HUMAN HEALTH RISK ASSOCIATED WITH BURNING AS A SPILL COUNTERMEASURE

by

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1.0 SUMMARY AND APPROACH

This paper presents a streamlined human health risk assessment to help evaluate the feasibility of burning as a spill countermeasure in a near-shore environment. Smoke inhalation and long-term ingestion of contaminated soil are evaluated. Estimates of particulate matter and chemical concentrations in smoke were derived using data from laboratory and mesoscale burns, and were supported by air dispersion modeling of a hypothetical burn of 10,000 gallons of crude oil.

Using reasonable maximum exposure duration and upperbound estimates of toxicity of polycyclic aromatic hydrocarbons (PAHs) in smoke, it is shown that estimated health risks at modeled exposure points are below levels of concern established by United States Environmental Protection Agency (U.S. EPA) for protection of public health from exposure to hazardous chemicals released from a source to the environment. However, concentrations of particulate matter (PM) could exceed U.S. national and state ambient air quality criteria by a factor of about 2 on a temporary and transient basis. These temporary exceedances of a PM standard could be considered acceptable, given that the countermeasure is implemented for overall protection of the environment. Possible synergistic effects of inorganic air pollutants on PAH toxicity are also discussed.

This human health risk evaluation is conducted using methodology developed by U.S. EPA (1989a). It describes the chief pathways by which humans could be exposed to potentially hazardous chemicals during in smoke, estimates exposure concentrations and intake (dose) of chemicals of concern, provides conservative (health-protective) chemical toxicity values, and estimates carcinogenic and noncarcinogenic risks associated with exposures to those chemicals. Conservative (health-protective) assumptions are used so that potential risk will not be underestimated.

2.0 HUMAN EXPOSURE PATHWAYS

The chief pathways by which humans may be exposed to chemicals of concern during an oil burn are inhalation of airborne particulate matter (soot) and incidental ingestion of soot or contaminated soil. Other possible exposure routes are dermal absorption of chemicals from skin contact with soot and ingestion of contaminated

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seafood. Dermal absorption is expected to be negligible, because organic compounds bind tightly to soot and they are not expected to partition to skin in significant amounts, especially under short-term exposure conditions as would occur during an oil burn. The seafood ingestion pathway is considered negligible because burning considerably reduces the quantity of oil that could result in contamination of aquatic species compared to other control measures. Direct human contact with oil residual is not evaluated because the opportunity for exposure of the public is low, and the potential for exposure is much reduced if the oil is burned. Therefore these pathways are not considered important in assessing a burn/no burn decision based on potential human health risks.

3.0 CHEMICALS OF CONCERN

Chemicals of concern for human health risk assessment are those chemicals that, at certain exposure levels, could have significant adverse health effects. The greatest concern in a burn scenario is for exposure to contaminants in the smoke plume. Therefore, this section focusses on identifying chemicals of concern in the plume to which shoreline residents might be exposed.

3.1 Constituents of Crude Oil

Crude oil is a complex mixture of thousands of organic compounds, most of which are saturated hydrocarbons or aromatics. Table 1 lists concentrations of C_{12} - C_{24} straight-chain alkanes (n-paraffins) and aromatic hydrocarbons in the API reference oils (Kuwait Crude and Louisiana Crude) and in Alberta Sweet Crude (PAHs only). Benzene and alkyl-substituted benzenes are present in the crude oil at concentrations of 8 to 9 percent, and naphthalenes are present at concentrations of 0.5 to 0.8 percent. PAHs, many of which are known to be toxic or carcinogenic, are present in much lower concentrations; total PAHs analyzed constitute less than 0.1 percent of the total hydrocarbons analyzed in each sample.

Table 1 does not include the lower molecular weight saturated hydrocarbons (C_1 - C_{11}) that are present in crude oil. Pentanes through decanes tend to dominate in this group. These compounds are volatile and may be lost by evaporation.

Compounds containing sulfur and nitrogen, and metals such as vanadium, nickel, and zinc, are also present in low concentrations ranging from less than 10 ppm to several hundred ppm depending on the metal and the crude. The metals occur primarily as large, high molecular weight, stable organometallic complexes that are not readily soluble or biologically available. Other trace metals are also found in crude oil, generally in concentrations much less than 1 ppm.

