic** health risks to Department of Defense (DOD) troops and DOD civilian employees exposed to the environment affected by the oil fires during and after Operation Desert Storm. This final **HRA** includes data analyzed from 5 May 1991 through 3 **December** 1991.

IV. **GENERAL.**

A. Background.

1. The exposure of DOD and Allied personnel to more than 700 burning and gushing oil wells destroyed during the conflict in the Persian Gulf region raised concerns about potential health effects. The initial monitoring reports concluded that pollutants emanating from the oil wells were at levels which would not cause severe short-term health problems. The groups conducting the monitoring, however, (i.e., EPA, French, Norwegians, and the Kuwait Environmental **Protection** Department) emphasized that the long-term health effects to exposed individuals were not evaluated because of **insufficient** data.

Use of trademarked names does not imply endorsement by the U.S. Army but is intended only to assist in **identification** of a specific product.

2. The concern with potential long-term health effects to DOD troops and civilians prompted the DOD to initiate this study. The DOD Health Affairs tasked the U.S. **Army Office** of The Surgeon General (OTSG) to chair a **Tri-Service** medical working group to evaluate the potential health effects of the oil smoke on DOD personnel. The group consisted of medical personnel from each **military** service, in addition to representatives from the Veterans Administration, DOD Health Affairs, and the **Office** of the Deputy Assistant Secretary of Defense for Environment, Safety and Occupational Health. As part of the working groups effort a team from the U.S. Army **Environmental** Hygiene Agency (**USAEHA**) was dispatched on 1 May 1991 to collect samples and monitor the health effects in South West Asia, and to prepare an HRA. The Interim Kuwait Oil Fire **HRA** was released 19 June 1992. Many health complaints were registered with the Veterans Administration by individuals who **served** in **Operation** Desert Storm/Desert Shield since the interim report was released. The health complaints/symptoms experienced by these veterans are variable and many are very nonspecific. The collection of symptoms is referred to as the "mystery illness. " The **search** continues for possible causes.

3. This **HRA** looks at the theater-wide risks associated with oil **fire** smoke, industrial pollution (which is **difficult** to separate from smoke related contaminants), natural background (i.e., heavy metals), and radioactivity (both natural associated with the oil bearing strata and qualitatively that resulting from depleted **uranium**).

**B. Project Scope.** This project consisted of three main areas: an environmental monitoring effort, with subsequent HRA; an industrial hygiene sampling study; and a biologic surveillance initiative. Upon completion, these studies will be integrated to **obtain** a comprehensive assessment of the environmental situation in the gulf region and the resultant health consequences to DOD personnel. A Troop Unit Exposure Model is being conducted as an additional effort to the **HRA**. This effort supports Public **Law** 102-190, **Section** 734, which requires a means to calculate exposure to the Kuwait oil **well** fires for DOD personnel deployed in Operation Desert Storm. This modeling effort is required to augment the risk assessment for two reasons. First, data were only collected at nine fixed sites in the Theater of Operation; however, troops were very mobile throughout the entire theater. Second, sampling started 5 May **1991**, **2** months after the fires were ignited.

1. Environmental Monitoring. The environmental monitoring study attempted to characterize the **concentration** of pollutants that DOD personnel were exposed to during their deployment in the gulf region. The period of exposure (i.e., time) and the location where that exposure occurred were very variable for the large number of DOD personnel in-theater (approximately 550,000). By the end of February, there were 605 oil wells on fire and 46 gushing oil.

a. The **USAEHA** monitoring effort commenced on 5 May 1991 and continued until 3 December 1991. When environmental monitoring began, 558 oil wells remained on **fire**. Data collection continued until all the fires were extinguished (approximately 6 November 1991). In addition, 1 month of background data were collected from 6 November - 3 December 1991. While in the gulf region, troops occupied approximately 880,000 square miles within Kuwait, Saudi Arabia, and Iraq. This extensive **area** made **quantification** of exposure by troop location very **difficult**. Therefore, as a **starting** point for exposure measurement, permanent ambient air monitoring stations were established at four locations in Saudi Arabia and six locations in Kuwait, although two in Kuwait were quickly abandoned due to logistical **difficulties**. The locations were selected because they were major sites where DOD troops were stationed long term. To augment the fixed location sampling, air modeling is being conducted **in** conjunction with the National Oceanic and Atmospheric Administration (NOAA), to predict pollutant concentrations at locations and times when no sampling was being conducted.

b. In addition to air monitoring, soil sampling was done at the air sites to ensure that all potential exposure pathways and media were evaluated. The data generated from the environmental monitoring activities were used to calculate the exposure point concentrations for the individual **HRAs** contained in this **final** report. Risk assessments were conducted for each of the seven permanent air/soil monitoring sites where DOD personnel were located and the Ahmadi Hospital site that was within visual distance of the burning Ahmadi Oil Field.

2. Industrial Hygiene Survey. The industrial hygiene (**IH**) air survey monitored and characterized occupational exposures of DOD personnel who had potential high risk exposure to oil **fire** emissions. The **IH** air sampling was conducted from 3 May 1991 to 17 June 1991 at various locations within Kuwait and Saudi Arabia. The focus was on individuals working outdoors and on worst-case situations within the oil fields next to Kuwait City. The **IH** air sampling results were compared to recognized occupational health standards to assess the health risk to exposed individuals.

### 3. Biologic Surveillance.

a. The Biologic Surveillance Initiative (**BSI**) was conducted to refine and corroborate the results obtained from the **HRA**. This was accomplished by collection of objective biologic measurements of exposure and effect in real-time, and by establishment of any observable biologic effect or marker of exposure to oil **fire** pollutants in a cohort of U.S. soldiers. When an element of U.S. troops was **identified** [the 11th Armored Cavalry Regiment (11th ACR)] for deployment from Europe to Kuwait and return, a team from **USAEHA** and **cooperating** agencies was assembled to plan and **carry** out an assessment that would measure the health effects of the oil fires on the **troops**.

b. The 11th ACR was given questionnaires, had biologic fluids (blood and urine) collected for analysis, and had **pulmonary** function tests performed between 1 June 1991 and 14 October 1991. These activities were accomplished in Germany prior to deployment, in Kuwait following exposure to the environment, and upon return to Germany. **The** predictive results from the **HRA** (which were generated from environmental data) were compared to the BSI results from actual biologic samples of potentially exposed troops. The comparison lends validity to the HRA and shows that its predictions of no significant **health** consequences from oil fire exposure are correct.

### C . Methodology.

1. Sampling and Analysis. Sampling and analysis methodologies for ambient air, soil, industrial hygiene air, and biologic samples are detailed in their respective appendices and the Analytical Methodology and Quality Assurance appendix (Appendices B through **H**).

2. Risk Assessment. The methodology selected for completion of this **HRA** was EPA guidance developed for the Comprehensive Environmental Response, Compensation and Liability Act (**CERCLA**) sites, also known as "**Superfund**" sites. The calculations result in a quantitative estimate of **health** risk based on the **contaminant** concentrations and the site exposure characteristics. Assessments conducted using **Superfund** guidance **are** based on the **reasonable** maximum exposure (**RME**) scenario. The **RME** is **defined** as the highest exposure that is reasonably expected to occur at a site. The methodology does not use the absolute worst-case scenario, but is nevertheless very conservative because the data that are selected for use and the exposure and risk factors that are incorporated into the assessment are conservative. The risk assessment **has six** distinct steps which will be discussed below.

a. Data Collection. The collection of an environmental data base is the heart of any risk assessment. An early determination of the types of data that will be required to complete the risk assessment is essential. Items such as contaminant identities; environmental fate, transport, and persistence of contaminants; **characteristics** of the source; and contaminant concentrations in the key exposure pathways **are** required for a quality data base. As with risk assessment, data collection has certain key steps that must be accomplished.

(1) Reviewing Available Information. The initial step in formulating data needs is to review the available information on the site characteristics (i.e., climate, topography, contaminant sources), on the **hazardous** substances to be monitored (i.e., crude oil analysis, products of incomplete combustion, breakdown products), and on potential exposure pathways.

(2) **Defining** Background Sampling Needs. Background sampling is conducted to distinguish site-related (oil fire) contaminants from naturally occurring or other **nonsite**-related levels of chemicals (industrial/vehicle emissions). This was a particularly difficult task for this **HRA** due to the large and diverse geographical area assessed, the very complex and varied nature of the contaminants produced by the **fires**, and the large number of sources contributing background contaminants to the area.

(3) Identifying Potential Human Exposure. This **area** of data collection involves determining the following: environmental media that may be contaminated and to which individuals may be exposed and/or through which chemicals may be transported to the potential receptors; areas of concern (i.e., locations where **the** environmental media is to be sampled); types of contaminants expected at the sampling sites and their environmental behavior, persistence, and accumulation; potential routes of contaminant transport through the environment and to which receptor populations may be exposed; and potential receptor populations, particularly sensitive subgroups.

(4) Developing an Overall Strategy for Sample Collection. In developing a sampling strategy **that** will adequately address the questions the risk assessment is trying to answer the following factors must be determined: sample size, sample location, sampling strategy (i.e., random, purposive, systematic, etc.) and sample type.

(5) Measuring Quality Assurance/Quality Control (**QA/QC**). The **QA/QC** issues that need to be addressed in the data collection plan are: sampling protocols, sample collection' **devices/equipment**, QC samples, collection procedures, holding times, chain of custody, and sample preservation.

b. Data Evaluation. After all the environmental samples have been collected and analyzed, the data set that is produced must be **evaluated** to determine its suitability for **incorporation** in the risk assessment. To evaluate the data and prepare a data set for the risk assessment the following must be accomplished: evaluate the analytical methods, evaluate the data with respect to **QA/QC** parameters (i.e., blanks, data qualifiers, quantitation limits, holding times), evaluate tentatively identified compounds, **compare** potential site-related contamination with background, and evaluate the chemicals to be carried through the risk assessment.

c. Exposure Assessment. The exposure assessment portion of the risk assessment attempts to estimate the type and magnitude of exposures to the chemicals of potential concern that are impacting the receptor populations. The exposure assessment consists of the following:

(1) Characterization of the Exposure Setting. **In this** step the physical environment is characterized (i.e., climate, meteorology, soil type, topography) along with a characterization of the potentially exposed populations (i.e., location relative to the source, activity patterns, and sensitive subgroups).

(2) Identification of Exposure Pathways. This step of the exposure assessment identifies the pathways (i.e., air, soil) by which the previously identified populations may be exposed. The determination of complete exposure pathways involves the following: identify contaminant release sources (i.e., oil **fires** and lakes) and receiving media (i.e., air and soil); evaluate fate and **transport** in **release** media; identify exposure points (i.e., population contact points with **contaminants**) and exposure routes (i.e., ingestion, inhalation, and dermal contact).

(3) Quantification of Exposure. In this step the risk assessor quantifies the magnitude, frequency, and duration of exposure for each **identified** pathway. This process occurs in two **steps**:

(a) Estimation of Exposure Concentrations. This step of the process involves determining the concentration of contaminants that **will** be contacted over the exposure period. Exposure concentrations can be estimated using monitoring data (as will be done in this risk assessment) or using chemical transport and environmental fate modeling (as **will** be for troop areas where no monitoring was conducted). The **EPA** methodology for Superfund uses the **RME** for each pathway. This value is the 95 percent upper confidence **limit** of the arithmetic average of the monitoring data for the pathway being evaluated. **This** methodology develops a conservative exposure concentration, **while** not using the maximum concentration detected which would not **be** reasonable.

(b) Calculation of Intakes. In this step of the exposure quantification the **chemical-specific** exposures for each identified pathway are calculated. Exposure estimates are expressed in **terms** of the mass of substance in contact with the body per **unit** body weight per unit time (e.g., mg chemical per kg body weight per **day**, also expressed as **mg/kg-day**). Chemical intakes are calculated using equations that include variables for exposure concentration, contact rate, exposure frequency, exposure duration, body weight, and exposure averaging time. There is a different equation for each exposure pathway/route (i.e., ingestion of soil, dermal contact with soil, inhalation of airborne chemicals, etc.).

d. Toxicity Assessment.

(1) The toxicity assessment determines the potential for each chemical of concern to cause adverse effects on the exposed populations. This assessment can also determine the relationship between the extent of exposure to a contaminant and the increased likelihood and/or **severity** of adverse effects. The toxicity assessment is accomplished in two steps: hazard **identification** and dose-response evaluation. Hazard identification determines whether exposure to a contaminant can cause an increase in the incidence of a particular health effect (e.g., cancer, birth defect) and whether the adverse health effect is likely to occur in humans. A dose-response evaluation characterizes the relationship between the dose of the contaminant **administered** or received and the incidence of adverse health effects in the exposed populations. The types of data considered in toxicological assessments come from human epidemiologic studies and work place exposures, animal studies, and supporting metabolic/physiologic studies.

(2) The toxicity assessment is conducted for both carcinogenic and noncarcinogenic effects. When assessing carcinogenic effects, the critical toxicity value is the slope factor which estimates the upper bound probability of a response (cancer) per unit intake of a chemical over a lifetime. Another important factor when assessing cancer risk is the **weight-of-evidence** classification. This EPA system groups chemicals based on the available toxicity data to their status as human carcinogens (i.e., human carcinogen, probable human carcinogen, etc.). When assessing **noncarcinogenic** effects, the most often used **critical** toxicity value at **Superfund** sites is the reference dose (**RfD**). The **RfD** is an estimate (with uncertainty of an order of magnitude or greater) of a daily exposure level for the human population, including sensitive subpopulations, that is likely to be without an appreciable risk of deleterious effects during a **specified** period of time. There are different **RfDs** for different periods of time [i.e., chronic (lifetime), subchronic (2 **weeks** to 7 years), etc.]. In addition to time periods for **RfDs**, **both** slope factors and **RfDs** are derived for the **specific** route of exposure (i.e., inhalation and ingestion, no **RfDs** or slope factors are available for the **dermal** route of exposure). The EPA has listed a **hierarchy** of sources for toxicity information used **in** Superfund risk assessments and these were used throughout this assessment.

e. Risk **Characterization**. Risk characterization is the final step in the baseline **HRA** process. In this step, the toxicity and exposure assessments are integrated into quantitative and qualitative expressions of risk. To characterize potential **noncarcinogenic** effects, comparisons **are** made between projected intakes of contaminants and toxicity values (**RfDs**). Potential carcinogenic effects (i.e., probabilities that an individual will develop cancer over a

lifetime of exposure) are estimated from projected intakes and chemical-specific **dose-response** values (slope factors). In accordance with EPA guidelines, intakes for estimating carcinogenic effects are averaged over the receptor populations lifetime, while intakes for estimating noncarcinogenic effects are averaged over the actual exposure period.

