Pure Appl. Chem., Vol. 71, No. 1, pp. 113–133, 1999.
Printed in Great Britain.

Occupational-health aspects of marine oil-spill response*

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INTRODUCTION

This paper addresses chemical aspects of occupational health and marine oil-spill response and is restricted to exposures to crude oil in its various forms. Thus *in-situ* burning of oil is included, but ancillary chemicals such as surfactants or bioremediation agents are not. The content of this paper is largely based on the literature published after 1985, the date of a comprehensive review conducted by Politzer *et al.* [1] for the American Petroleum Institute, and on a review carried out for the Marine Spill Response Corporation early in 1993 [2].

Concern about health and safety is a normal part of every oil spill. In general, safety is easier to understand and address than are concerns about exposure to crude oil and other chemicals which might be used in the response. At one level, human exposure can be addressed through the enforcement of very conservative requirements for the use of personal protective equipment (PPE). In the real world, however, conditions at a spill site make the use of such equipment inconvenient or even hazardous, and so the goal becomes to balance the risk from exposure with the appropriate level of PPE.

While oil-spill cleanup is a comparatively new aspect of occupational-health practice, and dates from the formalization of response measures by companies and national and international agencies (something that occurred over the last 30 years), exposure to crude oil itself is a 'mature' occupational-health matter. Workers have been exposed, both by inhalation and dermally, to the effects of crude oil for the past century. The exposure of response workers during the early phases of the oil-spill response can be likened to that experienced by oil-well-drilling crews and, to a lesser extent, by oil-well-maintenance personnel or fighters of oil-well fires. In contrast, exposures in the later stages of the cleanup are less clearly related to occupations within the oil industry. The crude oil will have been altered by weathering, and exposure to cleanup chemicals (e.g. dispersants, bioremediation agents) will become relatively more prominent. Such substances are beyond the scope of this paper, and in any event, few data are available on the compositions or mammalian toxicity of dispersants. Although there are frequent references to toxicity in connection with dispersants, these invariably seem to refer to ecotoxicity. Human hazard does not appear to be an issue. For example, in a recently published paper entitled, 'Effectiveness and safety of biosurfactants as agents of oil spill response' [3], 'safety' refers to possible toxicity to crustaceans and fish.

EXPOSURE TO CRUDE OIL

Oil-spill cleanup workers may be exposed to crude oil through inhalation and dermal contact. Personal protective equipment can, of course, reduce these exposures, but care must be taken to ensure that the use of such equipment does not create hazards additional to the ones the equipment was originally intended to protect against. One can, for example, easily imagine situations where the wearing of excessive amounts of impermeable clothing may generate a hazard from heat stress. Similarly, prolonged wearing of respirators, particularly by workers involved in strenuous activity, can induce physiological stress (see, for example, Seliga *et al.* [4]) or simply fatigue, and thus increase the potential for accidents.

Another consideration is the applicability of occupational exposure limits to the situation faced by oil-spill workers. The uncritical application of such exposure limits may be open to question because the

Pure Appl. Chem. **71**(1) (1999). An issue of special reports reviewing oil spill countermeasures.

limits are based on a worker being exposed five days per week, 50 weeks per year for a 40-year working life. Oil-spill workers, unlike their counterparts in the petroleum industry (or indeed in industry in general), are likely to have a considerably more intermittent exposure, unless they are employed elsewhere in the oil industry between oil spills.

Exposure and the nature of crude oil

All other aspects being equal, the hazard to the oil-spill worker will depend on the chemical and physical nature of the crude oil. To this end, various attempts have been made to classify crude oils. One such approach, adopted by CONCAWE [5], used physical parameters to group crude oils on the basis of expected 'spreading behavior' in a spill (see Table 1). The first criterion was a classification of the oils by their pour points. The CONCAWE authors argued that crude oils with pour points above 5–10 °C will 'probably solidify very quickly under conditions with a sea water temperature of about 10 °C'—and this identified the first group. The other four groups were classified by the fractions expected to evaporate within 24 h from a hypothetical spill of 10 000 m^3 (0–20%, 20–40%, 40–50%, and over 50%). Although the prime motive for using such a classification was to aid cleanup, evaporative loss criteria also have direct bearing on the inhalation exposure of oil-spill workers. Chemical composition is less usefully categorized—and represents, in any event, a much more complex task. Traditionally, the chemical classification of crude oils is made with the petroleum refiner in mind—the 'generic' descriptors, paraffins, aromatics, naphthenes, asphaltenes and maltenes [6] and even such 'elemental' descriptors as sulfur content, do not provide sufficiently detailed information for exposure assessment. (An idea of the type of information generally available may be found in Table 2.) The relationship between physical properties and the composition of crude oil is so complex that prediction of toxic properties from physical properties alone is clearly impracticable.

Table 1 Classification of 58 crude oils by physical properties related to 'expected spreading behavior' (derived from CONCAWE [5])

Group (No. of crude oils represented)	Pour point (°C)	Volume distilled at 100 °C	Density (kg/L)	Viscosity at 40 °C (cSt)
I (16)	13 ± 7	11 ± 4%	0.847 ± 0.021	13 ± 22
II (3)	− 15 ± 16	1.5 ± 0.6%	0.958 ± 0.029	1400 ± 2000
III (6)	− 34 ± 3	7.5 ± 3.5%	0.904 ± 0.015	27 ± 23
IV (16)	− 15 ± 10	11 ± 3%	0.859 ± 0.013	6.7 ± 2.0
V (17)	− 15 ± 13	15 ± 4%	0.829 ± 0.015	3.4 ± 0.8

Further, the composition of oil changes as a result of weathering during an oil-spill cleanup. Volatile components are usually rapidly lost and inhalation exposure becomes negligible, leaving essentially dermal contact as the only exposure route. (This is a generalization, for, as discussed in subsequent sections, it appears that oil-contaminated mists and aerosols may be formed in all stages of the cleanup operation.) The constituents and composition of the remaining oil (essentially the nonvolatile components) continue to change as weathering proceeds. In the *Exxon Valdez* spill, for example, the concentration of polycyclic aromatic hydrocarbons (PAHs) in samples of the crude oil remaining in the ship's hold were orders of magnitude higher than the concentrations found in weathered crude oil samples collected off shore 30–90 days after the spill [ref. 7, table 1]. In commenting on the same spill, Griest *et al.* [8] observed that the concentration of the neutral polar fraction in the weathered crude oil was approximately double that of the PAH fraction and concluded that this was a result of PAHs reacting with oxygen to form oxygen-containing polar compounds.