3.2 Constituents of Concern During Burning

Only some of the constituents of crude oil are known to be toxic or carcinogenic. For example, many straight-chain and cyclic hydrocarbons and some of the PAHs have no known adverse human health or ecological effects. Even fewer of the

TABLE 1
CONCENTRATIONS OF N-PARAFFINS AND AROMATIC HYDROCARBONS IN CRUDE OILS, PPM

Compound	Kuwait Crude(1)	Louisiana Crude(1)	Alberta Sweet Crude(2)
Total C_{12}-C_{24} n-paraffins	40,000	39,800	
Benzenes	80,000	92,000	
Naphthalene	400	400	
1-Methylnaphthalene	500	800	
2-Methylnaphthalene	700	900	
Dimethylnaphthalenes	2,000	3,600	
Trimethylnaphthalenes	1,900	2,400	
Biphenyls	<100	<100	
Acenaphthylene			13
Acenaphthene			57
Anthracene			11
Fluorenes	<100	200	209
Phenanthrene	26	70	150
1-Methylphenanthrene	-	111	-
2-Methylphenanthrene	89	144	-
Fluoranthene	2.9	5.0	6
Pyrene	4.5	3.5	17
1-Methylpyrene			39
Benzo(a)anthracene	2.3	1.7	-
Chrysene	6.9	17.56	30
Triphenylene	2.8	10	30
Benzo(ghi)fluoranthene	<1	1	-
Benzo(b)fluoranthene	<1	<0.5	4
Benzo(j)fluoranthene	<1	<0.9	-
Benzo(k)fluoranthene	<1	<1.3	-
Benzo(a)pyrene	2.8	0.75	-
Benzo(e)pyrene	0.5	2.5	5
Perylene	<0.1	34.8	-
Benzo(ghi)perylene	<1	1.6	-
2-Methylcholanthrene	-	-	3

(1) From Neff, J.M. and J.W. Anderson. 1981.
(2) From Benner, et al. 1991.

potentially hazardous constituents of crude oil are present in smoke emanating from a burn. The following paragraphs describe which chemicals are of concern for risk assessment and which are not.

VOCs: Many of the low-molecular-weight constituents that are potentially hazardous, such as benzene, toluene, n-hexane, and naphthalenes, are readily volatilized and combusted during burning. These compounds are released to air during an oil spill and can contribute to acute health effects in humans (such as nausea and headache) if exposure to high concentrations occurs. High concentrations of volatile organics have been shown to occur within 200 meters from mesoscale burns (up to 5,000 gallons or 120 barrels; Fingas et al. 1993). Since the air concentrations of these compounds are much reduced during burning, burning will reduce risk of exposure of shoreline receptors to potentially hazardous concentrations of volatile organics. Since burning an oil spill results in lower air concentrations of volatile organic compounds compared to other remedial actions, these compounds are not considered chemicals of concern for evaluating a burn/no-burn decision.

Metals: Because metals occur in low concentrations in crude oil and are bound up in organometallic complexes that are largely retained in the burn residue (to which exposures are expected to be minimal), metals are not chemicals of concern for risk assessment in evaluating the burn countermeasure.

PAHs: The primary chemicals of concern from burning are toxic or carcinogenic PAHs in the plume that are not destroyed during burning or that may be created during incomplete combustion. (Most PAHs in crude oil are destroyed by combustion.) Table 2 lists PAHs in particulates and residue measured in a laboratory scale burn of Alberta Sweet Crude (Benner et al. 1990). The PAHs are grouped as carcinogenic, noncarcinogenic (toxic), and other. "Other PAHs" are PAHs that either have not been tested, have no known adverse effects, or for which data is insufficient to demonstrate toxicity. These "other PAHs" are not evaluated in the human health risk assessment.

The relative proportions of the PAHs shown in Table 2 (derived from Benner et al. 1990) are comparable to those measured in mesoscale experiments conducted near Mobile, Alabama sponsored by the U.S. Minerals Management Service (Fingas et al. 1993). However, a greater number of individual PAHs were identified in the laboratory experiments. Acenaphthene and phenanthrene were detected in the greatest concentrations in both the laboratory and mesoscale experiments. Toxic or carcinogenic effects for these compounds have not been established. Pyrene, fluoranthene, and other noncarcinogenic PAHs with known non-cancer effects were detected in relatively lower concentrations. Carcinogenic PAHs were detected in much lower concentrations in both the laboratory and mesoscale experiments. Benzo(a)pyrene and benzofluoranthenes were detected in both series; additional carcinogenic PAHs were identified in the laboratory experiments. Therefore, the data in Table 2 are considered to be a reasonable representation of PAH concentrations in smoke from an oil burn.

TABLE 2
CONCENTRATIONS OF CARCINOGENIC AND NONCARCINOGENIC PAHS (VAPOR AND PARTICULATE) PRODUCED BY COMBUSTION OF ALBERTA SWEET CRUDE OIL (1)
(ug of PAH/g of sample)