(1) Cancer Risk. Excess lifetime cancer risks are obtained by multiplying the intake rate at the exposure point of the **contaminant** by its cancer slope factor. Under the **Superfund** Program, the **EPA** has determined the acceptable range of excess cancer to be  $1 \times 10^{-4}$  to  $1 \times 10^{-6}$  (i.e., the probability of one excess cancer in a population of 10,000 to one excess cancer in a population of **1,000,000**, respectively, under the conditions of exposure). A risk level of  $1 \times 10^{-6}$ , representing a probability of one in **1,000,000** that an individual could develop cancer due to an exposure to potential carcinogens at a site, is often used as "the point of departure" by regulatory agencies to trigger action. The total cancer risk for a site is **generally** determined by adding the individual cancer risks for each chemical **in** the pathways and then summing the risk for **all** the pathways. If there are known synergistic **and/or** antagonistic relationships between carcinogens or specific target organs are involved, these factors can be taken into account when determining cancer risk.

(2) Noncancer Risk. Noncancer hazards are obtained by dividing each chemicals daily intake by its **RfD** (for this assessment the subchronic **RfD**, because exposure duration was between 2 weeks and 7 years). These hazard quotients (**HQ**) are summed for the various contaminants to obtain a hazard index (**HI**) for the pathway. The **HI**s for the various pathways are then combined and this represents the total noncancer risk for the site. Under the EPA **Superfund** Program a hazard index of unity (1) is considered the threshold of concern. As with cancer risk the combining of **HI**s and **HQ**s can be **modified** by specific toxicological information such as mechanism of action, effect, or target organ/system.

(f) Uncertainty Analysis. Uncertainty analysis is a key modifying element of any risk assessment. The uncertainty analysis discusses those issues and factors that are not completely understood or known, such as: were sufficient environmental samples collected to adequately characterize the media being evaluated; or using dose-response information from animal studies to predict effects in humans. Some uncertainty factors overestimate risk, others underestimate risk, while some may be capable of doing either under different circumstances. These issues may cause the assessment to either over or underestimate risk. Uncertainty analysis should discuss in detail all issues that may cause risk to be over or under estimated.

## V. HEALTH RISK ASSESSMENT.

A. Data Collection and Evaluation. Data collection and evaluation methods, background sampling, QA/QC measures, site characteristics, etc., are discussed in detail in each respective appendix. Any questions or specific issues relating to the above subject areas should be contained in the appropriate appendix.

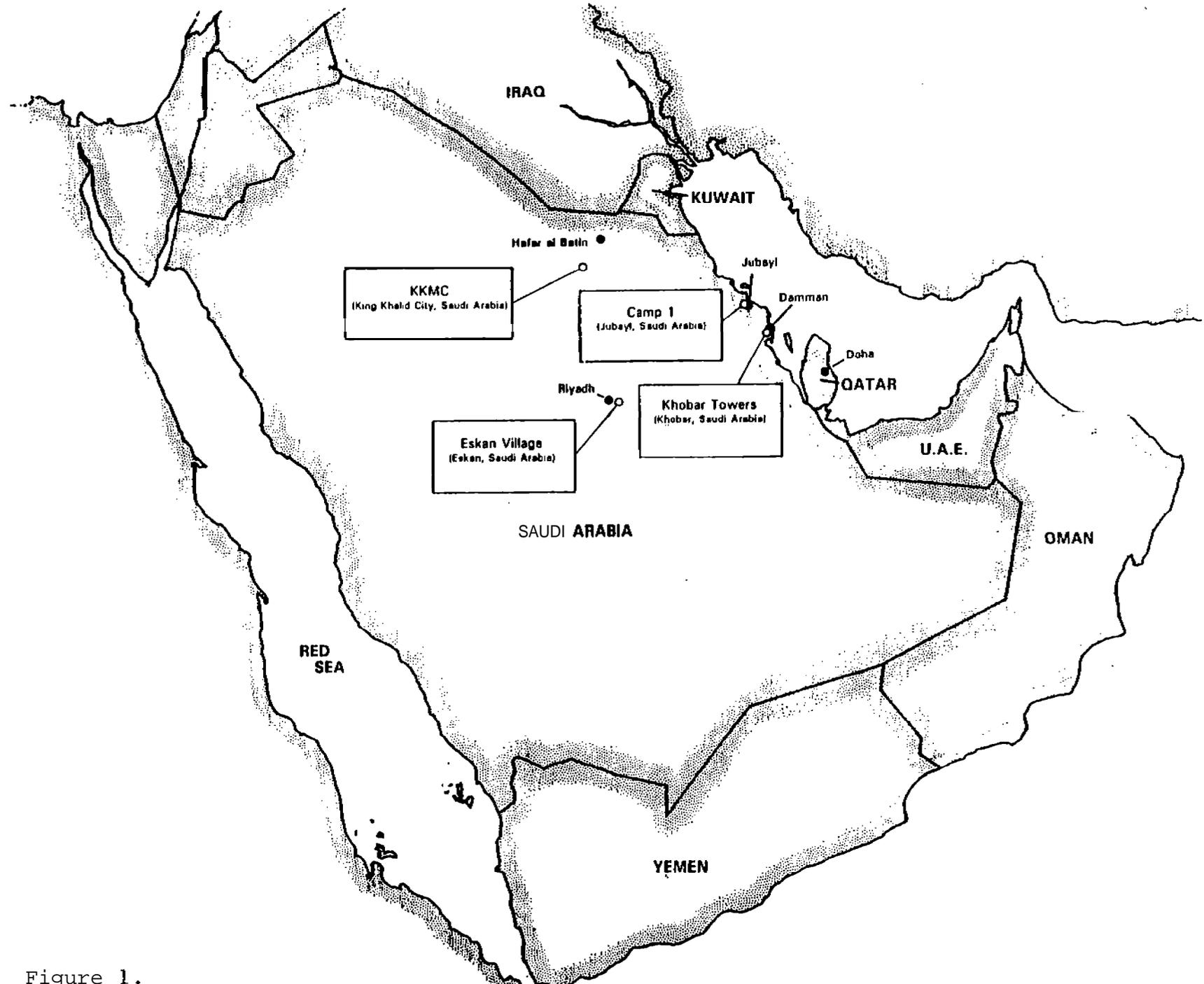
### B. Exposure Assessment.

#### 1. Characterization of Exposure Setting.

a. Physical Environment. Environmental sampling was concentrated along the eastern portion of the Arabian Peninsula where DOD personnel were concentrated. Figure 1 is a map of the Arabian Peninsula and the surrounding countries. The eastern provinces of Saudi Arabia, where the sampling occurred, is composed of gradually undulating plains. Natural vegetation is sparse in the region except near oases. Saudi Arabia is **2,149,690 km<sup>2</sup>** with a population of **16,108,539**. Figure 2 is a map of the Emirate of Kuwait and the location of the environmental sampling sites. Kuwait is **17,820 km<sup>2</sup>** with prominent terrain and geographical features that consist of flat sandy desert, **costal** beaches, and some areas of elevated rocky outcrops or gently rolling arid **sageland**. Most of the indigenous population [**1,700,000** (680,000 Kuwaitis, **1,020,000** expatriates)] is located along the coast around Kuwait City and near interior oil collection areas.

b. Climate. Kuwait and the eastern portion of Saudi Arabia can be classified as a hot desert climate. Mean annual temperatures average 80 degrees Fahrenheit (**°F**), with average daily maximum temperatures of approximately 90 **°F** and average daily **minimum** temperatures in the mid to upper 60's. **Mean** extreme maximum daily temperatures for the summer **season** generally exceed 110 **°F**. Average annual precipitation ranges from less than 1 inch in **costal Saudi Arabia** to approximately 4 inches in Kuwait and occurs during the winter through **early** spring months. **This** region is characterized by an increased frequency of blowing sand and dust due to the sandy soil (desert environment) and increased wind speeds (commonly referred to as "**Shamal** winds"). **The** prevailing wind direction is from the northwest for Kuwait and from the north/northwest for Saudi Arabia. Associated annual average wind speeds range from 13.8 miles per hour (mph) for Kuwait to 11.5 mph and 6.9 mph for Dahrhan and Riyadh, Saudi Arabia, respectively.

c. Potentially Exposed Populations. The potentially exposed populations for this assessment were all DOD military and civilian personnel that were in the Persian Gulf region (Kuwait, Saudi Arabia, Iraq) during the oil fires. This population group(s) **covers** a wide geographical **area** (approximately 880,000 square miles) and **has** a very diverse exposure period (weeks to several months). Their locations relative to the source of pollution was also



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Figure 1.

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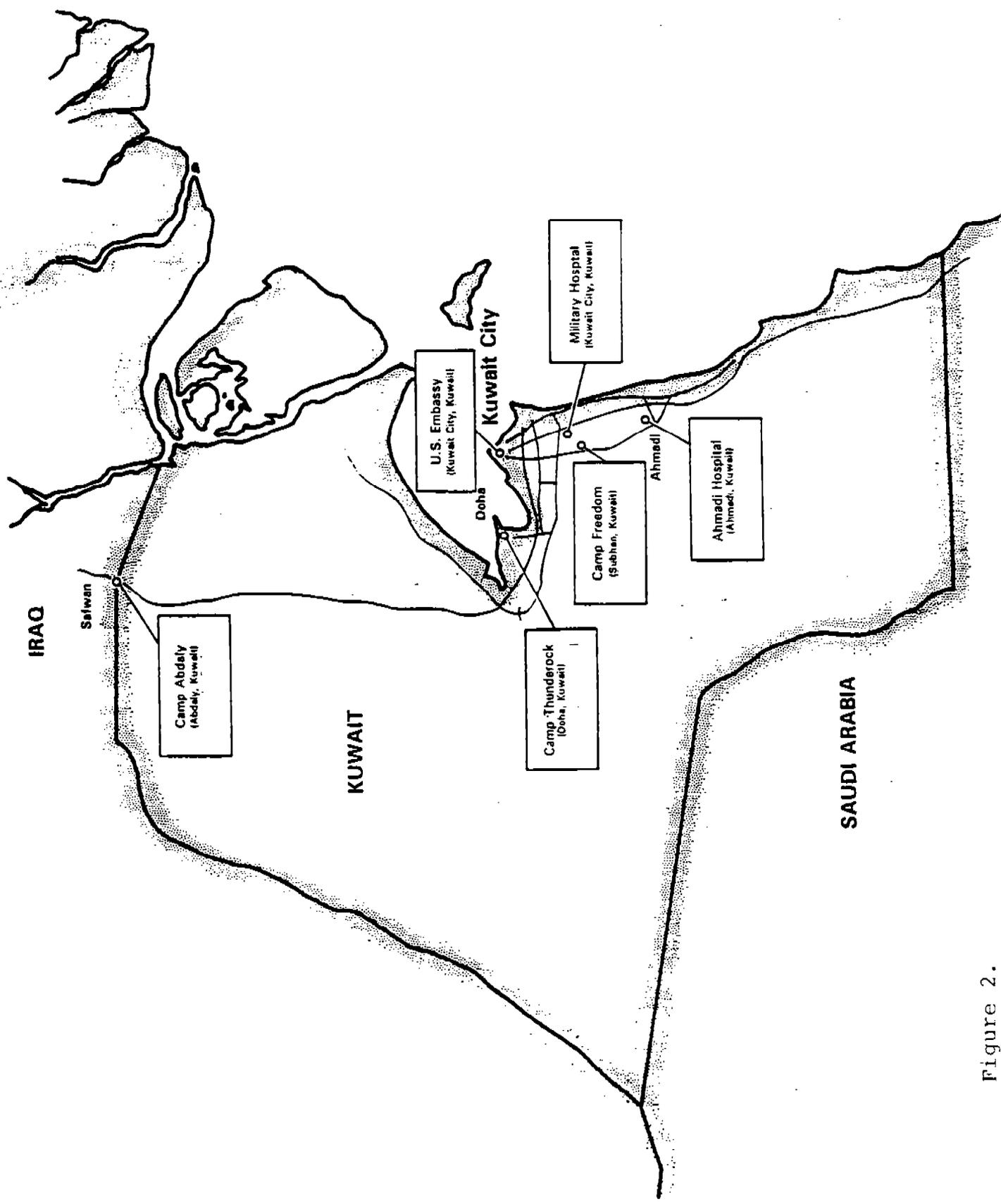


Figure 2.

of chemicals these populations were exposed to was, therefore, very **difficult**. To accomplish this function, eight ambient air monitoring stations were established at **fixed** facilities set up for long-term military/civilian presence. These sites in no way covered all the locations where DOD personnel were present during the assessment period. To cover these “blind spots” air **modelling** will be conducted in conjunction with the NOAA to predict exposure point concentrations in these **areas**. These two efforts should adequately address the impact of oil fire contaminants on the potentially exposed populations.

2. Identification of Exposure Pathways. An exposure pathway describes the course a contaminant takes from the source to the exposed individual (see Figure 3). An exposure pathway generally consists of four elements:

- \* A source and mechanism of chemical release (i.e., oil fires and lakes releasing vapors and combustion products to the air and soil).
- \* A receiving and transport medium (i.e., air and soil).
- \* A point of potential contact with the contaminated medium (the “exposure point”).
- \* An exposure route (i.e., inhalation, ingestion, **dermal** contact) at the contact point.