Routes and magnitudes of exposure

The dermal and inhalation routes of exposure tend to produce exposures to different components of crude oil. This distinction between the two is, however, not clear-cut. Depending on how fresh the oil is, oil-spill

Table 2 Chemical composition and physical properties of two crude oils (derived from [91])

Bulk property & fraction	Heavy arabian crude	Light arabian crude
Pour point	−34 °C	−34 °C
Density	0.886 kg/L	0.831 kg/L
Viscosity at 38 °C	18.9 cSt	3.78 cSt
Sulfur content	2.84%	1.10%
Light naphtha (boiling range: 20 to 100 °C)	yield: 7.9% (paraffins 89.6%; naphthenes 9.5%; aromatics 0.9%)	yield: 10.5% (paraffins 87.4%; naphthenes 10.7%; aromatics 1.9%)
Heavy naphtha (boiling range: 100 to 150 °C)	yield: 6.8% (paraffins 70.3%; naphthenes 21.4%; aromatics 8.3%)	yield: 9.4% (paraffins 66.3%; naphthenes 20.0%; aromatics 13.7%)
Kerosene (boiling range: 150 to 235 °C)	yield: 12.5% (paraffins 58.0%; naphthenes 23.7%; aromatics 18.3%)	yield: 18.4% (paraffins 58.9%; naphthenes 20.5%; aromatics 20.6%)
Light gas oil (boiling range: 235 to 343 °C)	yield: 16.4% (class breakdown not given)	yield: 21.1% (class breakdown not given)
Heavy gas oil (boiling range: 343 to 565 °C)	yield: 26.3% (class breakdown not given)	yield: 30.6% (class breakdown not given)
Residual oil (boiling range: above 565 °C)	yield: 26.8% (class breakdown not given)	yield: 7.4% (class breakdown not given)

workers may be dermally exposed to both volatile and nonvolatile components, some of which are capable of being absorbed through the skin. In fact for volatile substances, although inhalation may be the dominant exposure route, dermal exposure can make an appreciable contribution to overall exposure during a cleanup. Nevertheless, many of the components of crude oil are so involatile that dermal exposure is likely to be the only route of exposure (unless inhalation of oil-containing mists and aerosols is found to be a factor). For the nonvolatiles, quantitative estimates of exposure are difficult to obtain and one has to look for qualitative or indirect evidence of dermal absorption—biochemical markers may play a role here. Boogaard & van Sittert [9] in studies of petrochemical workers exposed to PAHs concluded, through the measurement of urinary 1-hydroxypyrene (a PAH metabolite), that dermal exposure (in the absence of personal protective equipment) made a significant contribution to total uptake of PAHs.

The obvious source of inhalation exposure is volatile chemicals, of which the main classes are alkanes, aromatics and sulfur compounds. The first group consists of straight-chain, branched-chain and cyclic hydrocarbons, containing up to about 10 carbon atoms. (This arbitrary cutoff is based on the fact that the *Handbook of Chemistry and Physics* [10] lists few alkanes with 10 or more carbon atoms and vapor pressures greater than 1 mmHg at ambient temperatures.) The aromatics are dominated by the lower molecular weight homologs of benzene: benzene itself, toluene, the xylenes and the trimethylbenzenes. The sulfur compounds are chemically more diverse, encompassing both the inorganic hydrogen sulfide (ubiquitous in sour crudes) and such organic compounds as methyl and ethyl mercaptans and the corresponding thioethers, and the unsaturated cyclic compound thiophene. They are major contributors to the odor of crude oil.

There are few data on the composition of vapors over oil spills. Bowes [11] measured atmospheric hydrocarbon levels near an experimental burn of some 40 tons of artificially weathered crude oil and recorded time-weighted-average levels of individual hydrocarbons of fractions of a p.p.m. or less. Hanna & Drivas [12] modeled air concentrations of volatile organic compounds in the early hours of the *Exxon Valdez* spill. Their model predicted that essentially all benzene, toluene and *n*-hexane evaporated from the spill within about 12 h, and that benzene, for example, reached a maximum airborne concentration of about 5 p.p.m. during the first hour of the spill; its modeled concentration fell to about 1 p.p.m. after 6 h

and to less than 0.02 p.p.m. after 12 h. On the basis of this model, less volatile compounds such as dodecane were appreciably more persistent than benzene but generally did not reach airborne concentrations much above 0.1 p.p.m. Recent modeling calculations by Zhou & Wong [13] similarly suggested that *n*-hexane and toluene would be completely lost from a spill within a few hours, and *n*-pentane within one hour. These authors also cited measurements of airborne hydrocarbon levels during the *Braer* spill. On 11 January 1993, when the vessel broke up, airborne levels of C_3–C_8 hydrocarbons reached over 6 p.p.m. at a nearby site, but the following day, the levels at the same site had fallen to below 0.1 p.p.m. At two other sites within a few kilometers of the wreck, the hydrocarbon levels also dropped by more than an order of magnitude over the same period [14].

It also seems possible that oil-containing mists and aerosols can be generated by wave action and/or spill cleanup activities at sea and, onshore, by wave action and the use of high-pressure water hoses. Unlike the other three classes of inhalants, mists and aerosols could be a source of inhalation and dermal exposure to both fresh and weathered crudes, and they appear to be a possible source of exposure to the diverse mixture of nonvolatile compounds in crude oil.

In addition to producing dissimilar exposures to the various components of crude oil, the dermal and inhalation exposure routes are likely to be important in different circumstances and at different stages of a typical spill response. In general, inhalation exposure to volatile components is unlikely to be important after the first few hours of a spill—and in fact in fact might be negligible by the time a major cleanup was under way. Dermal exposure, on the other hand, remains a possibility throughout a cleanup.

An exception to the above generalization about inhalation exposure would be a spill where the oil being cleaned up is continually renewed—the *Ixtoc* blowout, which took months to contain [ref. 15, p. 567], is a notable example—as would a situation where oil that had been 'protected' in some way (e.g. by 'skinning') is disturbed, thus yielding a fresh surface. Another exception would be exposure to crude-oil-containing aerosols. The formation of aerosols in large quantities at sea requires sea state and weather conditions that would probably restrict cleanup activities. Shoreline cleanup workers might remain exposed in such conditions; and the use of high-pressure hoses in cleanup appears to make oil-aerosol formation a possibility even with calm winds and seas.

Special cases

Although a discussion of cleanup procedures in general is beyond the scope of this paper, two aspects of oilspill cleanup have implications for the exposure of workers to crude-oil components and merit discussion here. The first, alluded to above, is the question of aerosol formation and the second, in light of its increasing prominence as a spill-cleanup technology, is *in situ* burning.

Aerosol formation

Reports in *The Times* (of London) of the wreck of the *Braer* lend credence to the idea of mist and aerosol formation during an oil spill [16,17], as does the study by Campbell *et al.* [18]. It was also reported that, after the *Amoco Cadiz* oil spill, exposure to the workers was increased by mists and aerosols resulting from the use of high pressure water and steam during the cleanup [ref. 15 p. 476, ref. 1, p. 52]. And two reports describe exposure of *Exxon Valdez* cleanup workers to oil mists: personal exposure monitoring by the National Institute of Occupational Safety and Health (NIOSH) of beach-cleanup and waste-handling workers [7], and personal and area sampling conducted by Med-Tox Associates on behalf of Exxon [19]. The exposure monitoring conducted by NIOSH failed to detect any oil mist exposure (33 samples; detection limit, 0.4 mg/m^3). However, the more extensive monitoring by Med-Tox (114 samples; detection limit not identified) recorded exposures from 0.01 to 20 mg/m^3, with a geometric mean of 0.6 mg/m^3. Politzer *et al.* [1] considered that aerosol formation was part of the transport and fate process for petroleum following an oil spill (see Fig. 6 of that reference).

While the composition of such aerosols is very difficult to assess, it seems very unlikely that they would contain significant amounts of volatile components such as benzene; the very large relative surface area of aerosol droplets would enable such substances to evaporate almost instantaneously. The Med-Tox data on the *Exxon Valdez* spill [19] suggest that the concentration of aerosols might be roughly 1 mg/m^3. (To put this estimate in rough perspective, most national jurisdictions that regulate occupational exposure

to mineral oil mists have set a TWA of 5 mg/m^3 for them [20].) The aerosols might, however, contain up to about one per cent PAHs, [ref. 21, appendix A.1]. These figures suggest a possible airborne PAH concentration of about 10 μg/m^3 in the aerosols. If one assumes, as a worst-case scenario, that the whole of this 10 μg/m^3 consists of carcinogenic compounds in respirable form, then a daily 8-h exposure to such aerosols would, assuming 10 m^3 inhalation volume for light to moderate work, lead to an intake of about 100 μg carcinogen per day. Such an exposure would not be insignificant, particularly if repeated with any frequency over a working life. Further investigation would be needed to determine how far this worst-case scenario is from reality.