PAHs	Crude Oil	Soot (2)		Residue (2)	
		Minimum	Maximum	Minimum	Maximum
Carcinogens (3)					
benzo(a)anthracene		155	260		
chrysene/triphenylene	30	78	260	24	34
benzofluoranthenes (4)		260	430		
benzo(a)pyrene		190	310	3	4
2-methylcholanthrene	3			3	3
indeno(1,2,3-cd)pyrene		120	200		
Totals		803	1460	30	41
Non-Carcinogens (5)					
acenaphthene	57			10	15
fluorene	59			35	47
2-methylfluorene	150			110	160
anthracene	11	210	440	13	19
fluoranthene	6	580	950	11	22
pyrene	17	610	1070	25	30
benzo(a)fluorene		70	70		
benzo(b)fluorene		70	70		
1-methylpyrene	39	56	56	16	19
Totals		1596	2656	220	312
Other (6)					
acenaphthylene	13	1740	1740	26	54
phenanthrene	150	910	1820	120	140
3-methylphenanthrene		16	36		
2-methylphenanthrene		21	40		
1-methylphenanthrene		12	33		
methylphenanthrenes	370			330	330
dimethylphenanthrenes	500			520	520
acephenanthrylene		330	520		
perylene		5	56		
benzo(e)pyrene	5	90	140	6	6
benzo(ghi)perylene		120	250	2	2
coronene		45	252		
Totals		3289	4887	1004	1052
Grand Total (ug PAH/g sample)		5688	9003	1254	1405

(1) From Brenner et al., 1990
(2) Thickness of oil slicks ranged from 2 mm to 30 mm.
(3) Based on identification of carcinogenicity by U.S. EPA (IRIS) or NAS 1972.
(4) Benzofluoranthenes are grouped together as carcinogens, although some isomers are not known to be carcinogenic (such as benzo(ghi)fluoranthene).
(5) Based on EPA (IRIS) and assumption that methylated and benzo-compounds also have non-cancer effects.
(6) No or insufficient evidence of carcinogenicity or toxicity.

Chlorinated Dibenzo-p-dioxins and -Dibenzofurans: Some compounds within this group are highly toxic and may be produced in small quantities during incomplete combustion of hydrocarbons in the presence of chlorine, in facilities such as municipal waste incinerators. Results from trace analyses of particulate matter from mesoscale burns showed that these compounds were not produced during combustion of crude oil (Fingas et al. 1993).

Inorganic Air Pollutants: PM, NOx, and SO_2 that are emitted during an oil burn can be respiratory irritants and aggravate existing respiratory or cardiac ailments in humans. There is also some evidence that they may enhance carcinogenic effects of PAHs (see Health Effects of PAHs, below). However, it appears that ground-level concentrations of NO_x and SO_2 are not likely to exceed short-term air quality criteria within the area of plume impact. For example, Table 3 lists modeled maximum ground-level air concentrations of these pollutants from a hypothetical burn (10,000 barrels) and compares them to U.S. National Ambient Air Quality Standards and the most limiting coastal state standard.

The hypothetical burn scenario used in modeling assumes that 10,000 barrels are ignited as a single event, rather than being separated by booms into smaller pools for controlled burning. Air dispersion modeling of a large single event is likely to predict greater emissions impacts at greater distances from the source than would be predicted for several smaller sources. As shown in Table 3, only modeled PM concentrations exceeded national or state criteria for short-term air concentrations at distances between 1200 and 4500 meters from the hypothetical burn site.

Note: The air dispersion modeling was done using SCREEN2; input assumptions and modeling results are not detailed in this paper but are available upon request from the authors.

The modeled concentrations are consistent with the findings of Fingas et al. (1993), where it was concluded that ground-level air concentrations of combustion gases, including SO_2, were not of concern during mesoscale oil burn tests. During an emergency situation in which oil spill countermeasures are being implemented, short-term exceedances of air quality criteria for PM could be considered acceptable given the overall benefit of burning the oil to reduce impacts to the environment.

In conclusion, chemicals of concern for quantitative risk assessment are the carcinogenic and noncarcinogenic PAHs shown in Table 2.

4.0 HEALTH EFFECTS OF PAHs

4.1 Carcinogenic PAHs

U.S. EPA has identified the following PAHs as carcinogenic, based primarily on experimental evidence in animals: benzo(a)anthracene, benzo(a)pyrene, benzo(b)-fluoranthene, benzo(k)fluoranthene, chrysene, indeno-(1,2,3-cd)perylene, and

TABLE 3

MODELED MAXIMUM 24-HOUR AIR CONCENTRATIONS OF CRITERIA POLLUTANTS COMPARED TO AMBIENT STANDARDS, $\mu g/m^3$

Constituent	Modeled 24-hr Maximum Concentration in Plume (1)	U.S. National Ambient Air Standard	Most Limiting State Standard
PM	323 806 (1 hr)	150 (24 hr)	50 (annual)
CO	10 (8 hr)	10,000 (8 hr)	10,000 (8 hr)
NO_x	120	100 (annual)	100 (annual) 1,300 (3 hr)
SO_2	65	365 (24 hr)	365 (24 hr)

(1) 24-hour maximum, except where noted. Results of modeling a 10,000-barrel burn using emission factors estimated from Kuwaiti oil field fires showed maximum concentrations at 1540 m downwind of the source. Modeling approach and assumptions are not detailed in this paper but are available upon request from the authors.

dibenzo(a,h)anthracene. Other carcinogenic PAHs listed in Table 2 are identified as carcinogenic by NAS (1972). Benzo(a)pyrene (BaP) is one of the most carcinogenic of the PAHs.