An exposure pathway is considered complete only if all these elements are present. Only complete pathways are evaluated in risk assessments. The following pathways are considered complete for this risk assessment:

- a. Soil Pathway. For the soil pathway the **following** routes of exposure are operative:

(1) Ingestion of Chemicals in Soil. This exposure route requires direct contact with contaminated soil onto hands **and/or** lips, followed by inadvertent hand-to-mouth contact or licking of the lips. The intake of the various contaminants is estimated by relating the measured contaminant concentrations in surface soil to the estimated soil ingestion rate, modified by the other parameters in the intake formula. Some contaminants (metals) **were** detected in the surface soils at the monitoring sites. The detections could potentially have resulted from particulate fall-out generated by the **oil fires**. There are also many other sources for the metals in the soil, including natural background and industrial pollution. This fact is obvious since not all of the metals found in **soil** are associated with the crude oil that was consumed by the **fires**. The **soil/sand** in the region is very loose and sparsely vegetated making it **readily** available for contact and incidental ingestion.

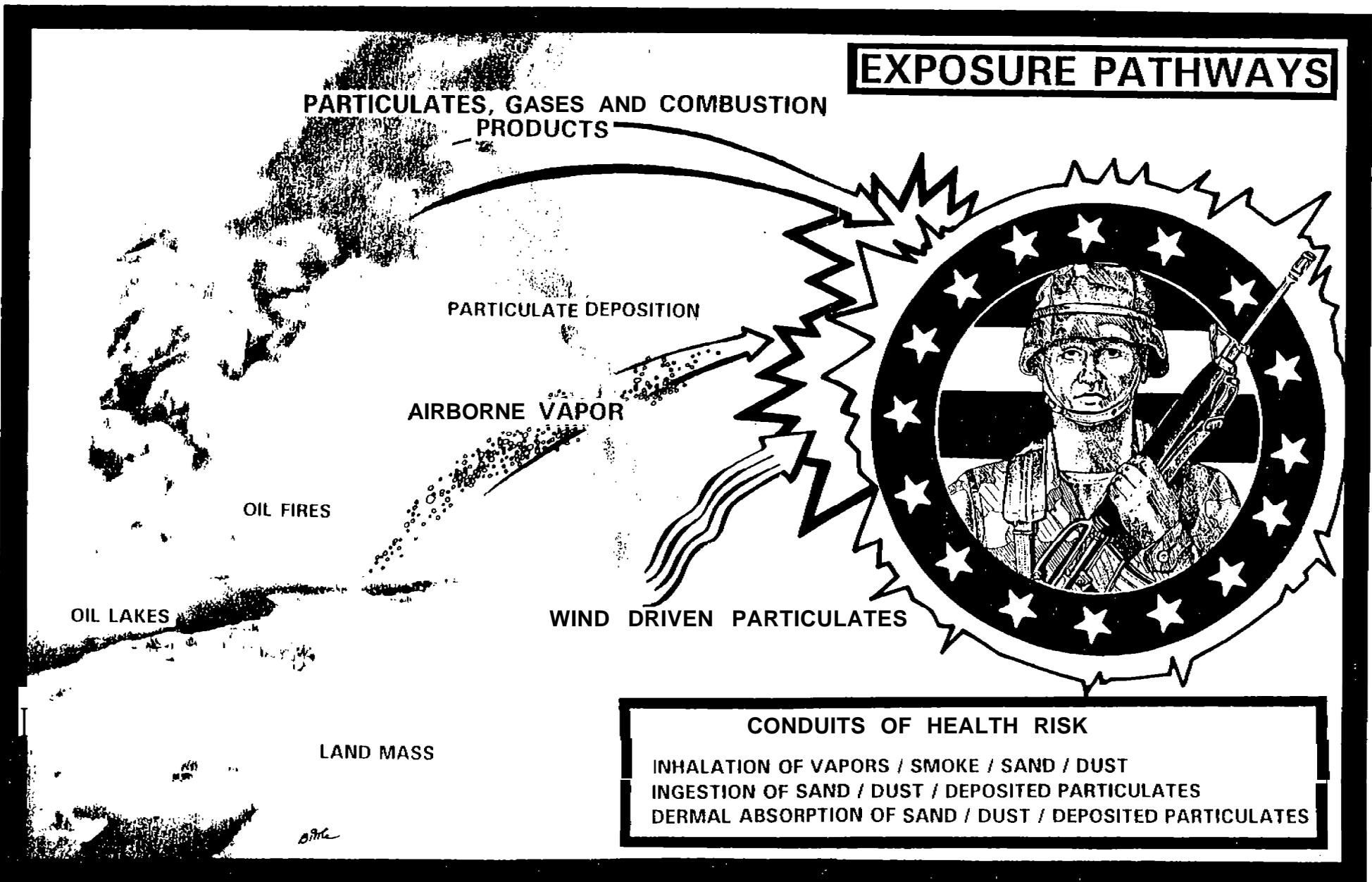


Figure 3.

(2) Direct Dermal Contact.

(a) This exposure route requires direct contact of exposed skin with the contaminated soil. Calculation of exposure for this pathway results in an estimate of the absorbed dose. Absorption factors are used to reflect the **desorption** of the chemical from soil and the absorption of the chemical across the skin and into the blood **stream**. Of all the exposure routes, the dermal route is the least understood and the most difficult to accurately evaluate. For the **contaminants** detected in surface soil only cadmium has both a Soil to **Skin** Adherence Factor and an Absorption Factor. These factors help determine and modify the absorbed dose that is calculated using the contaminant concentration in soil. Cadmium, however, does not have an oral cancer slope factor which is then used with the absorbed dose to compute the risk.

(b) As with the soil ingestion route, the loose material and sparse vegetation made soil readily available for **dermal** contact. The troops' hands, arms, **neck**, and head were generally uncovered and available for surface contact with soil and the high temperature caused sweating that increased particulate adherence. The dermal exposure route was not evaluated **in** the interim **HRA** because EPA was in the process of releasing their new Dermal Exposure Assessment: Principles and Applications, January 1992. This exposure route is included in the final **HRA**, although there is **still** a great deal of associated uncertainty.

(3) Inhalation of Soil Particles. This exposure route estimates the intake of contaminants in soil by breathing air in which contaminant-bearing soil particles are suspended as wind-eroded dust. Intake of contaminants from this exposure route are estimated from air sample analysis. The inhalation route of the soil pathway (i.e., wind eroded soil) is not separated from the **direct** air pathway (i.e., inhalation of vapors and airborne **particulates** and combustion products). These particulate materials are collected on the same air sampling **filters** and can not be separated for analysis.

b. Air Pathway. For the air pathway, the only major operative exposure route is the inhalation of airborne vapors (from oil lakes and gushing oil wells), gases (**SO<sub>2</sub>** and NO, formed during combustion), particulates, and combustion products. A full discussion of this pathway and the contaminants produced by the oil fires is contained in Appendix B. The inhalation of airborne contaminants produced by the oil fires and oil lakes had a potential impact on DOD personnel throughout the **theater**. Due to the geographical diversity of troop locations and the potential for multiple background interferences (natural, industrial, and vehicular sources), the estimation of fire-related contaminant exposures through this pathway is very difficult. Appendix A-3, which contains the toxicity profiles and values, also contains a discussion of health effects of inhaled particulates. This discussion attempts to clarify the impact of particle size on deposition in the respiratory system and subsequent health impacts.

c. Incomplete Pathways. The following pathways were considered but not evaluated, because they were judged to be either incomplete or of no health consequence.

(1) Ground Water/Drinking Water. DOD troops and civilians were provided with sealed containers of bottled water for their consumption. **Local** drinking water supplies were not utilized. Drinking water, therefore, was not included in the risk assessment because it was considered a safe, uncontaminated medium.

(2) **Surface** Water. The surface water medium was not evaluated due to its scarcity in the desert environment. There were **swimming** pools available for use by the troops; however, an **examination** of the soils data (Appendix C) indicated that the deposition of contaminated **particulates** emanating from the oil **fires** was negligible.

(3) Food. All food DOD troops and civilians consumed was provided by the military, in the form of Meals Ready to Eat (**MREs**), or the like, sealed in plastic, or provided in mess halls. Large numbers of meals were not eaten on the local economy. In addition, there was no **reason** to believe that the local food was contaminated with oil **fire** residue.

3. **Quantification** of Exposure. To quantitatively assess the potential exposures associated with complete pathways, estimates of **chemical** concentrations at the exposure point (i.e., monitoring data) are combined with values describing the extent, frequency; and duration of exposure to provide estimates of the chronic daily intakes (**CDIs**). The **CDIs** are quantified by estimating the RME associated with the pathway of concern. The RME is intended to represent a possible upper-bound exposure to a **typical** individual and is combined with upper-bound toxicity criteria to estimate risk.

a. Approach. The procedures used by **USAEHA** to calculate exposures were those presented in Risk Assessment Guidance for Superfund, Volume I, Human **Health** Evaluation Manual (Part A) (see reference 10). The methods **used** to calculate these intakes included use of the **RISK\*ASSISTANT** COMPUTER MODEL and LOTUS 1-2-3 spreadsheets. The approaches have been previously reviewed and approved by the EPA Office of Health and Environmental Assessment. Parameters for calculating exposures (i.e., inhalation rates, body weight, etc.) have been extracted from EPA risk assessment documents (see references 1, 2, and 3). For this **final** report the use of the **RISK\*ASSISTANT COMPUTED** MODEL has been eliminated due to certain inherent **idiosyncracies** in the model that caused the air pathway intake values to double. This fact was discovered after **publication** of the interim report when it became apparent that the program was making the outdoor 20-hour exposure a **24-hour** exposure and the indoor **4-hour** exposure a 24-hour exposure. The program would then add the two values for a **48-hour** exposure, with a subsequent increase in the exposure point concentration.

b. Sample Data. Table 1 lists the environmental media considered in this analysis (out of a possible set of ground water, surface water, air, soil, sediment, crops, and biota). Table 1 also indicates the technique used to combine data from multiple samples in each medium and the sample set that was included. The final column indicates the approach used to assign concentrations when a chemical was not detected in some of the samples. If there were no detects of a particular chemical in a media, the **chemical** was not carried through the risk assessment.

**TABLE 1. ENVIRONMENTAL MEDIA EVALUATION**

Medium	Aggregation Strategy	Sample Set	Treatment Nondetects
Air	Sample Mean & Upper 95% of C.I. about the arithmetic mean (RME)	PM-10 Volatiles Metals PAHs Nontarget analytes Radionuclides	1/2 of detection limit     +
soil	" " " " "	Metals Semivolatile Organics	" " " "

\* For **IH** air samples, the detection limit was used in treatment of nondetects.

+ For radionuclide air samples, no reported activities were considered as nondetects. All measurements, both above and below the Lower Limit of Detection (LID), were reported for **evaluation**. If activities below the **LLD** were rejected and considered nondetects, when making measurements near background levels, possible distortion or serious errors in long-term trend analysis could result.

c. Exposure Pathways. It is important to remember that the calculated doses and concentrations presented in this assessment refer only to the **specific** exposure pathways enumerated in this assessment. An exposure pathway combines contamination in an environmental medium, a scenario describing how a person contacts that medium, and a route of exposure (oral, inhalation, or dermal). An assessment that incorporates other



EF = Exposure Frequency (**days/yr**), 30 **days/yr** was used in this assessment such that monthly cancer risks could be summed and monthly hazard indices could be averaged.

ED = Exposure Duration (yr), 1 year was used as this was the maximum exposure duration possible with the **fires** extinguished in less than 1 year (approximately 9 months).

BW = Body Weight (kg), 70 kg was used for the average person.

AT = Averaging Time (days), 70 yr X 365 **days/yr** was used for carcinogenic effects and 30 **days/yr** for noncarcinogenic effects so that monthly hazard indices could be averaged.

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(2) Ingestion intakes for chemicals in soil are calculated using the equation:

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$$\text{Intake(mg/kg-day)} = \frac{\text{CS} \times \text{IR} \times \text{CF} \times \text{FI} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad (2)$$

Where:

CS = Contaminant Concentration in Soil (**mg/kg**)

**IR** = Ingestion Rate (mg **soil/day**), 300 mg of soil/&y was used.

CF = Conversion Factor (lob kg/mg)

FI = Fraction Ingested from a contaminated source (unitless), 1 was used.

EF = Exposure Frequency (**days/yr**), 30 days/yr was used in this assessment such that monthly cancer risks could be summed and monthly hazard indices could be averaged.

ED = Exposure Duration (**yr**), 1 year was used as this was the maximum exposure duration possible with the **fires** extinguished in less than 1 year (approximately 9 months).

BW = Body Weight (kg), 70 kg was used for the average person.

AT = Averaging Time (days), 70 yr X 365 **days/yr** was used for carcinogenic effects and 30 **days/yr** for **noncarcinogenic** effects so that monthly hazard indices could be averaged.

(3) Dermal Exposure intakes are calculated using the equation:

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$$\text{Absorbed Dose (mg/kg-day)} = \frac{\text{CS} \times \text{CF} \times \text{SA} \times \text{AF} \times \text{ABS} \times \text{EF} \times \text{ED}}{\text{BW} \times \text{AT}} \quad (3)$$

Where:

**CS** = Contaminant Concentration in Soil (**mg/kg**)

**CF** = Conversion Factor (**10<sup>-6</sup> kg/mg**)

**SA** = Skin Surface Area Available for Contact (**cm<sup>2</sup>**), 3460 **cm<sup>2</sup>** was used for work exposure; 7510 **cm<sup>2</sup>** was used for recreational exposure. This exposure was partitioned 80% work and 20% **recreational**, for a daily average of 4270 **cm<sup>2</sup>**.

**AF** = Soil to Skin Adherence Factor (**mg/cm<sup>2</sup>**); 1 mg, the upper value from the EPA **Dermal** \*sure Assessment guidance was **selected**.

**ABS** = Absorption Factor (unitless); Chemical-specific value (this value accounts for desorption of the **chemical** from the soil matrix and absorption of the chemical across the skin. Note: for this assessment only cadmium has both AF and ABS factors available. Cadmium, however, does not have an **oral** slope factor which is used to compute the absorbed dose and resultant risk.

**EF** = Exposure Frequency (events/yr), 30 **days/yr** was used in this assessment such that monthly cancer risks could be summed and monthly **hazard** indices could be averaged. **Events/yr** is equal to days/yr in this equation because the amount of soil adhering to a soldier skin (SA times **AF**) occurs **as** a single **daylong** event in the field.

**ED** = **Exposure** Duration (**yr**), 1 year was used as this was the maximum exposure duration possible with the fires extinguished in less than 1 year (approximately 9 months).