In-situ *burning*

In-situ burning of a crude oil spill generally modifies chemical hazards of cleanup by oxidizing some of the hazardous components of the oil, altering the evaporation of volatile components and releasing combustion products. These phenomena have been investigated in two large burns (48 and 30 m^3 of crude oil) carried out in the ocean in 1993 off Newfoundland—the Newfoundland Offshore Burn Experiment (NOBE) [see, for example, refs 22,23]. (The results of these experiments seem consistent with data from a series of smaller burns—each using between about 1 and 14 m^3 of crude oil in a specially built steel pan—carried out in 1991 and 1992 at Mobile, Alabama [24].) The main observations can be summarized as follows:

- Dense gases and vapors spread out horizontally from the spill but remained within a few meters of the surface. (It appears that such gases and vapors form a doughnut-shaped 'plume' centered on the fire; the shape of such a plume can be modified by winds, but its location and composition remain distinct from those of the main smoke plume, which initially rises steeply.)
- Levels of hydrocarbon vapors in these surface plumes were lower than levels from the nonburning spill by as much as an order of magnitude [ref. 22, tables 3, 17].
- Carbonyl compounds, such as aldehydes, which might be formed by incomplete combustion, were also found at levels lower than those associated with a nonburning spill [23].
- Mean carbon dioxide levels measured on boats 50 and 100 meters downwind of the burn were ≈ 500–600 p.p.m. greater than ambient levels, but 500 m downwind of the burn they fell to less than 100 p.p.m. above ambient. [ref. 22, tables A12, A13]
- Other gaseous combustion products (carbon monoxide and the oxides of sulfur and nitrogen) were at or near background levels [ref. 22, table 3]. Sulfur dioxide was, however, detected as an acid aerosol at levels of ≈ 10 p.p.m. at the same two downwind sampling boats close to the burn [ref. 22, tables A8, A9].

Fingas *et al.* [25] summarized the NOBE results on PAHs by saying that these compounds 'were found to be lower in the soot than in the starting oil and were consumed by the fire to a large degree.' Mean PAH levels in air and airborne particulates, measured at the two downwind sampling boats within 100 m of the burn, reached about 12 μg/m^3 during the burns (compared to levels of about 7 μg/m^3 measured before the day of the first burn and a 'trip blank' of 3.7 μg/m^3 presumably measured on the day of the burn, but at a more remote site). Once again, 500 m downwind, levels were essentially ambient. Levels of ≈ 40–130 μg/m^3 were found at higher altitudes, in or near the smoke plume. During the burns, mean respirable particulate levels (PM_{10}) measured at the two nearby downwind boats were in the range 9–14 mg/m^3, in comparison to a 'background' of 0.04 mg/m^3 (time and location unclear). Levels 500 m downwind were less than 0.15 mg/m^3. (These levels might be compared with the occupational exposure limits for carbon black set by several US and other national bodies, namely eight-hour time-weighted averages of the order of 3 mg/m^3 [20].) It may be noteworthy, however, that after the first of the two burns, perhaps as a result of the loss of the updraft from the fire, the 'background' particulate level rose to about 13 mg/m^3 [ref. 25, table 5].

In general, it appears that workers more than a few hundred meters from an *in-situ* burn are unlikely to be significantly exposed to either volatile components of crude oil or substances in particulate matter, provided that the workers do not come into contact with the smoke plume.

HEALTH EFFECTS OF INHALATION EXPOSURE

The volatile components of crude oil—those most likely to be inhaled—comprise, as discussed above,

the lower-molecular-weight hydrocarbons (containing up to about 10 carbon atoms) and sulfur compounds (predominantly mercaptans and the gas, hydrogen sulfide). There is little specific information related to inhalation effects arising from exposure to crude oil as a single entity. Most of the discussion in this section therefore deals with the exposure effects of individual petroleum-derived compounds or classes of compounds.

Acute effects

By and large, the acute effects of substances that readily evaporate from spilled oil are well characterized. At low airborne concentrations, the effects are essentially those of discomfort and irritation. As concentrations increase, there are progressively more severe effects to the central nervous system (albeit with different results—the narcotizing effects of the organics and the lethal effects of high concentrations of hydrogen sulfide) [see for example, refs 26,27].

Aromatic and aliphatic components

The overt signs of acute intoxication in humans—dizziness, incoordination, nausea, headache, fatigue—are markedly similar for both the aromatic and aliphatic hydrocarbons [27–29]. Such nonspecific symptoms start to be observed at exposure concentrations above 100–200 p.p.m. and are typical of central nervous system involvement. In addition, moderately high concentrations are mildly irritating to the eyes and mucous membranes [26].

Uptake, distribution and elimination: Although overt symptoms of acute exposure are similar, there are differences between aromatic, and saturated and unsaturated aliphatic hydrocarbons in uptake and tissue distribution measured in experimental animals. And these differences, in turn, may point to differences in toxic response. In an elegant series of experiments involving rats exposed to single hydrocarbons (C_6 to C_{10} *n*-alkanes, naphthenes and benzenes; and C_8 to C_{10} *iso*-alkanes and 1-alkenes; all at 100 p.p.m. in air) 12 h/day for three days, Zahlsen *et al.* [30,31] demonstrated that comparable groups of hydrocarbons possess distinctly different toxicokinetic properties when inhaled—their findings are summarized in Table 3. In subsequent inhalation experiments (single exposures of 12-h duration) involving *n*-nonane, trimethylcyclohexane and trimethylbenzene, in mixtures (containing either all three compounds or pairs of the compounds in equimolar proportions) and as single compounds (at total concentrations of 75, 150, 300 and 450 p.p.m. in air), Eide & Zahlsen [32] reported that the presence of multiple hydrocarbons in the exposure mixture did not affect the uptake of the individual compounds—a finding that suggests that uptake data from single compounds can be combined directly in predicting the uptake from mixtures of vapors. In addition, Eide & Zahlsen claimed that the 'quadratic' form of the tissue-level-response with respect to airborne exposure levels observed for the aromatic (and to a lesser extent the naphthene) was evidence that metabolism influenced the uptake; no such 'quadratic' dose–response behavior was observed for the *n*-alkane, demonstrating that, at least at the airborne concentrations studied, metabolism did not affect uptake of the compound. In all these experiments, elimination of the hydrocarbons from the tissues occurred rapidly once exposure had ceased—clearance times appeared to be of the order of a few hours.

Clearance of inhaled hydrocarbons in humans is also rapid. For example: Mutti *et al.* [33] reported a biphasic clearance of *n*-hexane from the lungs with half-times of 11 and 99 min; Nise *et al.* [34] fitted a three-phase clearance model for the removal of toluene from the blood of 10 printers exposed to toluene-containing solvents and obtained half-times (mean ± SD) of 11 ± 11 min, 3 ± 3 h and 118 ± 86 h for each phase; and Järnberg *et al.* [35] fitted a four-phase clearance model to the uptake and removal of trimethylbenzenes (all three isomers) from the blood of 10 male volunteers who inhaled airborne concentrations of 25 p.p.m. for 2-h periods while undertaking light physical activity (a work load of 50 W on an ergonometer bicycle)—clearance half times of about 1–2 min, 21–27 min, 4–5 h and 78–120 h, for each of the four phases were obtained. Somewhat in contrast to the findings of Zahlsen and co-workers, both Nise *et al.* and Järnberg *et al.* interpreted their data as demonstrating the potential for the aromatic compounds to accumulate in adipose tissue. However, the data from both Nise *et al.* [34] and Järnberg *et al.* [35] do, like the animal experiments of Zahlsen and co-workers, indicate high respiratory uptake and moderately rapid metabolism of the aromatic compounds.