In laboratory studies, ingestion, inhalation, dermal, and subcutaneous administration of BaP to mice, rats, and hamsters have produced a variety of tumors in the kidney, stomach, lungs, respiratory tract, and skin. Tumors have also been produced in some but not all studies performed in primates. Most tumors occur at the point of contact.

The carcinogenicity of some PAHs in humans is supported by historic evidence from workers exposed for many years to soot, other products of coal combustion and gasification, and lubricating oils under unhygienic working conditions (e.g., saturated clothing worn for long periods of time). Lung cancer has also been associated with occupational exposures at coal gasification plants and coke ovens and with cigarette smoking.

It should be noted that the doses given to laboratory animals are many thousands of times higher than conservative estimates of doses to humans from exposure to smoke from an oil burn. For example, daily doses given to laboratory animals in the experiments used to quantify the cancer potency of BaP ranged from approximately 2×10^{-2} to 1 milligram BaP per kilogram body weight per day (mg/kg-day) (U.S. EPA IRIS). These high doses can be compared to the much lower dose of 6.6×10^{-6} mg/kg-day total carcinogenic PAHs estimated for smoke inhalation and particulate ingestion from oil burning (this value is the sum of doses shown later in Table 6). As shown in Section 7, these low doses in humans are not likely to produce a measurable increase in cancer incidence.

4.2 Noncarcinogenic PAHs

The PAHs acenaphthene, fluoranthene, fluorene, and pyrene have been shown to affect the liver, spleen, kidney, and blood in laboratory mice. Pyrene has adverse effects at somewhat lower doses than the others, and is therefore the most conservative (health-protective) measure of potential toxicity. The lowest dose of pyrene that elicited toxic effects in laboratory animals was 125 mg/kg-day (U.S. EPA IRIS). No effect was seen at 75 mg/kg-day. This dose is many thousands of times higher than those to which the public would be exposed during burning of an oil spill. The maximum dose estimated in Table 7 for human exposures is 0.02 mg/kg-day (child eating soot for 5 days). This dose would not be expected to have adverse effects. Furthermore, the acute toxicity of pure PAHs appears low when administered orally or dermally to rats or mice (U.S. EPA IRIS).

4.3 Effects of Other Air Pollutants and Sunlight on PAHs

There is some epidemiological and experimental evidence to suggest that exposure to inorganic air pollutants such as SO_2, NO_x, and ozone may potentiate the carcinogenic effects of PAHs. It is possible that irritation of respiratory passages

by inorganic air pollutants increases the sensitivity of respiratory tissues to carcinogenic PAHs. Some supporting evidence from laboratory experiments is available. For example, in one experiment (Laskin et al. 1970, reported in NAS 1972), simultaneous exposure to SO_2 and BaP appeared to enhance the tumor rate in rats but not in hamsters. Exposure was to 10 ppm SO_2 for 6 hours/day plus 10 mg/m^3 BaP and 3.5 ppm SO_2 for 1 hour/day. These concentrations are considerably higher than those to which humans would be exposed at distances of approximately 1000 to 2000 meters from a burn site, and the exposure conditions do not include weathering, photooxidation, and other processes that degrade chemicals in a smoke plume. Therefore, the experimental conditions are not representative of human exposures in the environment and, because combustion gases such as SO_2 are not likely to be of concern at a distance of approximately 1000 meters from the burn, this possible potentiating effect can be neglected.

Furthermore, the possible potentiating effects of other air pollutants may be offset by natural degradation of PAHs, especially as a result of photooxidation. In some experiments, exposure for about 40 minutes to light equivalent to one-fourth that of noon sunlight caused 35 to 65 percent loss of BaP in smoke samples (Tebbens et al. 1966 and Thomas et al. 1968, reported in NAS 1972). Other experiments provided comparable results. Half-lives of PAHs in air may therefore be on the order of hours. Some experiments show that the carcinogenic potency of a crude mixture is lower than expected when the potency of the known carcinogenic constituents is considered (Falk et al. 1964). It appears that the presence of a related but less potent compound can inhibit the activity of the more potent compound. Anticarcinogenic as well as co-carcinogenic mechanisms are both likely to occur. Therefore, the potentiating and degradation effects of other chemicals and environmental factors are likely to offset each other and are neglected in estimating potential risk.

5.0 TOXICITY FACTORS

The U.S. EPA has established toxicity factors that are used to provide a quantitative estimate of health risk from exposure to chemicals in the environment. Toxicity factors for carcinogenic effects are called slope factors (SFs) and those for non-cancer toxic effects are called reference doses (RfDs).

Toxicity factors are derived primarily from laboratory studies in animals. There is considerable uncertainty in extrapolating from animal studies to humans because of differences in the doses to which experimental animals and humans are exposed and because of the considerable variability in responses to chemicals among different species. Laboratory experiments are normally conducted in susceptible species; humans may be more resistant to the effects of the chemical than the animals used in many experiments. Therefore, SFs and RfDs are conservative (health-protective) measures of potential carcinogenicity or toxicity in humans.

