POTENTIAL FOR PHOTOENHANCED TOXICITY OF SPILLED OIL IN PRINCE WILLIAM SOUND AND GULF OF ALASKA WATERS

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Prepared by:

Mace G. Barron
1134 Avon Lane
Longmont, Colorado 80501
t / f: 303-684-9646
e: macebarron@hotmail.com

Prepared for:

Prince William Sound Regional Citizens' Advisory Council
3709 Spenard
Anchorage, Alaska 99503

Abstract

This report examines the potential for photoenhanced toxicity of spilled oil in Prince William Sound and associated Gulf of Alaska waters. Photoenhanced toxicity is the increase in the toxicity of a chemical in the presence of ultraviolet light (UV), compared to a standard laboratory test conducted with fluorescent lighting (minimal UV). Oil products and weathered oil are phototoxic, as are specific polycyclic aromatic compounds present in oil. Photoenhanced toxicity may occur through two processes: photomodification and photosensitization, which are further detailed in the report. No studies have investigated the photoenhanced toxicity of oil in Alaska waters. Although there are substantial uncertainties, the results of this evaluation indicate there is potential for photoenhanced toxicity of spilled oil in Prince William Sound and associated Gulf of Alaska waters. Additional research needed to characterize the potential for photoenhanced toxicity may include determining the seasonal and spatial variability in UV irradiance of the habitats of potentially exposed organism. Dose-response studies could be performed to establish UV and oil exposure thresholds necessary for photoenhanced toxicity. Additionally, the phototoxicity of chemically dispersed oil could be evaluated in the laboratory and compared to the photoenhanced toxicity of non-chemically dispersed oil.

Executive Summary

This report examines the potential for photoenhanced toxicity of spilled oil in Prince William Sound and associated Gulf of Alaska waters. Photoenhanced toxicity is the increase in the toxicity of a chemical in the presence of ultraviolet light (UV), compared to a standard laboratory test conducted with fluorescent lighting (minimal UV). Specific chemicals have been shown to exhibit a 2 to greater than 1000 fold increase in toxicity in the presence of UV. This report provides a general overview of photoenhanced toxicity to aquatic organisms and is intended for a general audience.

Oil products and weathered oil are phototoxic, as are specific polycyclic aromatic compounds present in oil. Photoenhanced toxicity may occur through two processes: photomodification and photosensitization. *Photomodification* is the structural modification of chemical in water to more toxic/reactive compounds. In *photosensitization*, the bioaccumulated chemical transfers light energy to other molecules causing tissue damage. The available evidence indicates that the photoenhanced toxicity of oil occurs through a photosensitization mechanism, and the phototoxic components of oil are primarily 3 to 5 ring polycyclic aromatic hydrocarbons (PAHs) and heterocycles (PAHs containing an oxygen, sulfur, or nitrogen in place of a carbon). Photoenhanced toxicity should be considered in oil spill response because estimates of the spatial and temporal extent of injury to aquatic organisms may be greater. Additionally, the choice of counter measures and oil removal operations may influence the degree of photoenhanced toxicity.

Determinants of photoenhanced toxicity will include the chemical composition, physical properties, and environmental fate of the oil, environmental conditions, and oil exposure and bioaccumulation by aquatic organisms, and exposure of the oil residues in the organism to UV. Factors affecting UV exposure include photoperiod, sun angle, light reflectance, and cloud cover. Decreasing light penetration in the water column is termed attenuation, and is affected by turbidity, shading, phytoplankton concentrations, and dissolved organic carbon. The potential hazard of photoenhanced toxicity may be greatest for embryo and larval stages of aquatic organisms that are relatively translucent to UV and inhabit the photic zone of the water column and intertidal areas.

No studies have investigated the photoenhanced toxicity of oil in Alaska waters. Although there are substantial uncertainties, the results of this evaluation indicate there is potential for photoenhanced toxicity of spilled oil in Prince William Sound and associated Gulf of Alaska waters. North Slope crude is known to be phototoxic, phototoxic PAHs were present in Exxon Valdez spill water, and phototoxic PAHs accumulate in aquatic organisms exposed to weathered Exxon Valdez oil. Additionally, under some circumstances sufficient UV radiation may penetrate the water column to

elicit photoenhanced toxicity. Chemically dispersed oil is likely to exhibit photoenhanced toxicity because phototoxic PAHs are dispersed in the water column and can be bioaccumulated by aquatic organisms. However, no research has evaluated the photoenhanced toxicity of chemically dispersed oil, and it is unknown if chemically dispersed oil would be more or less phototoxic than non-chemically dispersed oil.