**BW** = Body Weight (kg), 70 kg was used for the average person.

**AT** = Averaging Time (days), 70 yr X 365 **days/yr** was **used** for carcinogenic effects and 30 **days/yr** for noncarcinogenic effects so that monthly **hazard** indices could be averaged.

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Values for each of the variables in the equations 1 through 3 above were selected based upon **site-specific** information available, or from exposure factors presented in the EPA Exposure Factors Handbook (**EPA/600/8-89/043, 1989**), the revised factors found in OSWER Directive **9285.6-03**, "Standard Default Exposure Factors," 25 March 1991, or **Dermal** Exposure Assessment: Principles and Applications, (**EPA/600/8-91/011 1B**, January 1992).

e. Applied Dose. When an exposure assessment will be used as part of a quantitative risk assessment, it is common to provide an estimate of applied dose. This is usually expressed as a dose rate (mass of chemical per unit time), adjusted for body weight; it is generally expressed in units of **mg/kg/day**.

(1) The EPA has recently decided to evaluate risks from inhalation exposure on the basis of contaminant concentration **in** air, **rather** than dose expressed in **mg/kg/day** (**Interim Methods for Development of Reference Doses, EPA/600/8-88/066F**).

(2) When evaluating the risk of chronic (greater than 7 years exposure), non-cancer health effects from oral exposures, **EPA** employs the Average Daily Dose (ADD) received during the period of exposure. When evaluating carcinogenic risks from oral exposures that last less than a lifetime, the ADD is adjusted to a dose rate that would yield an equivalent total dose if exposure continued for the entire lifetime. This is the Lifetime Average Daily D o s e (**LADD**).

$$\mathbf{LADD} = \text{ADD} * (\text{exposure period in years/lifetime in years})$$

(3) When evaluating the risk of subchronic (14 days to 7 years exposure) non-cancer health effects **from** various exposure routes, EPA employs contaminant concentrations. These are compared to reference concentrations (**RfCs**)/**reference** doses (**RFDs**) that correspond to continuous exposure.

(4) When evaluating carcinogenic risks from inhalation exposures that last less than a lifetime, the exposure concentration is adjusted to reflect the difference in exposure pattern from the assumption of continuous lifetime exposure that is used in deriving unit risks for inhalation exposure to carcinogens. Exposure estimates for the various sampling sites are found **in** Appendix A-2. Estimates are presented on a monthly basis for each chemical, pathway, and route of exposure.

C. **Toxicity Assessment.** The toxicity assessment presents available evidence regarding the potential for contaminants detected in soil and air samples to have an adverse impact on exposed DOD personnel. The essential toxicity values for the evaluation of carcinogenic and non-carcinogenic risk are discussed below. Toxicity values for this assessment were obtained using the following hierarchy of sources recommended for **Superfund** sites:

- \* The **EPA** Integrated Risk Information System (**IRIS**) and cited references.
- \* The **EPA** Health Effects Assessment Summary Tables (**HEAST**) and cited references.
- \* Consultation with the EPA Superfund **Health** Risk Technical Support Center (TSC).

1. **Health Effects Criteria for Carcinogens.** The slope factor, which relates the potency of a carcinogen, is developed by EPA's Health Assessment Group for chemicals that are potentially carcinogenic. Slope factors [expressed in units of **(mg/kg-day)<sup>-1</sup>**] are derived from the results of human epidemiological studies or chronic animal **bioassays**. Usually, the animal studies must be conducted using relatively high doses to detect possible adverse effects. Because humans are expected to be exposed to much lower doses, the data are adjusted (**i.e.**, extrapolated) by using mathematical models. The slope factors are derived **using** very conservative assumptions, and while the actual risks associated with exposure to potential **carcinogens** are unlikely to be higher than calculated, they could be considerably lower. The other major factor considered when **evaluating** carcinogenic effects is the **weight-of-evidence** classification. This system characterizes carcinogens to the extent that the available data indicate if the agent is a human carcinogen. Table 2 lists the groups and their respective classifications.

2. **Health Effects Criteria for Noncarcinogens.** Health **effects** criteria for noncarcinogenic chemicals are termed **RfDs** for oral exposure and **RfCs** for inhalation exposure. These criteria are generally developed by the EPA **RfD** Work Group or are obtained from Health Effects Assessments. The **RfD**, expressed in units of **mg/kg-day** and the **RfC**, expressed as **mg/m<sup>3</sup>**, are estimates of daily exposure levels for the human population, including sensitive subpopulations, that are unlikely to produce an appreciable risk of deleterious effect during a lifetime (chronic **RfD/RfC**) or a portion of a **lifetime** (2 weeks to 7 years - subchronic **RfD/RfC**). These **RfDs/RfCs** are usually derived from human studies involving work place exposures or from animal studies, and are adjusted using uncertainty/safety factors. Table A-3-1 (see Appendix A-3) lists the available toxicity criteria for the chemicals evaluated in this risk assessment. Appendix A-3 also contains the toxicity profiles for the chemicals evaluated in this risk assessment.

TABLE 2. EPA **WEIGHT-OF-EVIDENCE** CLASSIFICATION SYSTEM FOR CARCINOGENICITY

Group	Description
A	Human carcinogen.
<b>B1</b> or B2	Probable human carcinogen.  <b>B1</b> indicates that limited human data are available.  B2 indicates sufficient evidence in animals and inadequate or no evidence in humans.
C	Possible human carcinogen.
D	Not classifiable as to human carcinogenicity.
E	Evidence of <b>noncarcinogenicity</b> for humans.

D. Risk Characterization. The risk characterization quantitatively assesses the potential risk to human health based on the concentrations of chemicals in the exposure pathway and the relevant toxicity **value** for that chemical. For this risk assessment, calculated risk levels are presented for the seven permanent monitoring sites where DOD personnel were located on a continuous basis and the Ahmadi Hospital site. The risk levels for Camp Freedom and the Kuwait Military Hospital were combined because of the lack of long-term monitoring data at Camp Freedom and because of their close proximity to each other. The **Ahmadi** Hospital site risk assessment is presented for comparison purposes only, as its location was closest to the oil **fires** (within 1 mile). There was never a permanent DOD troop presence there for any sustained period of time.

1. Methodology. The EPA methodology used for this assessment was developed for **CERCLA/Superfund** sites. The calculations result in quantitative estimates of health risk based on the **contaminant** concentrations at the exposure sites, the exposure parameters, and the toxicity data.

## 2. Cancer Risk.

a. Excess lifetime cancer risks are obtained by multiplying the intake rate/intake concentration of the chemical of concern at the exposure point by the contaminant's cancer slope factor/cancer unit risk factor. Cancer slope factors are developed for each route of exposure (i.e., inhalation/ingestion) for which the **chemical** is a carcinogen. In many instances the chemical may only be a carcinogen by one route, or a slope factor may only be available for one route. The dermal route is the most difficult to assess because there are no **dermal** slope factors. In this case the oral slope factor is used if it is available.

b. Under the **Superfund** Program, the EPA has determined the acceptable range of excess cancer risk to be  $1 \times 10^{-4}$  to  $1 \times 10^{-6}$  (i.e., the probability of one excess cancer in a population of 10,000 to one excess cancer in a population of **1,000,000**, respectively, under the designated conditions of exposure). A risk level of  $1 \times 10^{-6}$ , representing a probability of one in **1,000,000** that an individual could **develop cancer** due to an exposure to potential carcinogens at a site, is often used as the point of departure for further action by regulatory agencies. For this assessment, we will use the  $1 \times 10^{-6}$  risk level as our point of departure to determine whether or not an excess cancer risk exists.

## 3. **Noncancer** Risk.

a. Subchronic (i.e., 14 day to 7 year exposures) **noncancer hazards** are obtained by dividing each chemical's daily intake by its subchronic **RfD/subchronic RfC**, then summing these Hazard Quotients (**HQs**) by receptor. This sum is known as the **Hazard** Index (HI). In the **Superfund Program**, EPA considers a HI of unity (1) to be the threshold of concern. For this assessment, we will use an **HI=1** as our threshold of concern. As a general rule, the greater the HI exceeds unity, the greater the level of concern. When subchronic **RfDs/RfCs** are not available for certain compounds, a chronic **RfD/RfC**, if available, is used to provide a conservative estimate of the noncancer risk. Chronic **RfDs/RfCs** are protective for a lifetime of exposure (i.e., 7 years to life).

b. As with cancer slope factors, **RfDs** are developed for each route of exposure and may not always be available for all routes. It must be noted that the **RfD/RfC** theory assumes **that** there is a level of exposure below which it is unlikely for even sensitive populations to experience adverse health effects. **RfDs/RfCs are** derived from the following values:

\* **Lowest-observed-adverse-effect** level (**LOAEL**)

\* **Lowest-observed-effect** level (**LOEL**)

\* No-observed-adverse-effect level (NOAEL)

\* No-observed-effect level (NOEL)

The RfD/RfC is then developed using the following formula:

$$[\text{RfD}] \text{ or } [\text{RfC}] = \frac{\text{NOAEL or LOAEL}}{\text{UF X MF}}$$

The uncertainty factor (UF) used in calculating the RfD or RfC reflects scientific judgement regarding the various types of data used to estimate RfD or RfC values. An uncertainty factor of 10 is generally used to account for variation in human sensitivity among populations. An additional ten-fold factor is generally used to account for each of the uncertainties assumed when extrapolating from animal data to humans, when extrapolating from an LOAEL to an NOAEL, and when extrapolating from subchronic to chronic exposure. In order to reflect professional assessment of the uncertainties of the study and the database not explicitly addressed by the above uncertainty factors, an additional uncertainty factor or modifying factor (MF) ranging from greater than 0 to less than or equal to 10 is applied. UF X MF values ranging from 100 to 10,000 are typical. With the values used to derive RfDs/RfCs (i.e., LOAELs/NOAELs) and the very large safety factors applied (i.e., 100-10,000), the resultant toxicity values are very conservative.

4. Data Presentation. The cancer risk estimates and noncancer HIs for the exposure sites are found in Tables 3 through 10. These tables show the total risk for the period of exposure from early May 1991 through 3 December 1991. Cancer risks for each month are summed, including a summation for each chemical and for each pathway (month to month). Noncancer hazards are averaged over the months of exposure since the individual HQs are specific to the period of exposure (i.e., number of months of exposure). Appendix A-2 contains tables with a monthly breakdown of cancer and noncancer risks by pathway, route of exposure, and individual chemicals.

E. Uncertainty Analysis. There is a large degree of uncertainty associated with the estimates of human health risk in any risk assessment. Consequently, the estimates calculated for this HRA should not be construed as absolute estimates of health risk, but rather as conditional estimates based on a number of assumptions regarding exposure and toxicity. An awareness of the uncertainties associated with the risk estimates is critical to understanding the nature of any predicted risks and to placing the predicted risks in proper perspective. To understand the meaning of the quantitative dose and risk estimates presented in this HRA, it is necessary to consider the key assumptions used in deriving them, and the uncertainties associated with those assumptions. The following discussion focuses on the major uncertainties associated with this HRA.

TABLE 3. CARCINOGENIC AND NON-CARCINOGENIC (HAZARD INDEX) RISK SUMMARY FOR **KHOBAR** TOWERS, AL **KHOBAR**, SAUDI ARABIA, MAY 1991 - NOVEMBER 1991

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**Carcinogenic Risk**

Total Inhalation of Metals*	=	8E-7
<b>Total</b> Inhalation of Volatile <b>Organics</b>	=	7E-7
Total Inhalation of Polycyclic Aromatic Hydrocarbons-j-	=	7E-8
Total Incidental Ingestion of Metals*	=	2E-8
Total Dermal Absorption of Metals in Soil*	=	2E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>2E-6</b>

**Non-Carcinogenic Risk**

Average Inhalation of <b>Volatiles</b>	=	2E+
Average Incidental Ingestion of Metals*	=	6E-2
Average <b>Dermal</b> Absorption- of Metals in Soil*	=	3E-2
<b>TOTAL HAZARD INDEX</b>	=	<b>2E+0</b>

---

\* AU chromium was **treated** as **chromium(III)**.

+ All polycyclic aromatic **hydrocarbon** concentrations were evaluated using **EPA** Carcinogenic Equivalency Factors (**CEFs**).

Find **Rpt**, Kuwait Oil Fire HEW No. **39-26-L192-91**, 5 May - 3 Dec 91

TABLE 4. CARCINOGENIC AND NON-CARCINOGENIC (**HAZARD INDEX**) RISK SUMMARY FOR CAMP 1, AL JUBAYL, SAUDI **ARABIA**, MAY 1991 - AUGUST 1991

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**Carcinogenic Risk**

Total Inhalation of Metals*	=	5E-7
Total Inhalation of Volatile <b>Organics</b>	=	2E-7
Total Inhalation of Polycyclic Aromatic Hydrocarbons+	=	2E-9
Total Incidental Ingestion of Metals*	=	4E-8
Total Dermal Absorption of Metals in Soil*	=	3E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>7E-7</b>

**Non-Carcinogenic Risk**

Average Inhalation of <b>Volatiles</b>	=	2E+0
<b>Average</b> Incidental Ingestion of Metals*	=	7E-2
Average <b>Dermal</b> Absorption of Metals in Soil*	=	3E-2
<b>TOTAL HAZARD INDEX</b>	=	<b>2E+0</b>

---

\* All chromium was **treated** as **chromium(III)**.

+ All polycyclic aromatic hydrocarbon concentrations were **evaluated** using EPA Carcinogenic Equivalency Factors (**CEFs**).

TABLE 5. CARCINOGENIC AND NON-CARCINOGENIC (**HAZARD INDEX**) RISK SUMMARY FOR **ESKAN VILLAGE**, RIYADH, SAUDI ARABIA, JUNE 1991 - AUGUST 1991

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**Carcinogenic Risk**

Total Inhalation of Metals*	=	2E-7
Total Inhalation of Volatile <b>Organics</b>	=	1E-7
Total Inhalation of Polycyclic Aromatic <b>Hydrocarbons</b> †	=	3E-9
Total Incidental Ingestion of Metals*	=	4E-8
Total <b>Dermal</b> Absorption of Metals in Soil*	=	3E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>4E-7</b>

**Non-Carcinogenic Risk**

Average Inhalation of <b>Volatiles</b>	=	1E+0
Average Incidental Ingestion of Metals*	=	8E-2
Average <b>Dermal</b> Absorption of Metals in Soil*	=	4E-2
<b>TOTAL HAZARD INDEX</b>	=	<b>1E+0</b>

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\* All chromium was **treated** as **chromium(III)**.