Table 3 Summary of the toxicokinetic results [30,31], in rats, of exposure (at 100 p.p.m.) to C_6–C_{10} hydrocarbons

	Alkanes	Isoalkanes	Alkenes	Naphthenes	Aromatics
C_6	*n*-hexane	*n*/s	*n*/s	cyclohexane	benzene
C_7	*n*-heptane	*n*/s	*n*/s	methylcyclohexane	toluene
C_8	*n*-octane	2-methyl heptane	1-octene	1,2-dimethylcyclohexane	*o*-xylene
C_9	*n*-nonane	2-methyl octane	1-nonene	1,2,4-trimethylcyclohexane	1,2,4-trimethylbenzene
C_{10}	*n*-decane	2-methyl nonane	1-decene	*t*-butylcyclohexane	*t*-butylbenzene

Results:
(a) Aromatic hydrocarbons show high solubility and concentrations in blood, but low concentrations in brain, liver and kidney.
(b) Naphthenic hydrocarbons show low concentrations in blood and high concentrations in brain.
(c) The 1-alkenes show high solubility and concentrations in blood, high concentrations in brain and a tendency for accumulation in fat with repeated exposures.
(d) The alkanes (both *n*- and *iso*-) show low concentrations in blood, high concentrations in brain and a high potential for accumulation in fat with repeated exposures.
(e) Biological concentrations of hydrocarbons within one class increase in general with increasing molecular weight (though there are specific exceptions).
(f) Accumulation is obviously influenced by differences in metabolism and enzyme induction potential.
(g) Lipid solubility is *not* (our emphasis) the only parameter relevant for the evaluation of hydrocarbon accumulation.
n/s: not studied.

Exposure effects: Rats exposed for up to eight hours to high concentrations (close to the saturated vapor pressure) of *n*-nonane exhibited tremor, spasms, limb paralysis and death—an eight-hour LC_{50} of about 4500 p.p.m. was estimated from the study—but neither sedative effects nor narcosis were observed [36]. Animals that survived the exposures had severe damage to the cerebellar cortex and extensive loss of Putkinje cells (cerebellar neurons); those that died, did so from cardiopulmonary insufficiency. In this same study, rats were also exposed to saturated vapor pressures of *n*-decane (1370 p.p.m.), *n*-undecane (440 p.p.m.), *n*-dodecane (140 p.p.m.) or *n*-tridecane (40 p.p.m.) for eight hours, but neither deaths nor adverse behavioral effects were observed.

In a study of the exposure of rats (6 h per day, five days per week for three weeks) to 400 p.p.m. or 800 p.p.m. of vapors from Stoddard solvent, Lam *et al.* [37] found that the exposure induced increased levels of several different central nervous system transmitters (noradrenaline, dopamine, and 5-hydroxytryptamine). They suggested that these increases would cause changes in the activities of the corresponding neurons which, in turn, would lead to neurotoxic effects.

There is a considerable literature on the human neurobehavioral effects arising from inhalation of *high* concentrations of hydrocarbon vapors by solvent abusers. The acute symptoms from such abuse—dizziness, euphoria, delirium—clear up after each abuse episode with, invariably, no apparent long-term effects. [27] Attempts have also been made to judge whether exposure to occupational levels of hydrocarbon solvents (i.e. somewhat lower concentrations than in the animal experiments described above) would result in definable, acute effects on the central nervous system.

Van Vliet *et al.* [28], for example, investigated 379 Dutch commercial painters occupationally exposed to organic solvents. They used a questionnaire to collect information about the workers' occupational history, exposure to organic solvents, and the occurrence of such symptoms as nausea, dizziness, headache, shortness of breath and absent mindedness. The replies were compared with those from a nonexposed population of 443 construction workers who were administered the same questionnaire. The results of the study indicated that workers in the exposed group had a higher reporting rate of prenarcotic symptoms than workers not exposed to solvents. Van Vliet *et al.* concluded that the study confirmed the existence of acute neurotoxic effects among workers exposed to solvents but it did not provide strong evidence for the existence of chronic neurotoxic effects as reflected by the occurrence of neurasthenic symptoms. Olson *et al.* [38] investigated the effects of four-hour solvent exposures of volunteers to toluene (at 80 p.p.m.), *p*-xylene (70 p.p.m.) or a mixture of the two solvents (54 p.p.m. and 23 p.p.m.,

respectively) on simple reaction time, short-term memory, and choice reaction time. The results indicated that performance on the tests was unaffected by exposure at these levels. Similarly, Järnberg *et al.* [35] in their study of the uptake of trimethylbenzenes by exercising volunteers concluded, as a result of their analysis of questionnaire responses on CNS-related symptoms, that there were no acute effects from short-term inhalation exposure at 25 p.p.m.

In none of these human studies were effects on organ systems other than the central nervous system investigated. Indeed, given that the studies dealt with, at worst, mild acute effects, and that retention times of such compounds in the body are short, it is difficult to imagine that effects on other organs could have been demonstrated.

Sulfur-containing compounds

The most obvious potential inhalation hazard from sulfur containing compounds in crude oil is hydrogen sulfide. It is acutely toxic and in high concentrations (above about 500–1000 p.p.m.) causes unconsciousness and death; between 50 and 500 p.p.m. it acts primarily as a respiratory irritant [39]. Typical sweet crude oils contain 5 p.p.m. to 6 p.p.m. hydrogen sulfide by weight, while well-head concentrations of hydrogen sulfide in sour crude oils as high as 18% by weight have been reported [40]. Because hydrogen sulfide is a gas, it will rapidly dissipate from spilled oil and therefore, even with high initial concentrations in the crude oil, it is likely to present only a very short-term hazard to spill cleanup workers except in cases where spilled oil is continually being renewed or exposed. Zhou & Wong [13] in fact suggest on the basis of numerical modeling that (if there is no continuous supply of fresh oil), hydrogen sulfide would be lost from a spill in a matter of minutes; in this case, the gas would present no hazard to cleanup workers, except perhaps during the first approaches to a spill.

Methyl mercaptan, also a gas at room temperature, has a similar acute effect to hydrogen sulfide—namely, it acts on the respiratory center producing death by respiratory paralysis [ref. 41, p. 405]. It also causes depression of heme-synthesizing enzymes [42]. The next homolog, ethyl mercaptan, is somewhat less toxic (with a toxicity some one-fifth that of hydrogen sulfide) [43].

The TLVs for the mercaptans, 0.5 p.p.m. in each case [43], are much lower than the probably more acutely toxic hydrogen sulfide (TLV 10 p.p.m.). All these levels, however, are set on the basis of odor and irritation rather than on acute respiratory toxicity [41,43]. It is these sensory reactions that work in favor of the spill cleanup worker—odor and irritation serve as warnings and enable actions to be taken before hazardous levels are reached.

Chronic effects

There is nothing in the literature pertaining to chronic effects arising from inhalation exposure to crude oil. In contrast, there are literally thousands of papers dealing with the chronic effects of inhalation exposure to specific crude-oil components. However, the observation of Eide & Zahlsen [32] that the uptake of individual hydrocarbons was unaffected by other hydrocarbons in the inhaled mixture indicates that, at least at the moderate exposure levels studied (up to a few hundred p.p.m.), effects observed for individual compounds might be used in a prediction of the effects of exposure to crude oil.