SFs and RfDs for PAHs are shown in Table 4. Their derivation and application in risk assessment is described in more detail below.

TABLE 4

TOXICITY FACTORS FOR PAHs

Compound	Slope Factor (mg/kg-day)$^{-1}$		Reference Dose (mg/kg-day)		
			Oral		
	Oral	Inhalation	Subchronic	Chronic	Inhalation
Benzo(a)pyrene (1)	7.3E+0	(2)			
Pyrene			3E-1	3E-2	NA
Acenaphthene			6E-1	6E-2	NA
Anthracene			3E+0	3E-1	NA
Fluoranthene			4E-1	4E-2	NA
Fluorene			4E-1	4E-2	NA

Sources:U.S. EPA. 1993. Health Effects Assessment Summary Tables (HEAST) (subchronic RfDs).
U.S. EPA. Integrated Risk Information System (IRIS). On-line database.

(1) SF has been established only for benzo(a)pyrene. Relative carcinogenicity of other PAHs is shown in Table 5.
(2) Value of 6.1E+0 (mg/kg-day)$^{-1}$ was withdrawn in 1993. The oral slope factor is adopted for both oral and inhalation exposures.
NA Not available.

TABLE 5

RELATIVE POTENCY ESTIMATES FOR CARCINOGENIC PAHs

Compound	Relative Potency
Benzo(a)pyrene	1.0
Benzo(a)anthracene	0.145
Benzo(b)fluoranthene	0.140
Benzo(j)fluoranthene	0.061
Benzo(k)fluoranthene	0.066
Chrysene	0.0044
Indeno(1,2,3-cd)pyrene	0.232

Source: Clement Associates 1988.

5.1 Slope Factors for PAHs

The SF describes a dose-response relationship between the level of exposure to a carcinogen and the probability of getting cancer from the exposure. The SF has units of risk per mg chemical/kg body weight per day.

In establishing SFs for carcinogens, the U.S. EPA has taken a conservative (health protective) approach in assuming (1) that exposure even to very low doses of a carcinogen has the potential to increase the risk of cancer (i.e., it is assumed that there is no threshold dose below which a response, however small, does not occur) and (2) that responses to high doses given to susceptible laboratory animals can be extrapolated to responses to low doses in humans using a simple linear model. Furthermore, the SFs are upper 95th percentile confidence limits of a linear model based on dose response relationship determined in the laboratory. U.S. EPA states that carcinogenic risks estimated using this approach are upper-bound estimates and that actual risks are likely to be lower (U.S. EPA 1989a) and could be zero.

Using such an approach, U.S. EPA has developed an oral slope factor for benzo(a)pyrene of 7.3 $(mg/kg\text{-}day)^{-1}$. The inhalation slope factor of 6.1 $(mg/kg\text{-}day)^{-1}$ was withdrawn for further review in 1993. For purposes of this risk assessment, the oral slope factor is adopted to assess risk from both oral and inhalation exposures.

Table 5 shows the relative carcinogenic potency of other PAHs compared to BaP, which is assigned a relative potency of 1. Other PAHs have lower relative potency. In this risk assessment, the SF for BaP is used for all carcinogenic PAHs identified in the plume (Table 2). This is a conservative but not unreasonable approach that may overestimate risk but is not likely to underestimate potential risk because:

- BaP is among the most carcinogenic of the PAHs and therefore its SF overestimates the potency of most other PAHs (see Table 5).

- Adopting the BaP slope factor for other PAHs that are less carcinogenic compensates for any uncertainty related to possible increased carcinogenicity of some methylated PAHs and for possible synergistic effects resulting from the presence of other pollutants that may enhance carcinogenicity.

5.2 Reference Doses for PAHs

The RfD is a daily dose of a chemical that is considered safe for a lifetime of exposure. The RfD has units of mg chemical/kg body weight per day (mg/kg-day). RfDs are derived for subchronic exposures (defined by U.S. EPA as 2 weeks to 7 years) and for chronic exposures (7 years or more). Subchronic Rfds are used to evaluate exposure to smoke during an oil burn of several days, whereas chronic RfDs are used to evaluate longer-term exposures of many years to PM deposited

in soil. In establishing RfDs for toxic effects, U.S. EPA usually identifies the highest dose that did not cause an adverse effect in laboratory animals and reduces that dose by "uncertainty factors," ranging from 10 to 10,000, to provide a large margin of safety for human exposures. Therefore RfDs are conservative measures of the potential for adverse effects.

In this risk assessment, the toxicity PAHs with known non-cancer effects identified in the smoke plume is represented by the oral RfD for pyrene. The chronic RfD for pyrene is 3 x 10^{-2} mg/kg-day and the subchronic RfD is 3 x 10^{-1} mg/kg-day (U.S. EPA IRIS). The uncertainty factor used to derive the RfD for pyrene is 3,000. Using the RfD for pyrene to represent PAHs with known non-cancer effects is a conservative approach because the RfD for pyrene is lower than the RfDs for other noncarcinogenic PAHs (Table 4). The oral RfD is adopted to assess both oral and inhalation exposures since no inhalation RfDs for PAHs have been established.