† All polycyclic aromatic **hydrocarbon** concentrations were evaluated using **EPA** Carcinogenic Equivalency Factors (**CEFs**) .

TABLE 6. CARCINOGENIC AND NON-CARCINOGENIC (HAZARD INDEX) RISK SUMMARY FOR KING KHALID MILITARY CITY, SAUDI ARABIA, MAY 1991 - AUGUST 1991

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**Carcinogenic Risk**

Total inhalation of Metals*	=	2E-7
Total Inhalation of Volatile Organics	=	7E-8
Total Inhalation of Polycyclic Aromatic Hydrocarbons†	=	8E-10
Total Incidental Ingestion of Metals*	=	5E-8
Total Dermal Absorption of Metals in Soil*	=	4E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>4E-7</b>

**Non-Carcinogenic Risk**

Average Inhalation of Volatiles	=	3E-1
Average Incidental Ingestion of Metals*	=	1E-1
Average Dermal Absorption of Metals in Soil*	=	6E-2
<b>TOTAL HAZARD INDEX</b>	=	<b>5E-1</b>

---

\* All chromium was treated as chromium(III).

† All polycyclic aromatic hydrocarbon concentrations were evaluated using EPA Carcinogenic Equivalency Factors (CEFs).

TABLE 7. CARCINOGENIC AND NON-CARCINOGENIC (**HAZARD INDEX**) RISK SUMMARY FOR MILITARY HOSPITAL AND CAMP **FREEDOM**, KUWAIT, MAY 1991 - **NOVEMBER** 1991 .

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**Carcinogenic Risk**

Total Inhalation of Metals*	=	9E-7
Total Inhalation of Volatile <b>Organics</b>	=	3E-7
Total Inhalation of Polycyclic Aromatic <b>Hydrocarbons</b> †	=	4E-9
Total Incidental Ingestion of <b>Metals</b> *	=	1E-7
Total <b>Dermal</b> Absorption of Metals in Soil*	=	7E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>1E-6</b>

**Non-Carcinogenic Risk**

Average Inhalation of <b>Volatiles</b>	=	1E+0
Average <b>Incidental</b> Ingestion of Metals*	=	2E-1
Average <b>Dermal</b> Absorption of Metals in Soil*	=	1E-1
<b>TOTAL HAZARD INDEX</b>	=	1E+0

---

\* All chromium was treated as **chromium(III)**.

† All polycyclic aromatic **hydrocarbon concentrations** were evaluated using EPA Carcinogenic Equivalency Factors (**CEFs**) .

**TABLE 8. CARCINOGENIC AND NON-CARCINOGENIC (HAZARD INDEX) RISK SUMMARY FOR UNITED STATES EMBASSY, KUWAIT CITY, KUWAIT, MAY 1991 - JULY 1991**

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**Carcinogenic Risk**

Total Inhalation of Metals*	=	4E-7
Total Inhalation of Volatile <b>Organics</b>	=	2E-7
Total Inhalation of Polycyclic Aromatic Hydrocarbons+	=	0E+00
Total Incidental Ingestion of Metals*	=	4E-8
Total <b>Dermal</b> Absorption of Metals in Soil*	=	3E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>7E-7</b>

**Non-Carcinogenic Risk**

Average Inhalation of <b>Volatiles</b>	=	2E+0
Average Incidental Ingestion of Metals*	=	9E-2
<b>Average</b> Dermal Absorption of Metals in Soil*	=	5E-2
<b>TOTAL HAZARD INDEX</b>	=	<b>2E+0</b>

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\* All chromium was treated as **chromium(III)**.

+ All polycyclic aromatic **hydrocarbon** concentrations were evaluated using EPA Carcinogenic Equivalency Factors (**CEFs**).

TABLE 9. CARCINOGENIC AND NON-CARCINOGENIC (**HAZARD INDEX**) RISK SUMMARY FOR CAMP THUNDEROCK, **DOHA**, KUWAIT, JUNE 1991 - NOVEMBER 1991

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**Carcinogenic Risk**

Total Inhalation of Metals*	=	1E-6
Total <b>Inhalation</b> of Volatile Orgaics	=	1E-6
Total Inhalation of Polycyclic Aromatic Hydrocarbons+	=	5E-9
Total Incidental Ingestion of Metals*	=	4E-8
Total Dermal Absorption of Metals in Soil*	=	3E-8
<b>TOTAL CARCINOGENIC RISK</b>	=	<b>2E-6</b>

**Non-Carcinogenic Risk**

Average Inhalation of Volatiles	=	4E+0
Average Incidental Ingestion of Metals*	=	1E-1
Average Dermal Absorption of Metals in Soil*	=	6E-2
<b>TOTAL HAZARD INDEX</b>	=	<b>4E+0</b>

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\* All chromium was treated as **chromium(III)**.

+ All polycyclic aromatic hydrocarbon concentrations were evaluated using EPA Carcinogenic Equivalency Factors (**CEFs**).

TABLE 10. CARCINOGENIC AND NONCARCINOGENIC (HAZARD **INDEX**) RISK SUMMARY FOR AL **AHMADI** HOSPITAL, AHMADI, KUWAIT, MAY 1991 - JULY 1991

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Carcinogenic, Risk

Total Inhalation of Metals*	=	1E-7
Total Inhalation of Volatile <b>Organics</b>	=	2E-7
Total Inhalation of <b>Polycyclic</b> Aromatic <b>Hydrocarbons</b> †	=	3E-8
Total <b>Incidental</b> Ingestion of Metals*	=	4E-8
Total Dermal Absorption of Metals in Soil*	=	3E-8
TOTAL CARCINOGENIC RISK	=	4E-7

Non-Carcinogenic Risk

Average Inhalation of <b>Volatiles</b> 266.91	=	5E+0
Average Incidental Ingestion of <b>Metals</b> *	=	7E-2
Average <b>Dermal</b> Absorption of <b>Metals</b> in <b>Soil</b> *	=	5E-2
TOTAL <b>HAZARD</b> INDEX	=	5E+0

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\* All chromium was **treated** as **chromium(III)**.

† All **polycyclic** aromatic **hydrocarbons concentrations** were evaluated using EPA Carcinogenic Equivalency Factors (**CEFs**).

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1. Site Selection- Sampling site locations were selected based on their proximity to where a majority of U.S. troops and DOD **civilians** were located for extended periods of time during and after the war. Environmental monitoring activities were conducted at seven major DOD troop sites and two non-troop sites (i.e., U.S. Embassy and **Ahmadi** Hospital). During the oil **fires**, DOD personnel were located at many other sites which were not monitored. Therefore, to obtain risk levels at locations where no monitoring was conducted, modeling will be carried out with NOAA to calculate risk levels based on model-generated data. To **validate** the model-generated exposure data, comparisons will be made with actual

monitoring results. In addition to generating exposure data for sites where no monitoring was **carried** out, data will also be generated for the time period when no sampling was being conducted (i.e., February, March, and April).

## 2. Chemical Selection.

a. The environmental samples collected were analyzed for a very broad spectrum of chemicals. The analysis was **based** on the crude oil composition, including contaminants, combustion products, potential combustion by-products, and any degradation/conversion products. The list of chemicals was not inclusive, but was as complete as possible in light of certain logistical, analytical and **QA/QC** constraints.

b. The selection of chemicals for risk **evaluation** allows for the elimination of certain chemicals due to factors such as comparison with natural and anthropogenic background levels and comparison with concentrations detected in blank samples. For this assessment all chemicals detected, except for those that were also found in blank samples, were evaluated with respect to health risk. This included chemical that were obviously not associated with the oil **fires** because they were not **contained** in the Kuwait crude oil. These contaminants were probably the result of oil **refining**, industrial activities, vehicular **traffic**, open refuse **fires**, and natural background materials (see Figure 4). Although these contaminants were not associated with the oil **fires**, they still contributed to the overall **risk** to which DOD **personnel** were exposed, and thus they were evaluated in the **HRA**.

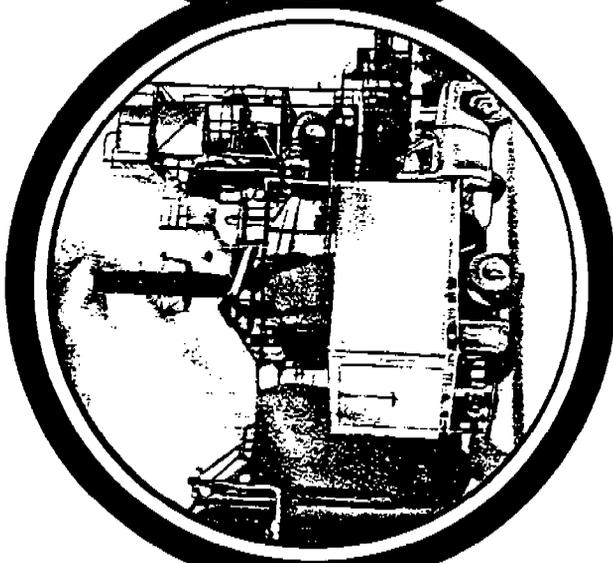
3. Sampling and Analytical Methodologies. Sampling and analytical methodologies used in this study could lead to unknown inconsistency of the **data** due to systematic and nonsystematic errors in procedures. However, the methodologies selected were standard EPA or other validated procedures, coupled with **sufficient QA/QC** techniques, that should hold data inconsistencies to a minimum.

4. Exposure Scenarios. The selection of exposure scenarios will also have a significant influence on estimated doses. Actual exposures to members of any **specified** population will vary **in** accordance with the degree to which they participate **in** the activities described by the exposure scenarios. Again, to ensure that the risk assessment protects potentially exposed DOD personnel conservative exposure **parameters** (i.e., **RME** values) and scenarios were used throughout this assessment. The values provided are estimates for the entire U.S. Troop population. Various demographic factors (including **geographic** region, rural or urban setting, socioeconomic status and ethnic heritage) may confound these values.

# TOTAL RISK FACTORS



**FIRE RELATED  
OIL FIRES / OIL LAKES**



**ANTHROPOGENIC  
BACKGROUND  
INDUSTRIAL / VEHICULAR**



**NATURALLY OCCURRING  
BACKGROUND**

Figure 4.

5. Exposure Parameters. The exposure parameters used in this **HRA** provide additional levels of conservatism that are protective of human health. For example, the 1.67 **m<sup>3</sup>/hr** and 0.71 **m<sup>3</sup>/hr** inhalation rates used for the outdoor and indoor inhalation exposure routes are RME values which total 36.24 **m<sup>3</sup>/day** of air (the average 24-hour inhalation is only 20 **m<sup>3</sup>**). Note also that the same exposure point concentration values were used for the outdoor/indoor inhalation calculations; there was no attenuation factor applied to the indoor concentration. The outdoor work exposure period/day was set at 20 hours and the indoor resting period was set at 4 hours for the military day. In addition, the soil ingestion rate was set at 300 **mg/day** which is 63 percent of the ingestion rate for construction/landscaping short-term exposures. These very **conservative** exposure factors lend another layer of safety to the risk assessment.

6. Media Concentrations. A key assumption is that the concentrations specified for various environmental media represent the true concentrations to which people were exposed. Contaminant concentrations will actually vary across both time and space. The data presented, therefore, may be higher or lower than true concentrations. Two factors that **increase** the reliability and conservatism of the data in this assessment are the use of the **RME**, which is well above the average exposure case (but still in the possible range of exposure) and the use of actual monitoring data as opposed to modeled data. The very large database of environmental measurements, particularly ambient air **monitoring** which represents **24-hour** samples, lends more validity to the exposure **point** concentrations generated in this assessment.

7. Toxicological Data. The toxicological data used in this risk assessment also contributes to the uncertainty. As noted by the EPA in their Guidelines for Carcinogenic Risk Assessment:

a. there are major uncertainties **in** extrapolating from both animals to humans and from high to low doses;

b. there are important species differences in uptake, metabolism, and organ distribution of carcinogens, as well as species and strain differences in target site susceptibility; and

c. human populations are variable with respect to genetic constitution, diet, occupation and home environment, activity patterns and other cultural factors.

These same uncertainties also apply to noncarcinogenic toxicity values (i.e., **RfDs** and **RfCs**). These values, however, have an additional uncertainty factor, the modifying/uncertainty factors that are applied to the **LOAEL/NOAEL** from which they are derived. This "safety factor," usually ranging from 100 to **3000**, helps to account for some of the **uncertainty**

discussed above and makes noncancer toxicity values very conservative and protective of human **health**. A major issue that needs to be understood by the reader is the change in toxicity values in the 20 months between the interim **HRA** and this **final** HEW. A number of the **RfDs/RfCs** have been reduced two or three fold (i.e., arsenic, cadmium, ethyl benzene) thus increasing the apparent risk level. In addition, toxicity values that were under review have been released, again **increasing** the apparent risk level.

8. Assessing Carcinogenic Risk from Short-Term Exposures. The most confounding uncertainty of this assessment involves the application of EPA's concept of carcinogenic risk. The hypothesized mechanism is referred to as "nonthreshold" due to the belief that there is essentially no level of exposure to such a chemical that does not pose a **finite** probability of carcinogenic response. A two-part evaluation is used in which the chemical is assigned a weight-of-evidence **classification** and then a slope factor is calculated. The slope factor is a plausible upper-bound estimate of the **probability** of an adverse response per unit intake of a chemical over a lifetime. This assessment involves short-term exposures to carcinogens for which there is currently no methodology for evaluating risk and the validity of the EPA carcinogenic risk methodology is questioned. Per telephone conversation with Mr. Jim Cogliano, Chief, Carcinogen Assessment and Statistical Epidemiology Branch, EPA, the default risk method for carcinogens is lifetime based.