In contrast to acute effects of inhalation exposure, which essentially involve only the central nervous system, chronic effects encompass a broader range of organ systems. It is convenient therefore to treat these effects by organ system rather than by classes of substance.

Hematopoietic toxicity

Benzene, an acknowledged carcinogen, provides perhaps the clearest example of a chronic effect associated with inhalation of a particular component of crude oil—namely leukemia in humans chronically exposed to high levels in their workplaces. (In one of the largest epidemiological studies into the potential effects of workplace exposure to benzene—a study discussed in the Conclusions—average benzene exposure levels were of the order of tens of p.p.m. [44].) Benzene toxicity in humans has been characterized as either early reversible hematotoxicity or, with prolonged exposure to high doses, irreversible bone marrow damage which ultimately is expressed in the form of aplastic anemia and acute

mylogenous leukemia [45]. This toxicity arises from a metabolite or metabolites of benzene—principal among which are phenolic and quinone intermediates in the oxidative metabolism of benzene (see for example: [45–51]).

Because benzene is a 'key' compound in regulative strategies aimed at controlling occupational and environmental exposures, there is considerable effort devoted to quantifying the risks to health of benzene exposure. Currently, some of this work is focusing on whether there is a 'threshold' exposure level below which irreversible damage to the hematopoietic system will not occur. Two recent re-analyses [52,53] of the leukemia incidences in the cohorts (Pliofilm workers in Ohio) on which current regulatory initiatives are based, suggest that the original risk determinations (conducted by Infante *et al.* [54], with an update by Rinsky *et al.* [55]) considerably overestimated the risk; and one of these workers, Paxton [53], claimed that her analysis of the data is consistent with a threshold model for leukemogenesis by benzene. Cox [56] employed biologically based risk-assessment-modeling—using, as the measure of exposure, an 'internal dose', derived by physiologically based pharmacokinetic modeling, instead of merely the 'administered' benzene dose-levels themselves. He concluded 'There is no evidence of a positive relation between benzene exposure and tumor probability... at [airborne] benzene concentrations below 1 p.p.m.' and 'analytic approaches... suggest that the...curve relating benzene concentrations to AML [acute mylogenous leukemia] risk at sufficiently low, constant concentrations of benzene approaches a zero or negative slope as concentration falls below about 10 p.p.m.'—in other words, he too is predicting a threshold for benzene-induced leukemia. Definitive conclusions regarding these 'threshold' assertions will have to await a better understanding of leukemogenesis in general and of benzene-induced leukemia in particular. Considerable progress is being made in this regard with explanations of the processes at the genetic level becoming available (see, for example, [48,51]).

Currently, in the United States, the US Occupational Safety and Health Administration (OSHA) mandates a permissible 8-h time-weighted average occupational exposure limit (PEL) of 1 p.p.m. [57]. The American Conference of Governmental Industrial Hygienists has set a TLV of 0.5 p.p.m. [58]. The (U.S.) National Institute for Occupational Safety and Health (NIIOSH) has set a recommended 8-h time-weighted average occupational exposure limit (REL) of 0.1 p.p.m. [59]. Current occupational exposure limits for benzene in a number of other industrialized countries are in the range 1–10 p.p.m. (as 8-h time-weighted averages) [20]. Given that one might expect benzene concentrations in the vicinity of a large spill of crude oil to be in excess of 1 p.p.m. for several hours (see, for example, the modeling of the *Exxon Valdez* spill by Hanna & Drivas [12]), such exposure limits could present a requirement for oil-spill workers to wear air-supplied respirators, at least at the start of a cleanup—or when dealing with a continuous blowout, for instance.

Nephrotoxicity

There have been numerous experimental studies linking hydrocarbon exposure with kidney damage. The response has been found only in adult male rats, which have a kidney biochemistry that makes them uniquely susceptible [60]. In humans, the available data consist of case reports, case control studies, cohort mortality studies, and cross-sectional studies [61], but the association between hydrocarbon exposure and kidney disease is tenuous at best. The majority of epidemiological studies discussed by Phillips *et al.* [61] were either inconclusive or their methodology sufficiently flawed that positive findings could be called into question.

Neurotoxicity

There is strong evidence that hexane is neurotoxic. The effects of exposure, generally a polyneuropathy with muscular weakness and sensory impairment in the extremities, have been observed among workers involved in footware manufacturing where glues containing hydrocarbon solvents were used. Hexane is metabolized to 2,5-hexanedione which is the identified neurotoxin [26]. The nerve damage takes several months to develop and on cessation of exposure there is slow recovery [62]. Such extended exposure is unlikely for a typical oil-spill clean up, particularly as hexane is volatile and would be lost within a few hours at most [see for example ref. 13]. Exposure to hexane might remain a concern in spills where fresh oil is continuously released or otherwise exposed to the air.

Experimental studies on the effects of chronic exposure of rats to toluene at 100 p.p.m., 300 p.p.m. and 1000 p.p.m. (8 h a day, 6 days a week for 16 weeks) found a dose-dependent increase in glial cell markers while, at the same time, neuronal markers essentially showed no change [63]. The increase in glial markers was noticeable at the lowest exposure level (the TLV for toluene) and was evidence of a proliferation of the glial cells. However, the implications of this proliferation are uncertain, and, as the authors themselves reported, neither signs of peripheral neuropathy nor behavioral abnormalities were seen.

Occupational exposure to mixed hydrocarbon vapors have also been examined for evidence of chronic effects with equivocal results. In an apparently carefully designed study using 21 pairs of monozygotic twins in Finland, Hänninen *et al.* [64] administered a series of psychological tests. The findings showed that the exposed twins had lower performance in associative learning and digit span tests (both of which measure verbal memory), and block design (which measures visual and spatial ability) than their nonexposed siblings. Hänninen *et al.* added that even in the presence of a clear intrapair difference, most of the exposed persons still performed within the normal range; they would have been considered as unaffected if assessed as single individual cases.

Two other studies—Van Vliet *et al.* [28] and Antti-Poika *et al.* [65]—that did not have the singular advantage of a genetically identical control group failed to demonstrate significant differences between exposed and control groups. The study by Van Vliet *et al.*, which used a questionnaire to investigate neurasthenic effects in 379 Dutch painters and a similar number of construction worker controls, has already been described in the section on Acute Exposure effects. The study by Antti-Poika *et al.* involved 48 persons exposed to paint solvents, and 40 referents. These authors estimated that the lifetime exposure of the exposed group ranged from 10 to 330% (average 60%) of the Finnish occupational exposure standard. The methods used were a neurological examination, electroencephalography, brainstem auditory evoked potentials, electronystagmography, and posturography. Antti-Poika *et al.* observed that the findings classified as abnormal were slightly more common in exposed than in referents but the differences were not statistically significant.