6.0 EXPOSURE ASSUMPTIONS AND DOSE CALCULATIONS

The risk assessment is based on several assumptions regarding exposure point concentrations and exposure conditions. Conservative assumptions are used so as not to underestimate potential risk. The chief assumptions and calculation of dose are outlined below.

6.1 Exposure Concentrations

- Benzene and other volatile compounds present in crude oil are evaporated or combusted and are not significant constituents of the smoke at public exposure points (see Section 3.2).

- All PM in the plume is assumed to be respirable (< 10 microns). This is a conservative assumption that will overestimate the amount of particulate inhaled.

- PM concentration at the exposure point is 1 mg/m^3. This is a conservative value based on estimates from mesoscale burns (PM < 2 mg/m^3 at 500 m downwind; Walton et al. 1993) and is supported by results of screening-level air modeling. Results of the air modeling showed a maximum 1-hr PM concentration of 806 µg/m^3 and a maximum 24-hr concentration of 323 µg/m^3 at about 1500 meters downwind of the burn site. The concentration of 1 mg/m^3 used in this risk assessment probably overstates typical ground-level concentrations to which people would be exposed during a burn lasting several days by a factor of 3 (1/0.323 mg/m^3) or more, given that smoke from large-scale tests was nearly invisible from the ground at 800 m (one-half mile) (Raloff 1993), and that most people would be exposed to concentrations significantly lower than the maximums.

- For purposes of calculating PAH intake based on mg PAH per mg PM in air, PAHs measured in smoke, whether in vapor or particulate phase, are assumed to be associated with PM.

- Maximum concentrations in smoke of carcinogenic PAHs (1460 μg PAHc/g PM or 0.0015 mg/mg) and of noncarcinogenic PAHs (2656 μg/g PM or 0.003 mg/mg) are based on laboratory experiments using Alberta Sweet Crude, a medium crude oil (see Table 2). These data are considered representative of smoke emitted from burning most crude oils shipped by tanker. These PAH/PM ratios and the resulting estimates of air concentrations of PAHs are highly conservative estimates of exposure concentrations. They are comparable to concentrations measured at less than 100 m downwind of mesoscale burns reported in Fingas et al. (1993). For example, using the maximum total concentration of PAHs in soot from Table 2 of 9,000 μg/g, a PM concentration at the exposure point of 1 mg/m^3, and a conversion factor of 1 g/1000 mg yields a total PAH concentration of 9 $\mu g/m^3$ at 500 m. In the 1991 mesoscale burns, ground-level concentrations of total PAH ranged from 10.5 $\mu g/m^3$ at 30 m to 3 $\mu g/m^3$ at 60 m (Fingas 1993). Dilution and dispersion would reduce these concentrations significantly at greater distances. A second round of mesoscale tests performed in 1992 measured even lower PAH concentrations than reported for the 1991 tests. Therefore, the concentrations of PAHs used in the risk calculations are considered worst-case and are not likely to be exceeded. Resulting risk estimates are also considered worst-case.

- Long-term PM concentrations in soil are estimated by assuming that particulate deposition results in an evenly distributed film of soot 0.5 mm thick and that over time the soot is either dispersed by wind erosion or mixes in the top 10 cm of soil, resulting in a dilution factor of 0.005. Therefore long-term PM concentration in soil is expressed as 0.005 mg PM/mg soil. Assuming an even distribution of deposited PM of 0.5 mm is extremely conservative. In large-scale tests, little fallout was apparent at 800 m (one-half mile) (Raloff 1993).

6.2 Exposure Assumptions

- Exposure duration (ED) for inhalation: The burn lasts 5 days, and plume concentrations remain constant for 5 days. Therefore, the exposure duration for inhalation is 5 days. This is a conservative assumption that probably overestimates the duration of a burn by at least a factor of 2.

- Exposure duration (ED) for soot and soil ingestion: Risks from ingestion of deposited particulate matter are calculated for three scenarios and summed: child ingestion of soot for 5 days, child ingestion of contaminated soil for 6 years, and adult ingestion of contaminated soil for 24 years. Total

exposure duration to contaminated soil is therefore 30 years (U.S. EPA 1989a; 1991a).

- Inhalation rate (IR): The inhalation rate used to calculate chemical dose from inhalation exposure is 20 m^3/day (U.S. EPA 1989a; 1991a).

- Ingestion rate (IR): A soil ingestion rate of 200 mg/day is used for children ages 1 < 6 (U.S. EPA 1989a) and a rate of 10 mg/day is used for older children and adults (U.S. EPA 1989b).