Additional research is needed to reduce the uncertainties of the photoenhanced toxicity of spilled oil in Alaska waters. Research that would better characterize the potential for photoenhanced toxicity may include determining the seasonal and spatial variability in UV irradiance of the habitats of potentially exposed organisms. Dose-response studies could be performed to establish UV and oil exposure thresholds necessary for photoenhanced toxicity. Additionally, the phototoxicity of chemically dispersed oil could be evaluated in the laboratory and compared to the photoenhanced toxicity of non-chemically dispersed oil.

INTRODUCTION

Traditionally, toxicological studies used to define the hazards of PAHs and oil have been conducted under fluorescent light, which has minimal ultraviolet light (UV) (Arfsten et al., 1996). As discussed below, laboratory studies have demonstrated that the toxicity of polycyclic aromatic compounds increases 2 to greater than 1000 times in the presence of UV. This photoenhanced toxicity occurs at the UV wavelengths that occur in the water column of aquatic environments: UVB (280 to 320 nm) and UVA (320 to 400 nm). Not all chemicals exhibit photoenhanced toxicity because specific structural features are necessary for phototoxicity. Recent research has shown that oil and components of oil exhibit photoenhanced toxicity under simulated natural sunlight (Pelletier et al., 1997; Calfee et al., 1999).

This report provides a general overview of photoenhanced toxicity to aquatic organisms and discusses the potential for photoenhanced toxicity of oil spilled in Prince William Sound and associated Gulf of Alaska waters. This report is intended for a general audience. The mechanisms of photoenhanced toxicity and types of phototoxic chemicals present in oil are reviewed. The importance of considering photoenhanced toxicity in oil spill response and the determinants of photoenhanced toxicity in aquatic environments are presented. This report also discusses the potential for photoenhanced toxicity of spilled oil and chemically dispersed oil in Alaska waters, although research specifically addressing these issues has not been performed.

Overview of Photoenhanced Toxicity

A number of chemicals are known to be photoactivated, showing a 2 to greater than 1000 fold increase in toxicity in the presence of ultraviolet light (Oris and Giesy, 1987; Landrum et al., 1987; Larson and Berenbaum, 1988; Arfsten et al., 1996; Boese et al., 1997; Pelletier et al., 1997). For example, Pelletier et al. (1997) exposed mysid shrimp and bivalve embryos to individual PAHs (anthracene, fluoranthene, pyrene) for 48 hours using a 16 hr photoperiod under UV (UVA: 397 $\mu W/cm^2$; UVB: 134 $\mu W/cm^2$) or fluorescent lighting (UVA: 9.7 $\mu W/cm^2$; UVB: 3.4 $\mu W/cm^2$). Under fluorescent lighting, aqueous PAH concentrations reducing larval shrimp and bivalve embryo survival (LC50, EC50) ranged from 25 to >11,900 $\mu g/L$. Under UV, PAHs were 12 to greater than 50,000 times more toxic (Pelletier et al., 1997).