Effects of exposure to oil mists and aerosols

There is little in the literature regarding the possible health effects of inhaling crude-oil-containing aerosols. Nevertheless, it is well known that even very small quantities of aliphatic hydrocarbons in contact with the lining of the lung (through direct introduction into the lung or through aspiration during emesis) give rise to a severe chemical pneumonitis, characterized by pulmonary edema, hemorrhage and tissue necrosis, which can be fatal [26]. And there has been a report of accidental aspiration of crude oil causing such chemical pneumonitis—the individual, in this case, also developed symptoms of hepatic and renal toxicity, but recovered completely [66]. Campbell *et al.* [18], studied acute symptoms among the population living within 4.5 km of the grounding (and subsequent break up) of the oil-tanker *Braer*—an event that released some 85 000 tons of crude oil to the ocean over six days. With the exception of somewhat higher incidences of headaches, throat irritation, and itchy eyes among the target population compared with the control population located 95 km from the scene of the spill, no significant differences were found for any of the biological markers investigated. These tests included urinalysis for the hydrocarbon toxicological markers, methylhippuric isomers and hippurate to creatinine ratios, and blood analyses to determine liver and renal functions. A follow-up study some five months after the oil spill, found that the exposed group was more likely to report being in poor or declining health than would the controls, and the exposed group also reported a higher incidence of weakness [67]. Given that the control and exposed groups were clearly identifiable *a priori*, the possibility of subjective bias in such reports seems to leave their significance open to question.

Systemic effects in rats and mice arising from single, acute exposures to high concentrations of mists generated from components of synthetic crude oil from tar sands have been reported [68]. The effects were weight increases in liver and kidney, and in many cases the animals died. The causes of death were not identified in the paper. The significance of this experiment, for the purposes of this section, is that it establishes that components of crude oil inhaled as an aerosol can pass through the alveolar membrane and therefore have the potential to cause toxic responses in other organs.

During a spill cleanup, mists and aerosols provide a route by which non-volatile components of crude oil (both fresh and weathered) may enter the body and give rise to toxic responses. In the case of PAHs, Gerde & Scholander [69,70] have argued that the uptake of such components is facilitated when the compounds are contained in particles that can penetrate the protective bronchial lining layer and make contact with the underlying bronchial epithelium. It seems probable that oil-containing mists and aerosols could provide such a penetrating vehicle. Mists and aerosols containing polynuclear aromatic hydrocarbons (PAHs) are, in principle, a cancer risk, but it is not clear whether such particles could be formed in sufficient quantities or of the appropriate size under spill cleanup conditions to make the risk significant. Similarly, the evidence provided by the acute exposure experiment of Stubblefield *et al.* [68] that exposure to synthetic crude-oil aerosols gives rise to measurable changes in organ weights in rats and mice, suggests that systemic effects from exposure to crude-oil aerosols cannot be ruled out.

Occupational exposure to oil-based mists is common in metalworking activities that employ cutting fluids, and there is a substantial body of literature dealing with the effects of such exposure. Cutting fluids may be grouped into four major categories: straight metalworking fluids, which are undiluted mineral and fatty oils; soluble metalworking fluids, which are water emulsions of mineral and fatty oils; synthetic metalworking fluids, which are chemical solutions of organic compounds and inorganic salts in water; and semisynthetic metalworking fluids, which are emulsions of mineral oil and the chemicals found in synthetics [71]—oil mists generated from the use of straight cutting oils and soluble cutting oils would probably be the closest in properties to mists generated during oil-spill cleanup.

Straight cutting oils have been in widespread use since the 19th century, and exposure to such oils have been linked, through a mortality study of machining-fluid exposure in the automobile industry, to slightly increased incidences of rectal, laryngeal, and prostatic cancers [72]—the cancers were attributed to the presence of PAHs in straight cutting oils. Another mortality study of machinists exposed to oil mist (predominantly from straight cutting oils), however, failed to detect significant differences in mortality between exposed and nonexposed workers [73]. This study also failed to detect significant differences in respiratory performance between the exposed and nonexposed groups.

HEALTH EFFECTS OF DERMAL EXPOSURE

In contrast to the information available regarding the effects of human inhalation exposure to crude oil, the situation with regard to dermal exposure is much more limited. This is not to imply that dermal exposure to crude oil produces no toxic effects. It appears, however, that most studies have involved animal models and components or derivatives of crude oil, rather than crude oil itself.

There are a number of possible effects from dermal exposure to hydrocarbons found in crude oil, and they are not necessarily restricted to effects on the skin. As with the effects of inhalation exposure discussed in the previous section, their nature and degree depend on the composition of the oil, but can be broadly categorized as chronic or acute.

Acute effects

Irritating effects of crude oil on skin have been reported sporadically, but do not appear to be overly prevalent. For example, in a survey of about 1000 California and Texas oil field workers conducted in the late 1930s, about 15% were identified as having some form of dermal lesions (mainly keratotic in nature and mild in effect) [74]. Birmingham [75] suggested that the majority of these lesions were probably caused by workers using solvents to remove crude oil from their skins.

More recently, in the cleanup of the *Exxon Valdez* spill, medical personnel reported cases of work-related dermatitis of the hands and forearms of cleanup workers (some 10 000 such workers being involved)—effects that were attributed to the improper use or nonuse of personal protective equipment [ref. 7, p. 27].

Although such dermatitis is probably the result of exposure to the lighter components of crude oil, there is also evidence that involatile components—specifically PAHs—can produce acute effects. It is generally accepted that the dermal toxicity (both acute and chronic) of PAHs is the result of oxidized products formed during metabolism or other processes [ref. 76, chap. 4 and 6]. Griest *et al.* [8] attributed a

large decrease in the level of PAHs in weathered oil (and an increase in the neutral polar fraction of compounds) from the *Exxon Valdez* spill to the effects of natural oxidation. Similar results were found by Gorman *et al.* [7].

An implication of such data is that PAHs in combination with sunlight could be expected to be more irritating than either component alone; such photosensitization of human skin by hydrocarbons has been found to occur after exposure to materials such as coal tar which contain relatively large amounts of PAHs [77]. And certainly the combination of dermal exposure to crude oil and sunlight is one that is likely to be experienced by oil-spill workers. However, there is little clear cut evidence on either the degree of exposure or the likely acute effects of such exposure in humans.

On the other hand, photosensitization caused by crude oil has been demonstrated in animal models. Crude oil administered dermally to the skin of mice and followed by exposure to ultraviolet light produced skin erythema, whereas the administration of either ultraviolet light or crude oil alone had no effect [77]. In a similar experiment, Burnham & Bey [78] found that the combination of dermally administered crude oil and ultraviolet light brought about more inhibition of immune function within the skin of mice than did ultraviolet light alone. The combination of crude oil and ultraviolet light also caused a sixfold reduction in the number of Langherans' cells (cells important to the immune response of the skin), which were unaffected by either agent separately. Burnham & Bey speculated that such immunosuppression could prevent the destruction of cells transformed by carcinogenic compounds, and therefore raise the risk of cancer induction by such agents as PAHs. Similar studies *in vitro* indicated that the effect on mouse skin was compound-specific; on exposure to ultraviolet light, Langherans' cells were significantly depleted in the presence of anthracene but not phenanthrene or benzo[a]pyrene [79]. The substances responsible for the UV-related depletion observed with crude oil itself were not identified; Burnham & Rahman suggest that anthracene, for instance, may be present at too low a concentration to account for the observations.

In addition to such photosensitizing effects, PAHs have been shown in animal experiments to produce acute biochemical changes after dermal application. For example, Rahimtula *et al.* [80] found that application of Kuwait Crude oil to the skin of rats increased dermal levels of the enzymes benzo[a]pyrene 3-hydroxylase and diphenyloxazole hydroxylase by an order of magnitude. A light oil (without PAHs) was much less effective. A later study by Rahimtula *et al.* [81] found that dermal application of Prudhoe Bay Crude oil to the backs of mice caused rapid induction of epidermal ornithine decarboxylase, a regulatory enzyme in the growth process. In each of these two studies, the effects were reversed after a few hours following a single exposure. It is not clear, however, what the toxicological implications are, or what might be the results of repeated exposures.