- Soil matrix effect (ME): A soil matrix effect of 0.5 (50 percent inhibition) is applied to represent the inhibition of uptake in the gastrointestinal tract resulting from contaminant adsorption to a solid matrix such as soil. PAHs and other organic compounds such as pesticides and polychlorinated biphenyls (PCBs) bind tightly to particulate matter (Calderbank 1989). The matrix effect is conservatively estimated at 50 percent and could be 10 percent (i.e., a 90 percent reduction in uptake) (McConnell et al. 1984; Shu et al. 1988; Goon et al. 1991).

- Averaging time (AT): For carcinogens, the averaging time used to calculate average daily dose over a lifetime is 25,550 days (70 years). For noncarcinogens, the averaging time is the exposure duration (10,950 days) (5 days for soot inhalation and ingestion, 6 years (2190 days) for childhood soil ingestion and 24 years (8.760 days) for adult soil ingestion) (U.S. EPA 1989a; 1991a).

- Body weight (BW): Average adult body weight is 70 kg; average body weight of children ages 0 - 6 is 15 kg (U.S. EPA 1989a; 1991a).

6.3 Dose Calculations

Dose is the daily chemical intake expressed as mg chemical per kilogram body weight per day (mg/kg-day). Doses are calculated separately for inhalation and childhood and adult soil ingestion using the following equation:

$$\text{Dose} = \frac{\text{PM} \times \text{PAH/PM} \times \text{ME} \times \text{IR} \times \text{ED}}{\text{AT} \times \text{BW}}$$

where:

PM is the estimated particulate matter concentration in air (1 mg/m^3) or soil (0.005 mg/mg).

PAH/PM is the ratio of PAHs to PM (mg/mg). For carcinogenic PAHs the ratio is estimated to be 0.0015 mg/mg, and for noncarcinogenic PAHs, the ratio is 0.003 mg/mg (see Section 6.1 and Table 2).

and ME, IR, ED, AT, and BW are as defined in the Section 6.2.

Tables 6 and 7 show the calculation of chemical dose for carcinogens and noncarcinogens using these concentrations and exposure factors.

7.0 RISK ESTIMATES

This section provides conservative estimates of potential carcinogenic risk and noncarcinogenic hazard from exposure to PAHs in smoke emanating from an oil burn via the inhalation and incidental ingestion exposure routes.

7.1 Carcinogenic Risk

Carcinogenic risks are estimated as the incremental probability (additional risk above the normal rate of getting cancer) of an individual developing cancer over a lifetime as a result of exposure to a carcinogen. For example, an excess cancer risk of 1×10^{-6} (1 in 1 million) means that there is a 1 in 1 million chance of getting cancer from the exposure, and that an individual's overall risk of getting cancer has been increased by 0.000001. This is a very low risk level that could not be measured or detected in individuals or even in large populations.

Cancer risk estimates are usually interpreted within policy guidelines that establish acceptable risk levels. U.S. EPA policy, expressed in the National Oil and Hazardous Substances Pollution Contingency Plan (NCP) (40 CFR Part 300) and other guidance documents, states that "For known or suspected carcinogens, acceptable exposure levels are generally concentration levels that represent an excess upper bound lifetime cancer risk to an individual of between 1×10^{-4} and 1×10^{-6}." Measures to restrict chemical release or exposure are not usually considered warranted unless cancer risk exceeds 1×10^{-4} (1 in 10,000) (U.S. EPA 1991b).

The estimate of cancer risk is calculated by multiplying the daily chemical dose averaged over a 70-year lifetime by the cancer slope factor:

Cancer risk = Dose x SF

where: Dose is the chemical intake (mg/kg-day).
SF is the chemical-specific slope factor (1/mg-kg-day).

Table 6 shows the estimated cancer risk calculated by multiplying inhalation and ingestion doses by the cancer slope factor for BaP (representing all carcinogenic PAHs in the plume). Risks from inhaling carcinogenic PAHs in the smoke plume and from ingesting carcinogenic PAHs deposited in the soil are summed to yield a total excess cancer risk of 5×10^{-5} (5 in 100,000). The largest contributors to the total risk estimate is childhood ingestion of pure soot and contaminated soil (see Table 6). The excess cancer risk estimate is a worst-case estimate that probably overestimates actual risk because it assumes no degradation of PAHs in air or in soil over a 30-year exposure duration (whereas the half-life of PAHs in air may be on the order of hours; see Section 4.3).

TABLE 6
ESTIMATED CANCER RISK FROM INHALATION AND INGESTION OF CARCINOGENIC PAHs IN SMOKE

Cancer Risk = SF x Dose
Dose = (PM x PAHc/PM x IR x ME x ED) / (AT x BW)

where: SF = Slope factor for benzo(a)pyrene (1/mg/kg-day).
Dose = mg chemical/kg BW-day.
PM = mg/cu. m air or mg/mg soil.
PAHc/PM = Maximum ratio of carcinogenic PAHs to PM (mg/mg); see Table 2.
Other parameters as described in text.