## 9. Chromium.

a. **All** chromium values used in the interim assessment were assumed to be the environmentally unstable **Cr<sup>+6</sup>** species. (**Cr<sup>+6</sup>**'s environmental instability is illustrated by its short environmental sample holding time of 24 hours.) Using **Cr<sup>+6</sup>** values produced conservative risk estimates, because **Cr<sup>+6</sup>** is the only species that is carcinogenic and because **Cr<sup>+6</sup>** is over two orders of magnitude more systemically toxic than **Cr<sup>+3</sup>**.

b. One of the key factors that determines which chromium species is present, is the soil **pH**. A **pH** range of 9.0 to 9.5 is generally required for **Cr<sup>+6</sup>** to persist in the environment. To determine the chromium species present at the various monitoring sites in Kuwait and Saudi Arabia, nine environmental soil samples were tested for soil **pH**. Table 11 illustrates that the soil **pH** values **ranged** from 7.4 to 8.0, with a **mean** of 7.6. These soil **pH** values would tend to favor the presence of **Cr<sup>+3</sup>**, resulting in lower carcinogenic and **noncarcinogenic** risk estimates.

c. To resolve the chromium species issues and more clearly define the health risk associated with chromium, a return trip to Kuwait and Saudi **Arabia** was made from 27 October to 12 November 1993. During the trip, soil samples were collected from all previously sampled sites, with the exception of King **Khalid** Military City (**KKMC**) (see Appendix C for details of the sampling). In addition, air samples were collected at Khobar

TABLE 11. **THE** PH OF SOIL SAMPLES FROM VARIOUS SITES IN **KUWAIT** AND **SAUDI ARABIA**

Site	Sample Designation	Soil pH
Khobar Towers	DA-3A	7.8
<b>Al</b> Khobar, SA	DA-1B-4	<b>8.0</b>
camp 1	<b>AJ-4A</b>	7.6
Al Jubayl, SA	<b>AJ-4B</b>	7.4
<b>Eskan Village</b>	<b>R-1A</b>	7.7
Riyadh, SA		
<b>KKMC</b> , SA	<b>KK-1A</b>	7.5
Camp Freedom, Kuwait	CF-2A	7.4
<b>Military</b> Hospital	<b>MH-1A</b>	7.7
Kuwait		
U.S. Embassy	EM-3A	7.6
Kuwait City, Kuwait		

SA = Saudi Arabia.

KKMC = King **Khalid** Military City.

Towers and Camp **Thunderrock**. The soil samples were analyzed in the field using a **Hach** Hexavalent Chromium Field Test Kit (see Appendix E for details of the methodology and the **QA/QC** method validation studies). The field analysis was necessary to overcome the short analytical holding **time** for **Cr<sup>+6</sup>**.

**d.** Seventy-six soil samples were **analyzed** in the field for **Cr<sup>+6</sup>**, with a detection limit of 100 **ppb**. All sample results were below the 100 ppb detection limit. A breakdown of the samples from each site, spike recoveries, and the surface versus depth samples are in Appendix E. In addition, 48 **Hi-Vol** air samples (i.e., filters), 24 from the first trip and 24 from the second trip, were also analyzed for **Cr<sup>+6</sup>** by the **RJ** Lee Company using computer

controlled and manual scanning electron microscopy (CCSEM, SEM, respectively), transmission electron microscopy (**TEM**) and a graphite furnace-equipped atomic absorption unit (AA). The limit of detection for the methodology is 10 ppb **Cr<sup>+6</sup>** in solution, or 0.3-0.5 **ng/m<sup>3</sup>** dependent on the total air volume filtered during the sampling event. The chromium detected on the air filters is associated with stainless steel rather than chromium metal. Presumably, the **Hi-Vol** air filters collected all **Cr<sup>+6</sup>** in the air. Once on the filters, it is unlikely that the **Cr<sup>+6</sup>** would reduce to **Cr<sup>+3</sup>**.

e. In this final **HRA**, all chromium was evaluated as **Cr<sup>+3</sup>** for the following reasons. Analysis of **Kuwaiti** crude oil (see Table C-1) did not show chromium to be a **contaminant** in the oil. Presumably, chromium found in the sand and air, therefore, originated from industrial activities or natural sources. As a result, these sources should be the same for both sampling periods. Analysis for **Cr<sup>+6</sup>** showed all soil and **air** samples to be below the detection limits. In addition, laboratory analysis for total chromium indicated levels in the soil that were similar to the levels in samples collected 2 years ago. The soil depth **profiles** (0-6", 6"-12", 1'-2', and 2'-3') collected and analyzed for total chromium also were similar with increasing depth, indicating the older (i.e., deeper) samples were similar to the more recent surface samples. All depth profiles were below the detection limit for **Cr<sup>+6</sup>**. These factors led to the conclusion that the chromium measured in Kuwait and Saudi Arabia during the first trip was **Cr<sup>+3</sup>**, just as it was during the second trip. This assumption may slightly underestimate cancer risk if a low level of **Cr<sup>+6</sup>** is present.

#### 10. Lead.

a. Lead, although not an oil **fire** contaminant, was detected in all soil and **air** samples. **Lead** is classified as a B2 carcinogen by EPA. It also has known noncarcinogenic effects. Lead does not currently have any **verified** toxicity values (i.e., slope factors or **RfD/RfC**) with which to assess the health risks.

b. There are however soil (state and EPA) and air [**National Ambient Air quality Standards (NAAQS)**] standards. To assess the health risk from exposure to lead in soil and air, the levels detected in **air** and soil were compared to the appropriate standard. Table 12 lists the soil and air standards that were found.

The levels of lead detected in soil samples collected in Kuwait and Saudi Arabia were all well below any of the standards presented above. Mean lead levels in soil ranged from a high of 37.7 **mg/kg** at the Ahmadi Hospital in Kuwait, to a low of 2.44 **mg/kg** at Camp Thunderock, **Doha**, Kuwait. All quarterly averages for all sites were below the **EPA NAAQS**.

TABLE 12. SOIL AND AIR STANDARDS

Source	Standard	Basis
<b><u>Soil</u></b>		
Centers for Disease Control and EPA (1989)	500-1000 <b>mg/kg</b>	Unlikely to cause <b>increased</b> blood lead levels in children and interim EPA criteria
EPA (1991)	<b>250-500 mg/kg</b>	Protect children based on EPA Lead Biouptake Model
Washington State Dept. of Ecology	250 <b>mg/kg</b> 1000 <b>mg/kg</b>	Residential areas Nonresidential areas
New Jersey Dept. of <b>Environmental</b> Protection and Energy	<b>100 mg/kg</b> 600 <b>mg/kg</b>	Residential areas Industrial areas
New York Dept. of Environmental Conservation ( <b>proposed</b> )	250 <b>mg/kg</b>	
<b>Minnesota</b> Pollution Control Agency	300 <b>mg/kg</b>	Residential <b>areas</b> and playgrounds
<b><u>Air</u></b>		
NAAQ	1.5 <b>µg/m<sup>3</sup></b>	

c. The risk from exposure to lead is considered minimal because all detected levels of lead in air and soil were below appropriate health-based standards. The most susceptible population to lead toxicity is young children (0-7 years old) who have developing nervous systems. A standard that is protective of children, therefore, is overly protective for adults. Adults absorb less lead in their digestive system (7-15 % versus 40-50 % for children), excrete lead more **rapidly**, and retain less in their bodies (1% versus 34 % for young children).

11. Polycyclic Aromatic Hydrocarbons (PAHs). In the interim **HRA**, all detected **PAHs** were assessed using the slope factor of **benzo-a-pyrene (BaP)**, the only PAH for which IRIS has a slope factor. Thus, all PAH concentrations were summed and calculated as **BaP**. It is generally agreed that using the **BaP** slope factor is excessively conservative because the other **PAHs** are not as carcinogenic as **BaP**. Again, this procedure provided a conservative estimate of the carcinogenic risk contribution of the **PAHs**. An examination of the environmental data shows that **BaP** was detected in only 4 of 114 air samples, while the other noncarcinogenic and less carcinogenic **PAHs** were found more frequently. In this **final** HRA, the carcinogenic evaluation of **PAHs** was conducted using carcinogenic equivalency factors (**CEFs**) recommended by EPA Region VII for use on **Superfund** sites. Under this approach, the slope factors for other **PAHs** are **modified** by rounding off to the nearest order of magnitude, based on their carcinogenic potential relative to **BaP**. The **PAHs** and their **CEFs** are presented below.

<u>Compound</u>	<u>CEF</u>
<b>benzo-a-pyrene</b>	1.0
benzo-a-anthracene	0.1
<b>benzo-b-fluoranthene</b>	0.1
<b>benzo-k-fluoranthene</b>	0.1
chrysene	0.01
dibenzo-a, <b>h-anthracene</b>	1.0
<b>indeno-1,2,3,c,d-pyrene</b>	0.1

This method of **evaluating PAHs** may slightly underestimate carcinogenic risk. However, due to the small number of detections of **PAHs** and their low **concentrations**, this method should have a small impact on assessing risk, if any at all.

## 12. Dioxins/Furans.

a. The potential exists for the formation of chlorinated dioxins and **furans** whenever there is uncontrolled/incomplete combustion of chlorinated **organics**. Although the 600 burning oil wells were uncontrolled, chlorinated **organics** were absent in the feed material. The formation of dioxins and furans therefore was improbable (see Table C-1). To validate

this hypothesis, the four PM<sub>10</sub> air filters that were analyzed for PAHs, and had the highest dibenzofuran levels, were analyzed for chlorinated dioxins and furans. The analysis consisted of total tetra through octa dioxin and furan isomers, and 2,3,7,8-tetrachlorodibenzo-p-dioxin (2,3,7,8-TCDD) and 2,3,7,8-tetrachlorodibenzofuran (2,3,7,8-TCDF). Table 13 lists the samples results.

TABLE 13. DIOXINS/FURANS RESULTS

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Military Hospital (23-24 Aug 91) - All below detection limit

Al Jubayl (24-25 July 91) - OCDD - 0.23 ng\*

Al Jubayl (30-31 July 91) - OCDD - 0.44 ng

**Khobar Towers (18-19 Sept 91) -**  
PeCDD - 2.7 ng  
HxCDD - 10.8 ng  
HpCDD - 4.2 ng  
OCDD - 2.1 ng  
HxCDF - 1.9 ng  
HpCDF -- 1.0 ng

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\* Concentrations are in total nanograms (ng) per extract.

OCDD - octochlorodibenzo-p-dioxin  
PeCDD - pentachlorodibenzo-p-dioxin  
HxCDD - hexachlorodibenzo-p-dioxin  
HpCDD - heptachlorodibenzo-p-dioxin  
HxCDF - hexachlorodibenzofuran  
HpCDF - heptachlorodibenzofuran

---

b. The recommended EPA Toxicity Equivalency Factors (TEFs) were used to evaluate the detected concentrations of dioxins and furans. The TEFs are derived by employing a multiple based on the relative toxicity of the isomer to 2,3,7,8-TCDD, which is the most toxic isomer. A 2,3,7,8-TCDD equivalent (i.e., the total of all isomers expressed as 2,3,7,8-TCDD) is then evaluated using the inhalation cancer slope factor for 2,3,7,8-TCDD. Table 14 presents the TEFs for dioxins/furans. Table 15 presents the 2,3,7,8-TCDD equivalent for the dioxins/furans detected in the Khobar Towers sample.

TABLE 14. TOXICITY EQUIVALENCY FACTORS FOR DIOXINS/FURANS

<b>Compound</b>	<b>EPA Recommended TEF</b>
<b>2378-TCDD</b>	1.0
other <b>TCDDs</b>	0.01
<b>2378-PeCDDs</b>	0.5
other <b>PeCDDs</b>	0.005
<b>2378-HxCDDs</b>	0.04
other <b>HxCDDs</b>	0.0004
<b>2378-HpCDDs</b>	0.001
other <b>HpCDDs</b>	<b>0.00001</b>
OCDD	0
<b>2378-TCDFs</b>	0.1
other <b>TCDFs</b>	0 . 0 0 1
<b>2378-PeCDFs</b>	0.1
other <b>PeCDFs</b>	0.001
<b>2378-HxCDFs</b>	0.01
other <b>HxCDFs</b>	<b>0.0001</b>
<b>2378-HpCDFs</b>	0.001
other <b>HpCDFs</b>	0.00001
OCDF	0

**PeCDF - pentachlorodibenzofuran**

**OCDF - octochlorodibenzofuran**

TABLE 15. TCDD EQUIVALENT FOR THE **KHOBAR** TOWERS SAMPLE

Khobar Towers **Sample** - 18-19 September 1991

<u>Isomer</u>	<u>Conc.*</u>	<u>TEF</u>	<u>Adjusted Conc.</u>
<b>PeCDD</b>	2.7 ng	0.5	1.35 ng
<b>HxCDD</b>	10.8 ng	0.04	0.43 ng
<b>HpCDD</b>	4.2 ng	0.001	0.0042 ng
OCDD	2.1 ng	0.0	0.0 ng
<b>HxCDF</b>	1.9 ng	0.01	0.019 ng
<b>HpCDF</b>	1.0 ng	0.001	<u>0.001 ng</u>
Total <b>2,3,7,8-TCDD</b> Equivalents			1.81 ng

\* Concentration is in total nanograms (ng) per extract.

c. The Kuwait **Military** Hospital site had no detections of any dioxin or **furan** isomer. The two samples from Al Jubayl only **contained** detectable quantities of OCDD, which has a TEF of zero because of its low toxicity. These three **samples**, therefore, were not evaluated because exposure to the air the samples represent would not present any significant health risk. The sample **from** Khobar Towers contained low levels of several dioxin and **furan** isomers, but none of the most toxic isomers, **2,3,7,8-TCDD** or **2,3,7,8-TCDF**. As stated above, the analysis performed on these samples was for total **tetra through octa dioxin/furan**, with specific analysis for the very toxic **2,3,7,8-TCDD** and **2,3,7,8-TCDF**.

d. Often when **dioxin/furan** analysis is conducted, the **2,3,7,8-substituted** isomers are also singled out for analysis due to their greater toxicity (see higher **TEFs** in Table 14). This **2,3,7,8-substituted** analysis was not done for our **samples**; therefore, all **dioxins** and **furans**

detected were considered **2,3,7,8-substituted**, and the higher **TEFs** were used in their evaluation. This is a very conservative assumption because combustion studies of dioxin/**furan** formation show that only a percentage of the isomers formed are **2,3,7,8-substituted**.

e. The calculated yearly inhalation risk, using this very conservative assumption, is **1.0E-08**. The volume of air collected for this sample was **2035.5 m<sup>3</sup>**. Therefore, the concentration of **2,3,7,8-TCDD** equivalents in air was **8.9E-10 mg/m<sup>3</sup>**. This concentration was applied to the standard **inhalation** intake formula used in this report. A monthly air intake of **5.65E-15 mg/kg-day** was calculated. This number was multiplied by 12 to determine a potential yearly intake, and then multiplied by the inhalation slope factor for **2,3,7,8-TCDD (1.5E+05)**, to obtain the cancer risk stated above.

f. This sampling and analysis was only intended to determine the potential presence of dioxins and **furans** and the health impact on DOD personnel. While this was not a major sampling effort, it still demonstrated the small potential for adverse health impacts from **dioxins** and **furans**.