Chronic effects

No data appear to be available on chronic human effects that are specifically and rigorously linked to dermal exposure to crude oil. Some studies, however, suggest that such effects may be possible.

Tumorigenesis

For example, in the 1930s survey of California and Texas oil workers discussed above, seven workers out of the 743 California workers had epitheliomas, and five of these had experienced heavy exposure to crude oil and sunlight [ref. 74, p. 315]. There were 209 workers in this heavily exposed group giving an incidence of epitheliomas of about 2.5%. It appears from the account given by Schwartz *et al.* that the incidence of epitheliomas among the general population of the area was of the order 0.3%. (In contrast, the Texas worker cohort (330 individuals) '... did not show a high incidence of epitheliomas or dermatitis. The wells in southern Texas are free flowing; therefore, pumping and well pulling are not necessary.... After a well is brought in, there is but little contact with oil. These factors may account for the comparatively lower incidence of dermatoses among the workers in the south Texas oil fields.' [74]).

In a case referent study, carried out in Poland, of 376 men suffering from skin cancer, the exposure to PAHs and other risk factors was assessed using data as to the length of the men's employment and their job descriptions. The only statistically significant source of risk identifiable was exposure to PAHs in mineral oils [82]. Exposure to PAHs without exposure to mineral oils showed no significant increases in cancer risk. The degree of exposure and implications for oil-spill workers are not obvious from this work,

but the importance of oils as 'carriers' of PAHs is consistent with the results of other studies which indicate that certain hydrocarbons may act as tumor initiators or promoters.

Several groups have investigated in animal models the dermal toxicity of various refinery streams, particularly middle distillates (petroleum fractions with a boiling range of ≈180 to 380 °C and a negligible PAH content). Broddle *et al.* [83] found that middle distillates were tumorigenic and that this property did not appear to be correlated with the irritancy of the specific distillate. Freeman *et al.* [84] similarly found that altering exposure conditions to reduce skin irritation (by diluting the distillate or carrying out only intermittent dosing) also reduced the tumor incidence; but on the basis of comparisons among various middle distillates, chronic irritation was not always associated with tumorigenesis. Both they and Broddle *et al.* accepted that middle distillates were tumor promoters rather than initiators. A less clear-cut picture was implied by the work of Jungen *et al.* [85]. They investigated several middle distillates and found evidence that such products could be either tumor promoters or initiators, or both, or neither. They also concluded that middle distillates with high mutagenic potency act as tumor initiators in the skin of CD-1 mice and those with a high dodecane content act as promoters.

In 1989, after reviewing 14 animal dermal carcinogenicity studies using mice, rats and rabbits, the International Agency for Research on Cancer (IARC) reported that there was limited evidence for the carcinogenicity of crude oil in experimental animals [86]. Four of the studies showed no carcinogenicity from crude oil (possibly because of the low doses used, or the short duration of treatment), and most of those showing positive results were criticized for deficiencies such as lack of appropriate control data or a small number of animals used. In their report, IARC also stated there was inadequate evidence for the carcinogenicity of crude oil in humans.

Non-tumorigenic effects

In a 28-day subchronic study of dermal exposure to rats, at 0.25 and 2.5 g/kg bodyweight, a crude oil produced very slight skin irritation at the higher dose, and some loss of appetite and depression in weight gain [87]. In female rats, the sizes of livers were increased compared to controls (both absolutely and as a fraction of body weight), and the adrenal glands showed a dose-related increase in weight. There were also indications of dose-related biochemical changes, including an increase in globulin levels in female rats. In a more detailed study, but one employing various refinery streams rather than crude oil, Feuston *et al.* [88] examined dermal subchronic toxicity and developmental toxicity to the rat. These workers found that a number of toxic end-points could be correlated with the levels of 3- to 7-ring PAHs and nonbasic nitrogen heterocyclic aromatics in the various products. Endpoints reflecting subchronic toxicity included decreased red blood cell count, decreased hemoglobin levels, decreased thymus weight, increased liver weight and increased cholesterol level. The measures of developmental toxicity were decreased fetal weight and increased incidence of fetal resorption. Unlike these developmental effects, the subchronic endpoints appear to have a less clear-cut toxicological significance. The implications of the endpoints for oil-spill workers are correspondingly obscure.

Some animal studies have provided evidence that the properties of the 'carrier' solvent can strongly affect the toxic properties of PAHs. Holland *et al.* [89], for example, investigated the residence of benzo[a]pyrene in the skin of mice in the presence of different amounts of mineral oil in cyclohexane, using the fluorescence of the PAH as in indication of its distribution in the skin, and tritium-labeled benzo[a]pyrene to follow the clearance of the PAH. They found that increasing the fraction of mineral oil in the cyclohexane reduced the residence time, evidently by facilitating PAH metabolism.

Holland *et al.* [89] interpreted their data in terms of the chances that the benzo[a]pyrene would become trapped in the sebaceous glands, which would act as a 'time release' reservoir of active carcinogen. In the presence of mineral oil, such trapping was minimal and the PAH was rapidly absorbed in the stratum corneum where it could undergo natural clearance. Holland *et al.* suggested that at low PAH concentrations this should result in less cumulative damage as a result of lower exposure times and less metabolite flux; however, at high PAH concentrations, systemic effects could be exacerbated because of the concentrations of reactive metabolites entering the circulation.

One of the implications of such findings is that weathering will be a factor in the toxicity of crude oil. Weathered oil is likely to be more viscous than unweathered oil, as the lighter components are removed

by evaporation, and this may alter its properties as a 'carrier' of PAHs. Weathering can also be expected to influence the toxicity of the oil by altering the concentration of PAHs. In general one might expect an initial rise in PAH concentration as the volatile components of the oil are removed, followed by a fall, due to weathering of the PAHs themselves. In this second phase, there remains unanswered the question of the toxicity of the degradation products of PAHs, in the presence of all the other components of the oil.

CONCLUSIONS

There is more evidence that various components of crude oil pose chronic or acute health risk than that crude oil itself does—an observation that stems, at least in part, from the inevitable difficulty of carrying out meaningful experiments with such a variable and complex substance as crude oil. In the case of chronic effects from inhalation exposure, no investigation of the question seems to have been published. On the other hand, there is ample evidence that inhalation of individual components can produce both acute and chronic toxic effects. For dermal exposure, in a few cases, crude oil itself has been shown to produce toxic effects in both humans and animals. In general, however, a clear link has not been established between the dermal toxicity of crude-oil components individually and their human toxicity within crude oil.

What is needed is to go from the data on individual effects to predictions about the effects of exposure—dermal or inhalation—to crude oil itself under spill cleanup conditions. Such predictions will have to take into account not only expected levels of exposure but also the probable durations of such exposures. Unfortunately there is no such thing as one 'crude oil' and so some investigation of its components in isolation seems inevitable. Work, such as that of Feuston *et al.* [88], suggests it may prove possible to correlate the compositions of complex hydrocarbon mixtures like crude oil with their dermal toxicity, while that of Eide & Zahlsen [32] suggests the uptake of inhaled hydrocarbon mixtures may be estimated from measured uptake data on the separate components. At present, the most obvious information gaps seem to be an estimate of the likely dermal exposures during a spill cleanup and, possibly, a realistic assessment of the chronic effects of exposure to crude-oil aerosols.