Exposure Route	PM mg/m3 or mg/mg	PAHc/PM mg/mg	IR m3/day or mg/day	ME unitless	ED days	AT days	BW kg	Dose mg/kg-day	SF	Risk
Inhalation (5 days)										
	1	0.0015	20	1	5	25,550	70	8.4E-08	7.3	6.1E-07
Child Soot Ingestion (5 days)										
	1	0.0015	200	0.5	5	25,550	15	2.0E-06	7.3	1.4E-05
Child Soil Ingestion (6 yr)										
	0.005	0.0015	200	0.5	2,190	25,550	15	4.3E-06	7.3	3.1E-05
Adult Soil Ingestion (24 yr)										
	0.005	0.0015	10	0.5	8,760	25,550	70	1.8E-07	7.3	1.3E-06
Total Cancer Risk										4.8E-05

TABLE 7
ESTIMATED NONCARCINOGENIC HAZARD FROM INHALATION AND INGESTION OF PAHs IN SMOKE

Hazard Quotient = Dose / RfD
Dose = (PM x PAHnc/PM x IR x ME x ED) / (AT x BW)

where: Dose = mg chemical/kg BW-day.
RfD= Reference Dose for pyrene (mg/kg-day).
PM = mg/cu. m air or mg/mg soil.
PAHnc/PM = Maximum ratio of noncarcinogenic PAHs to PM (mg/mg); see Table 2.
Other parameters as explained in text.

Exposure Route	PM mg/m3 or mg/mg	PAHnc/PM mg/mg	IR m3/day or mg/day	ME unitless	ED days	AT days	BW kg	Dose mg/kg-day	RfD mg/kg-day	Hazard Quotient
Inhalation (5 days)	1	0.003	20	1	5	5	70	8.6E-04	3.0E-01	2.9E-03
Child Soot Ingestion (5 days)	1	0.003	200	0.5	5	5	15	2.0E-02	3.0E-01	6.7E-02
Child Soil Ingestion (6 yr)	0.005	0.003	200	0.5	2,190	2,190	15	1.0E-04	3.0E-01	3.3E-04
Adult Soil Ingestion (24 yr)	0.005	0.003	10	0.5	8,760	8,760	70	1.1E-06	3.0E-01	3.6E-06
Cumulative Hazard Index										7.0E-02

The excess cancer risk of 5 in 100,000 is within U.S. EPA guidelines for acceptable risk levels. In the United States, the overall chance of getting cancer is 1 in 3 (American Cancer Society 1990). An excess cancer risk of 5 in 100,000 would increase the overall rate from 1 in 3 to 1.00005 in 3. This is a very small increase that would not be observable in individuals or in most populations.

7.2 Noncarcinogenic Hazard Index

The potential for adverse noncarcinogenic effects resulting from exposure to a chemical is evaluated by comparing an exposure level or dose with the reference dose (RfD). The resulting ratio is called a Hazard Quotient. If the Hazard Quotient is 1 or below, there is no cause for concern for noncancer effects. In general, the greater the value of the Hazard Quotient above 1, the greater the level of concern. However, since the HQ does not define dose-response relationships, its numerical value should not be construed as a direct estimate of risk.

To assess the overall potential for noncarcinogenic effects posed by exposure to multiple pathways, Hazard Quotients are summed. The resulting sum is referred to as the Hazard Index (HI). The HI approach assumes that exposures to several chemicals at doses that are not hazardous in themselves could cumulatively result in an adverse health effect. For the purposes of this report, pyrene is used as a worst case surrogate to represent the non-cancer effects of PAHs in the smoke plume. Additivity of effects is implicit in this approach.

The Hazard Quotient for exposure to a chemical is calculated using the following equations:

$$HQ = \text{Dose} / \text{RfD}$$

where: Dose is the chemical intake (mg/kg-day)
RfD is the chemical-specific reference dose (mg/kg-day)

The calculation of a Hazard Index for noncarcinogenic effects is shown in Table 7. The overall Hazard Index is 7.3×10^{-2} (0.072). Since the value is below 1, there is no cause for concern for adverse noncarcinogenic effects, even under the conservative exposure assumptions used in the analysis.

8.0 CONCLUSIONS

The risk assessment shows that, even under highly conservative exposure assumptions, the estimated cancer risk level and noncarcinogenic hazard index associated with exposure to PAHs in smoke from burning an oil spill are below levels of concern established by U.S. EPA for protection of public health. Therefore, adverse health effects from toxic and carcinogenic constituents of smoke may not be a significant factor in making a burn/no burn decision.

However, it is apparent from observations and from air modeling that somewhat elevated concentrations of PM (i.e., concentrations that exceed U.S. and coastal state standards) can occur within about 2000 m of the burn location, depending on site-specific conditions. Exposure to elevated PM concentrations can have acute respiratory effects and exacerbate existing ailments. Therefore, additional response measures may need to be taken to prevent such temporary exposures if a burn is conducted within approximately 1000 to 2000 m of a population center. Temporary and transient exceedances of ambient air quality standards could be considered acceptable if burning is conducted for the overall protection of the environment.

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