## VI. DISCUSSION OF HEALTH RISK ASSESSMENT RESULTS.

### A. Cancer Risk Levels.

1. Cancer risk levels were calculated for the air pathway (i.e., inhalation of metals, volatile **organics** and **PAHs**), and for the soil pathway (i.e., incidental ingestion of metals, inhalation of soil **particulates**, included with the inhalation of metals above, and dermal absorption of metals) (see Appendix A-2).

2. Health risks associated with the dermal exposure route are included in this **final** report. When the interim **HRA** was prepared, EPA **draft** guidance on evaluation of the dermal route of exposure was not finalized. Although this guidance has now been released, only a few dermal absorption factors for soil are available (namely cadmium and lead). In addition, many other uncertainties **still** exist in the **evaluation** of this pathway. As discussed in the interim **HRA**, we still feel the dermal route of exposure is a small contributor to overall risk levels. This conclusion is based on the following factors.

a. Although few quantitative dermal absorption factors are available in the **scientific** literature (**Pb, 0.06-3.0%** and **Cd, 0.1-1.0%**), qualitative statements concerning **dermal** absorption such as poor (**Cr<sup>+3</sup>** and **Ni**), minor (**Vd**), Small (**Zn**), and not significant (**Be**) are readily found. Therefore, the absorption of **metal in** soil through the skin barrier and into the body where they would exert their systemic toxic effect is expected to be minor.

b. In addition to the **limited** number of **ABS** factors, the formula for calculating dermal exposure gives a value for absorbed dose rather than an estimated daily intake. The available chemical toxicity values used to evaluate risk are given on the basis of intake (i.e., administered dose not absorbed dose). Consequently, the toxicity values must be adjusted to an absorbed dose. In the process, they become more conservative, and the risk from the dermal exposure route becomes **greater** than from the inhalation and ingestion routes.

c. Only two contaminants of concern (**COCs**) (As and Be) have oral slope factors and can be **evaluated** for carcinogenic **dermal** impact. The use of **oral** slope factors to evaluate dermal cancer risk is yet another confounder **in** the assessment of the dermal pathway. This situation is due to the fact that there are no dermal slope factors. To assess the dermal pathway for these two carcinogens an absorption factor (**ABS**) of 5 percent was used. To evaluate **noncancer** risk **5 percent** was used for all **COCs**, except for cadmium, where 1.0 percent was used. A 5.0 percent **ABS** should be protective of human health because the only **COCs in** soil were metals.

3. The total predicted excess cancer risk in Kuwait, from all pathways and routes of exposure, for the period 5 May through 30 November 1991, ranged from **2E-6** (2 per 1,000,000) at Camp **Thunderrock** to **4E-7** (4 per 10,000,000) at the Ahmadi Hospital site. These **overall** cancer risk levels do not differ a great deal from those observed in Saudi Arabia. The excess cancer risks ranged from **2E-6** (2 per 1,000,000) at Khobar Towers to **4E-7** (4 per 10,000,000) at **Eskan** Village and **KKMC**. In addition, the two sites that represented potential background risk, **Eskan** Village and **KKMC**, had risk levels that were not **significantly** different from the **Ahmadi** Hospital site located **near** the fires.

4. All the predicted cancer risk levels were well within the EPA acceptable risk range of **1E-4** through **1E-6**. Generally, no action is taken at a **Superfund** site if the cancer risk levels are within this range. Action is **almost** always taken at a site when the risk is greater than **1E-4**. The cancer risks estimated for four of the sites **are** less than the **1E-6** level and would not require action. The risk levels at the other four sites are at the upper end of the acceptable risk range (i.e., **1E-6**) and probably would not warrant a response.

5. The cancer risk levels presented in Table 16 are for the **different** monitoring periods at each site. **The** length of the monitoring period for each site was determined by how long DOD troops were deployed at that location. When a majority of troops permanently vacated a site, monitoring activities at that location **ceased**. Therefore, to compare the risk levels **each** was, multiplied by a correction factor that equalized the monitoring **period** duration. This is a conservative method of equalizing risk for different monitoring period lengths because the shorter (i.e., collected in the earlier months) monitoring results were obtained when a larger number of **fires** were burning. These equalized risk levels show very similar **cancer risk** levels in both Kuwait and Saudi **Arabia**.

TABLE 16. EQUALIZED CANCER RISK **LEVELS** FOR THE **SEVEN** MONITORING SITES

Site	Number of Months Monitored	Multiplication Factor	Cancer Risk (CR)	CR Adjusted CR
Khobar Towers Al Khobar, SA	7	1	<b>2E-6</b>	<b>2E-6</b>
<b>Camp 1</b> Al Jubayl, SA	4	1.75	<b>7E-7</b>	<b>1E-6</b>
<b>Eskan</b> Village Riyadh, SA	3	2.3	<b>4E-7</b>	<b>9E-7</b>
<b>KKMC</b> SA	4	1.75	<b>4E-7</b>	<b>7E-7</b>
Camp Freedom/ Military Hospital Kuwait	7	1	<b>1E-6</b>	<b>1E-6</b>
Camp Thunderrock <b>Doha</b> , Kuwait	6	1.2	<b>2E-6</b>	<b>2E-6</b>
U.S. Embassy Kuwait City, Kuwait	3	2.3,	<b>7E-7</b>	<b>2E-6</b>
Ahmadi Hospital Ahmadi, Kuwait	3	2.3	<b>4E-7</b>	<b>9E-7</b>

SA = Saudi Arabia.

**KKMC** = King **Khalid** Military City.

6. The total cancer risk levels for the soil ingestion and **dermal** routes were calculated by taking the soil concentrations from **all** three sampling rounds and calculating a 95 percent UCL. This concentration was used to calculate an intake, and subsequent risk level, as **if** it were for a 1-month exposure. The risk level was then multiplied by the number of months

of exposure the troops had at that particular site, for a total risk level for that pathway/route of exposure. This type of sampling interval and method of calculating risk seems appropriate since soil is a more static medium than air. In addition, an examination of the soil data from the **three** rounds of sampling (see Appendix C) showed it to be very similar over time. Thus, this method of treating the data should not effect the calculated risk levels.

7. The calculation of cancer risk levels for the inhalation of **PAHs** was handled in the same way. Due to the small number of positive detections of **PAHs**, the data for each site was combined for the total monitoring period. The maximum PAH level detected was then used to calculate intake and risk for that site. The risk level was treated like a monthly exposure and multiplied by the number of months troops were at that site to estimate a total PAH risk level.

#### B. Noncancer Risk Levels.

1. Noncancer risk levels were calculated for the air pathway (i.e., inhalation of volatile **organics**) and for the soil pathway (i.e., ingestion of metals and **dermal** absorption of metals) (see Tables 3 through 10).

2. For **noncancer** risk (i.e., systemic **toxicants**), **EPA** considers acceptable exposure **levels** to be concentration levels to which the human population, including sensitive subgroups, may be exposed without adverse effects during a lifetime or part of a lifetime. The EPA defines this **acceptable** exposure level as an **RfD**. The EPA divides **RfDs** into additional categories, including chronic **RfDs** and subchronic **RfDs**. A chronic **RfD** applies to exposures ranging from 7 years to **life**, while a subchronic **RfD** applies to exposures ranging from 2 weeks to 7 years. A noncancer health impact is probable if the population exposure is in excess of an appropriate **RfD** [i.e., hazard quotient (**HQ**) or hazard index (**HI**) exceeds 1].

3. A number of uncertainty and modifying factors may be applied to **RfDs** when developing numerical values, including the following:

a. Variations in the general population (i.e., sensitive subpopulations such as children and the elderly) a factor of 10.

b. **Extrapolation** from animal to human data (i.e., interspecies variability) - a factor of 10.

c. Use of a lowest-observed-adverse-effect level (**LOAEL**) study in place of a **no**-observed-adverse-effect-level (**NOAEL**) study - a factor of 10 (for extrapolating between studies).

d. Inclusion of a modifying factor to reflect a qualitative professional assessment of additional uncertainties in the critical toxicologic study and in the entire database for the chemical - a factor of greater than 0 to 10.

These uncertainty/modifying factors give at least a ten-fold margin of safety in the **RfD**. Therefore, **HQs/HIs** in the 1 to 10 **range** should not present an unreasonable health risk, particularly for short exposure periods. In many instances, subchronic **RfDs** are not available. Consequently, a chronic **RfD** is used instead. Subchronic **RfDs** are generally 10 times higher than the chronic equivalent for the same compound. The use of chronic **RfDs** for subchronic exposures, therefore, adds an additional ten-fold margin of safety to the calculated **HIs**.

4. The total predicted noncancer risk in Kuwait (i.e., **HI**) for all pathways and routes of exposure, for the period 3 May through 30 November 1991, ranged from **1E+0** at the Military Hospital to **5E+0** at the Ahmadi Hospital. These **HIs** are not substantially different from those in Saudi Arabia, where they ranged from **5E-1** at **KKMC** to **2E+0** at **Khobar Towers** and **Al Jubayl**. As with the cancer risk levels, the overall noncancer risk levels do not appear to differ a great deal between Kuwait and Saudi Arabia. The two potential background sites (i.e., **KKMC** and **Riyadh**) did exhibit the lowest **HIs**, but the risk levels were comparable to all the other sites.

5. **All of** the monitoring sites except **KKMC** and **Riyadh** had calculated noncancer risk levels **greater** than 1. The **HIs** exceeded unity (i.e., the chronic or **subchronic RfDs**) by a factor ranging from 2 to 5. These **HIs** only exceeded the **chronic/subchronic RfDs** by a relatively small amount, however. In addition, the exposure periods were short relative to the time period covered by the respective **RfDs**. The DOD personnel were exposed to oil fire smoke for a maximum of 9 months to a minimum of 1 month.

6. The noncancer risk levels presented in this **HRA** represent averages for the various periods of exposure. They were derived by first calculating monthly intakes and monthly risks. The risks for the various total exposure periods were then obtained by averaging the risks for the individual months of exposure. **This** methodology was adopted because the intake for **noncancer** effects is based on the exposure duration and an averaging time that is equal to the exposure duration. Addition of risks would be the equivalent of adding exposures from different places, since the indices calculated represent a discrete exposure for a discrete time period.

7. Inhalation of volatile **organics**, in particular benzene, contributed to over 99 percent of the noncancer risk at all monitoring sites. **Kuwaiti** crude oil is one potential source of the benzene. Other potential benzene sources include:

- a. industrial chemical production (i.e., ethylbenzene, cyclohexane, and cumene);
- b. solvent use;
- c. gasoline additive; and
- d. auto exhaust.

Therefore, it is not possible to identify the source of the benzene detected in the air samples.

8. In the interim **HRA**, the primary source of **noncancer** risk was from inhalation of chromium. **All** of the chromium in the interim report was **evaluated** as **Cr<sup>+6</sup>**, which has an inhalation **RfC**. The recent trip to Kuwait and Saudi Arabia, however, indicated that the chromium detected in the air and soil was in the noncarcinogenic trivalent form. Currently, no chronic or subchronic **RfC** for **Cr<sup>+3</sup>** is available. Therefore, a noncarcinogenic inhalation risk was not calculated for **Cr<sup>+3</sup>**.

9. **As** stated in the interim HRA, **Kuwaiti** crude oil does not contain chromium as an impurity. Therefore, the chromium found in the **air** and soil samples was from a source other than the oil fires. Other potential sources of chromium are the natural soil content (average content 200 ppm, with a **range** of **5-1000** ppm) and industrial pollution such **as** metal plating, protective coatings, inorganic pigments, tanning, dye fixing, gasoline additive, catalyst, photography, and ceramics. Due to the dry and windy conditions prevalent in the region, the chromium associated with the soil, from either natural or industrial sources, became airborne and was collected on the air **sampling** filters. Additional airborne industrial sources of chromium were also collected by the air sampling equipment. These **nonfire**-related activities **are** believed to be the source of the chromium.

### C. Incremental Versus Absolute Risk.

1. The risk numbers presented in Tables 3 through 10 are for the absolute (i.e., total) risk to DOD personnel for the exposure period 5 May through 3 December 1991 for all chemicals detected by the environmental sampling, regardless of source. There were certain exceptions noted due to problems **in** the assessment methodology or database (i.e., lack of toxicity and/or environmental data, lack of modeling results etc.). No attempt was made to separate the natural and anthropogenic **contaminant** levels from oil **fire-generated** contaminants, other than to qualitatively state what potential sources were available.

2. Historical data was available for total suspended **particulates (TSP)** and certain metals for 1988 at several sites in Kuwait (i.e., Kuwait City, Shuwaikh, and Jahra). These two major classes of contaminants/products were expected to be produced by the oil **fires**. A general comparison of the historical levels for these contaminants with the levels determined **during** this project revealed the 1991 TSP levels were below 1988 levels for June and July and above for May. In addition, the 1991 values for chromium, vanadium, and nickel were very similar to 1988 values, and zinc levels were much higher in 1988 than 1991. Both vanadium and nickel were considered potential oil fire-related contaminants due to their presence in the crude oil. The comparisons suggest that the oil well fires did not make a large contribution to ambient concentrations of TSP, chromium, vanadium, nickel, and zinc.