Partly because of the absence of data regarding exposure levels these workers face and partly because of an absence of information concerning the health effects of crude oil itself, there is very little to say regarding actual health effects occasioned by cleanup-workers' exposure to crude oil. Furthermore, there seem to be no comprehensive epidemiologic studies on oil-spill cleanup workers. This may reflect the fact that oil spills, and therefore the exposures of cleanup workers, are not particularly frequent, and comparatively limited numbers of workers are involved.

As indicated at the beginning of this paper, oil-industry workers are exposed to at least some of the same substances that may be encountered in an oil spill. And a considerable number of epidemiologic studies deal with the oil industry in general; several of these are summarized in Table 4. Although the primary exposure route is probably inhalation in all these epidemiological studies, dermal exposure may also be significant in certain instances (e.g. benzene exposure to oil-distribution workers, for whom there would also be a potential for dermal contact [90]). To this extent industrial exposure may serve as a model for exposure during a spill cleanup. Indeed, the *cumulative* occupational exposures in the oil industry are likely to be significantly higher than those resulting from the inevitably sporadic work of oil-spill cleanup. The results of oil-industry epidemiological studies may therefore provide an upper bound on the potential for adverse effects in oil-spill cleanup workers.

By and large, studies investigating the overall chronic-health status of oil industry workers show, at most, marginal evidence of elevated cancer incidences. (Non-cancer effects appear to have been studied relatively rarely.) The situation with studies focusing on relationships between benzene exposure and hematopoietic disjunction is somewhat clearer. The causality is relatively well established, but the quantitative relationship is still the subject of considerable debate.

Given the small numbers of oil-spill workers, similar epidemiological studies might be expected to be incapable of demonstrating the existence or absence of elevated cancer risk. Further, given the potentially lower cumulative exposure of cleanup workers compared to their oil-industry counterparts, such risk may well be negligible. Judicious employment of good industrial hygiene practices will help ensure that this is the case.

Table 4 Some epidemiologic studies on petroleum refinery and petrochemical workers

Paper	Methodology	Findings
General studies		
[92]	A mortality study of 14 179 current and former refinery workers. Data were based on work histories and death certificates. US national mortality rates were used as the basis of comparison.	Only lymphopoietic cancer showed an increased risk that was possibly related to an occupational exposure.
[93]	Retrospective study of 12 894 workers employed for at least 6 months in petroleum production or pipeline work. Data were derived from work histories and death certificates. Analysis was restricted to 11 098 white male workers; mortality rates and disease incidence were compared to those in the U.S. white male population.	Only thyroid cancer, leukemia and benign neoplasms appeared to occur more frequently than in the reference population, and none of these increases in frequency were statistically significant.
[94]	Follow-up of a mortality study of 1027 men employed in a Canadian oil refinery. Data were derived from work histories and death certificates. Mortality rates were compared with age-and cause-specific rates in the province of Québec.	Only deaths from brain cancer showed a significantly elevated rate, and this frequency could not be related to any workplace hazard.
[95]	Case referent study investigating risk of death from brain tumors among workers in the petroleum refining and petrochemical industries. The study looked at brain-tumor deaths in northern New Jersey, Philadelphia and the Gulf Coast of Louisiana over a three-year period. The analysis was based on 300 cases and 386 referents.	The authors concluded that there was 'little evidence to suggest that excess brain tumor risk is associated with long-term employment in the petroleum refining industry'.
[96]	Prospective study of mortality in ≈ 15 000 male workers in Australia, employed in the Australian petroleum industry during 1981–89.	No statistically significant increases in mortality compared to the Australian general population.
[97]	Blood-cell counting and testing of 2086 current and retired employees at a petrochemical complex, over 4 years.	No evidence was found for an unusual level of abnormal complete blood-cell counts. Four cases of myelodysplastic syndrome were reported, but published data were inadequate to confirm that these represented an elevated risk.
[98]	Study of morbidity in 3422 male refinery and petrochemical workers. Data were obtained from medical records, and morbidity rates were compared to rates among male employees in all the manufacturing locations of the same company.	Overall morbidity was slightly elevated among production workers. Elevated incidences of specific diseases either were not obviously associated with occupational factors or were based on very few cases.
[99]	Case-control study of 102 kidney cancers among 18 323 deaths in ≈ 100 000 male petroleum refinery workers.	The authors state, 'The results of the analyses of [hydrocarbon] exposure ratings are most consistent with the hypothesis of no effect on kidney cancer risk, or an effect too small for this study to measure with precision.'

Table 4 *continued*

[100]	Follow-up mortality analysis of 34 569 oil-refinery and 23 306 distribution-centre workers in the UK. Causes of death were obtained from death certificates; mortality rates were compared to those in the general population of England, Scotland or Wales, as appropriate.	Nonsignificant increases in kidney cancer and leukemia among distribution workers.
[101]	Meta-analysis of all cohort studies of petroleum workers (> 208 000) in the US and UK, to investigate leukemia mortality by cell type.	No significant increases in mortality for any cancer cell-types investigated.
[102]	Follow-up of mortality study among ≈ 18 000 employees at a Texas oil refinery. Mortality rates were compared to those in the Texas and US general populations.	Significant mortality increases for bone cancer, acute lymphocytic leukemia and benign/unspecified neoplasms; but none of these showed an exposure-response relationship with length of employment.
[103]	Follow-up mortality study of 9720 petroleum-refinery and chemical-plant employees. Mortality rates were compared to those in the local county of Texas.	Increased mortality from lymphatic and hematopoietic cancers, particularly among refinery workers; but none of the increases were statistically significant.
Benzene-related studies:		
[104]	Mortality study of 74 828 benzene-exposed and 35 805 nonexposed workers in China. Estimated exposure levels ranged to over 50 p.p.m. (cumulative exposures of over 400 p.p.m.-years).	Deaths due to lymphatic and hematopoietic malignancies showed significant increases with increasing benzene exposure.
[105]	Cohort study of 74 828 benzene-exposed and 35 805 nonexposed workers in China. Hematopoietic and lymphoproliferative disorders (HLDs) associated with benzene exposure were identified from slides, pathological or medical records and other sources.	Qualitative identification of a diversity of HLDs possibly associated with benzene exposure.
[53]	Update and re-analysis of the 'Pliofilm' cohort (1868 workers occupationally exposed to benzene 1936–75).	Confirmation of earlier correlation between benzene exposure and increased incidence of leukemia. Two sets of exposure estimates suggest that only cumulative benzene exposures > 50 p.p.m.-years had significant leukemogenic potential; an earlier set implies that much lower exposures may be leukemogenic.
[106]	Biochemical and physiological study of 44 healthy workers exposed to benzene and 44 unexposed controls in Shanghai, China.	In exposed workers compared to controls, total white blood cell, absolute lymphocyte, platelet, and red blood cell counts were reduced, and glycophorin A mutation frequencies elevated.

Table 4 *continued*

[107]	Case-control study of 14 leukemia cases (and 55 controls) among Canadian petroleum distribution workers exposed to low levels of benzene.	Duration of benzene exposure was more closely associated with leukemia risk than other measures of exposure, but results were not statistically significant.
[108]	Mortality study of 74 828 benzene-exposed and 35 805 nonexposed workers in China.	Significantly increased mortality (compared to unexposed workers) for leukemia, malignant lymphoma and lung cancer. Among leukemia subtypes, only acute myelogenous leukemia was significantly increased.
[90]	Case-control study of 91 leukemia cases among workers within the petroleum distribution industry and 4 controls per case selected from the corresponding companies.	No significant increase in risk of all leukemias with cumulative exposure to benzene or intensity of exposure, but risk consistently doubled in subjects employed in the industry for > 10 years.

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