3. While conducting the monitoring activities in Kuwait and Saudi Arabia, there were many sources of industrial **pollution** observed that could have contributed to the chemical loading of the environment. Numerous trash **fires** were observed in Kuwait City. Fires were set in an attempt to reduce the volume of waste material collected. These **fires**, many of which were near sampling sites (i.e., Camp Thunderrock, **Military Hospital**, and U.S. Embassy) probably contained many of the same pollutants that were **identified** as **COCs in this** project. Oil industry activities also emitted toxic organic chemicals into the atmosphere. Hydrocarbons, **particularly PAHs, n-alkanes, and VOCs** from storage tanks are likely to be the major chemicals **released**. In Kuwait, these emissions would initially be expected to be **small**, due to the cessation of oil **producing/refining** activities during and immediately after the war. Shortly after the war, however, **refining** output increased rapidly and its contribution to air pollution may have increased as well. Obviously the **refining/industrial** activities in Saudi Arabia would have the same impact on the environmental monitoring as in Kuwait, but without the loss of activity during the war. Vehicular **traffic** was another source of background anthropogenic contamination. The primary contaminants include lead, hydrocarbons, ozone, carbon monoxide, and oxides of nitrogen. Again, **in** Kuwait these anthropogenic levels would have been lower right after the war and rose rapidly as vehicular traffic increased.

4. In order to place the Persian Gulf wntaminant exposure levels and resultant risks in proper perspective, the air quality from various urban and industrial **areas** was examined. Appendix B of the interim and fmal reports, contains a complete assessment of the exposure data and trends analysis. Included **in** this discussion is a comparison of VOC levels from Kuwait and Saudi Arabia with levels found in selected U.S. cities representing different geographic and climatic regions. The VOC levels from Phoenix, Arizona; Los Angeles, California; Houston, Texas; and **Philadelphia**, Pennsylvania, were compared to the levels from Khobar Towers, Camp Thunderrock, and the Kuwait **Military Hospital** sites which had the longest monitoring periods. Overall, the median VOC concentrations for benzene, toluene, ethyl benzene, and the xylenes, from the Kuwait and Saudi Arabian sites, were just near or below the respective concentration values **for** U.S. cities.

5. A comparison of the risk levels between the background month and the **first** sampling month show very similar levels of risk (see Table 17). In many cases, the background month has higher risk levels than the fire month. These risk numbers give credence to the idea that the **fires** had a small theater-wide impact, and did not **greatly** contribute to the risk levels for the area.

TABLE 17.

Site	Background Risk		Fire Risk*	
	Cancer	Noncancer	Cancer	Noncancer
<b>Khobar</b>	M - <b>6E-8</b>		<b>1E-7</b>	
	v - <b>8E-8</b>	<b>2E+0</b>	<b>2E-7</b>	<b>4E+0</b>
Military Hospital	M - <b>6E-8</b>		<b>9E-8</b>	
	V - <b>9E-8</b>	<b>2E+0</b>	<b>2E-8</b>	<b>6E-1</b>
<b>Doha</b>	M - <b>1E-7</b>		<b>7E-8</b>	
	V - <b>2E-7</b>	<b>5E+0</b>	<b>2E-7</b>	<b>3E+0</b>

M = Risk from metals on PM., filters.

V = Risk from volatile **organics**.

\* Fire risk includes natural and anthropogenic risk also, since these **cannot** be separated.

The **fire** risk numbers are from the **first** month of valid monitoring data when the most fires were burning. **Khobar**, **Military Hospital**, and **Doha** (Camp Thunderock) were the only sites with monitoring during November.

6. In addition to the anthropogenic background contamination that becomes airborne and/or associated with the soil, there are natural metals that are an integral part of the soil/sand matrix. Table 18 shows the usual range and average level in soils for many of the metals of concern in this assessment. With the meteorological and climatic conditions that exist in the region, it is apparent how these soil metals can become airborne and captured on air sampling filters. The natural metals in soil may have contributed to the risk values which were **calculated**.

TABLE 18. **ELEMENT** VARIATIONS IN SOILS (in ppm of Dry Material)

Element	Average	Usual Range
Arsenic	5.0	1-50
Beryllium	6.0	
Cadmium	0.5	0.01-0.70
Chromium	200	5-1,000
Mercury	0.03	0.03-0.3
Nickel	40	5-500
Lead	10	2-200
Vanadium	100	20-500
<b>Zinc</b>	<b>50</b>	<b>10-300</b>

## VII. HEALTH RISK - RELATED STUDIES.

### A. Incorporation of the Biological Surveillance Initiative Results with the Health Risk Assessment Results.

1. The Biologic Surveillance Initiative (**BSI**) had two objectives. The first objective was to quantify exposure to environmental contaminants by measuring biological markers of exposure and internal dose in DOD troops (1 lth Armored Cavalry Regiment) which deployed from Germany to Kuwait. These objective measurements of exposure and dose could then **serve** to corroborate or counter the exposures derived from the environmental concentrations used in the risk assessment. Thus; the BSI effort was an attempt to validate the calculated human exposure levels made in the **HRA**. The second objective was to detect changes in the troop cohort's well-being through selected objective and subjective measures of health. The rationale for this component of the BSI was that any positive **findings** in the surveillance population could lead to the early identification of special **health care** needs and/or requirements for further surveillance in other potentially exposed soldiers. The first objective and validation or rejection of the results of the **HRA** will be discussed in this section.

2. Typically, actual biological measures of dosage and tangible health effects are not **evaluated** concurrently with the **environmental** measurements used to predict health risk in an **HRA**. The **BSI** was **an** attempt to accomplish this and validate the **HRA**. Data collection for the BSI was conducted in the three stages as shown below and designated as KOF (Kuwait Oil Fire).

KOF1 - June 1991, before deployment, in Germany.

KOF2 - August 1991, during deployment, in Kuwait.

KOF3 - October 1991, post deployment, in Germany.

The BSI consisted of the following segments:

- Comprehensive (general) Questionnaires.
- Supplemental Questionnaires - At the time of **specimen** collection (blood or urine).
- Pulmonary Function Tests (**PFTs**).
- Personal Diaries.
- Metals Analysis (blood and urine).
- Volatile Organic Compounds (**VOCs**) Assay (**blood**).
- Sister Chromatid Exchange Frequency Assay (**SCEs**).
- Polycyclic Aromatic **Hydrocarbon (PAH)-Deoxyribonucleic Acid Adduct** Assay (DNA Adducts).
- Urinary **Tetrols** Assay (in progress).

3. The results discussed here will only include the objective laboratory findings that can be compared to the **HRA** (i.e., metals, **VOCs**, SCE, DNA Adducts). **All** other results are discussed in Appendix **F**.

a. Metals Analysis. Arsenic and mercury levels were below detection limits and will not be further addressed. In general, the data indicate the presence of normal levels of metals **in** all specimens before, during, and after deployment. The only noticeable difference was found in the blood lead levels, where there **appears** to be a slight elevation of lead levels in blood **collected** in Kuwait. However, this difference is **statistically insignificant**, and all the lead concentrations are within the expected "normal" range for healthy young adults.

b. Volatile Organic Compounds Assay. In general, the results for **VOCs** measured in the three phases of this **surveillance** (**KOF1**, **KOF2**, and **KOF3**) do not show substantial phase-related differences. In most cases, the results were similar to and within the range of levels determined by the National Center for Environmental Health to be in their U.S. normal reference range.

c. Sister Chromatid Exchange (SCE) Frequency Assay. SCE, a nonspecific indicator of recent and near-past genotoxic exposures, did show **significantly** different values during deployment (**KOF2**) and post deployment (**KOF3**), compared to pre-deployment (**KOF1**). No health effects have been attributed to SCE changes, and they have been associated with a large number of exposures -in humans (i.e., chemicals, drugs, medical conditions, coffee, smoking, radiation, sleep deprivation, etc.).

d. Polycyclic Aromatic Hydrocarbon - Deoxyribonucleic Acid **Adduct** (DNA) Assay. DNA **Adducts** are **a measure** of exposure to **PAHs** by measuring the covalent bonding of PAH molecules with DNA. **PAH Adducts** were higher pre (**KOF1**) and post (**KOF3**) deployment, compared to time period of deployment in Kuwait (**KOF2**). The levels of PAH **Adducts** would appear to be related to a very low level of exposure to **PAHs** for soldiers deployed to **kuwait**. As with **SCEs**, there are many other factors that can effect potential PAH exposure such as diet, smoking, or occupation.

## B. Radiation Exposure.

1. The exposure of DOD troops to radiation from depleted uranium (**DU**) and other sources (i.e., oil **fires** and **earth crustal** material) was a **concern**. This **final HRA** attempted to **resolve** the theater-wide radiation concern by analyzing **PM<sub>10</sub>** air **filters** collected **from** all monitoring sites (for details of the laboratory analysis, data interpretation, and risk analysis see Appendix **H**). This radiation risk analysis does not apply to subgroups such as tank maintenance workers or soldiers injured with DU shrapnel.

2. Two hundred and **fifteen** PM<sub>10</sub> air **filters** were analyzed for gross alpha and gross beta-gamma activities per unit volume of air. These analyses were also used as a screening process for DU since it is primarily **an** alpha emitter. The average **radionuclide** concentrations of the samples were similar to the background concentration. Background was determined using the air samples collected at **Eskan** Village, Riyadh, and **KKMC**. These **areas** were located upwind from the oil **fires**.

3. There was negligible risk to the DOD population-at-large **in** Kuwait and Saudi **Arabia** from ionizing radiation based on the following **reasons**.

a. The **release** of **natural radionuclides** in the U.S., from the combustion of oil for the generation of electricity appears to be higher than the release of natural radionuclides from the oil well fires **in** Kuwait.

b. Dose assessments **calculated** using the measured **radionuclide** concentrations from the air **filter** samples are well below regulatory limits for the general public.

c. No radioactive contamination above background levels was detected **in** the air samples **analyzed** for gross alpha and beta radiation, which would include **radiation** from DU.

## VIII. CONCLUSIONS.

### A. Health Risk Assessment.

1. The total predicted excess cancer risk resulting from exposure to the Persian Gulf environment during 5 May 1991 through 3 **December** 1991 **ranged** from **2E-6** (2 per **1,000,000**) to **4E-7** (4 per **10,000,000**). These risk levels represent the eight permanent monitoring sites and include all exposure routes (i.e., inhalation, incidental ingestion, and dermal contact). These cancer risk levels are well within the EPA range of acceptable risk [**1E-4** (1 per 10,000) through **1E-6** (1 per 1,000,000)].

2. Excess **cancer** risk levels do not differ **significantly** between the monitoring sites **in** Kuwait, near the oil **fires**, and the monitoring sites in Saudi Arabia. **In** fact, there is very little difference **in** the **cancer** risk levels between any of the sites monitored.

3. The total predicted **noncancer** risk (i.e., **HI**s) for **all** pathways and routes of exposure ranged **from 5E+0** to SE-1. **As** with cancer risk levels, the noncancer risk levels do not greatly differ between any of the monitoring sites in Kuwait and Saudi Arabia. The **HI**s for noncancer risk did exceed the EPA level of **concern** of **1E+0** at all but one of the monitoring sites, however. **Inhalation** of volatile **organics**, in particular benzene, contributed to over 99 percent of the noncancer risk of **all** monitoring sites. Benzene contamination is believed to **be from** anthropogenic sources, as well as from the oil **fires**.

4. Background and historical environmental monitoring data from the Persian Gulf region, compared with industrial pollution data from various **areas** of the world, indicate that much of the risk associated with the region is not oil **fire** related, but is the result of **regional** background contamination.

B. **Specific Components**. Refer to each respective appendix for a list of conclusions.

1. Ambient Air Sampling and Air Pathway Analysis. Refer to Appendix B for a full presentation of the ambient air sampling and air pathway analysis.

2. Soil Sampling and Soil Pathway Analysis. Refer to Appendix C for a detailed and comprehensive discussion of the soil sampling and soil pathway analysis.

3. Industrial Hygiene Air Sampling. Refer to Appendix D for a complete discussion of the industrial hygiene air sampling.

4. Analytical Methodology and **Quality Assurance**. The analytical methodology and quality assurance procedures used throughout this study are detailed in Phase 1 and Phase 2, Appendix E. Phase 2 of Appendix E also contains the analytical data for the return trip (1993).

5. Biologic **Surveillance** Initiative. A technical summary, available results, and status report on the biologic surveillance initiative, to include methodology and schedule, are presented **in** Appendix F. This phase of the study will require additional time to complete; therefore, some of the results and **data** interpretation will not be available until an addendum to the final report is released.

6. Electron Microscopy Analysis. To help determine the source of the **particulates** collected during air sampling; and the associated contaminants, an electron microscopy analysis was performed on **sand** and ambient air samples. Parameters such as particle size distribution and chemical makeup were determined for ambient air and sand samples. The methodology employed and sampling results for this study are presented in Appendix **G**.

7. **Radiological** Analysis. To evaluate the potential theater-wide risk from radiation (i.e., depleted uranium, natural background, and oil **fire** related) air sampling **filters** were analyzed for radioactivity. For details of the methodology, results, and conclusions of this study see Appendix H.

8. Response to Reviewer Comments. Appendix I contains the response to reviewer **comments, indicating their acceptance**, rejection, or incorporation.

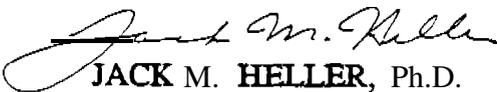
## **IX. RECOMMENDATIONS.**

A. Update the **HRA**, to include air modeling study results for DOD troop sites where no monitoring was conducted, when the modeling and troop location information becomes available.

**B.** Continue to separate natural and anthropogenic background risk from oil **fire-related** risk as more background, modeling, and particle analysis information becomes available.

**C.** Continue to update the **HRA** as new toxicologic information and cancer and noncancer risk assessment methodology becomes available.

**D.** Continue to **incorporate** new information and data from the Biologic Surveillance Initiative with the **HRA** results to refine the **findings** and conclusions of the study.



**JACK M. HELLER, Ph.D.**

Master Consultant

Environmental Scientist

Waste Disposal Engineering Division



**WILLIAM E. LEGG**

MAJ, MS

Kuwait Task Force **Leader**

APPROVED:



**ARTHUR P. LEE**

MM, MS

Chief, Health Risk Assessment Branch

Waste Disposal Engineering Division



**JOEL C. GAYDOS**

COL, MC

Medical Director