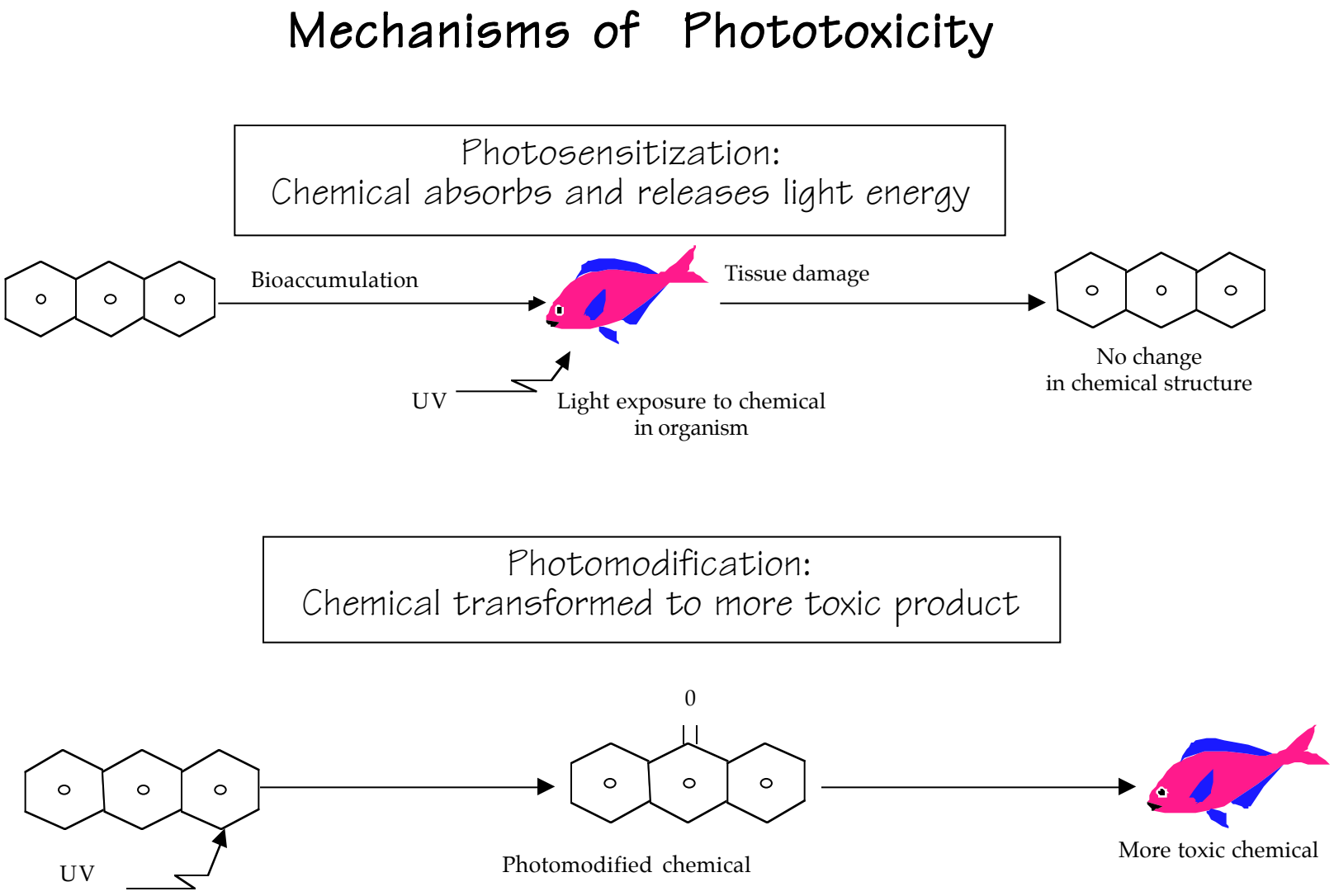
Photoenhanced toxicity to aquatic organisms may occur through two mechanisms as depicted in Figure 1: photosensitization and photomodification. In a photomodification mechanism, UV causes structural changes of the chemical in water to form a more toxic chemical (Huang et al., 1993). The postulated mechanism of photosensitization is absorption of UV energy by the chemical in the organism causing subsequent tissue injury; no change in chemical structure occurs (Landrum et al., 1987). Light energy excites the photosensitizing chemical to a triplet energy state, which is then transferred to molecules within the cell or cell membrane, possibly generating reactive oxygen

species (Landrum et al., 1987). Energy transfer can occur when the excited state energy of the photosensitizing chemical (e.g., PAH) exceeds that of an acceptor molecule such as oxygen which has a lower excited energy state (Zepp, 1980). The reactive oxygen species (e.g., superoxide anion) can then cause tissue damage that would not be observed in the absence of UV light. Consistent with a reactive mechanism, Weinstein et al. (1997) reported that the mode of action of the photoenhanced toxicity of fluoranthene (a PAH component of oil) was disruption of mucosal cell membrane function and integrity in the gill. Twelve to 48 hours of exposure of juvenile fathead minnows to 6 to 12 $\mu g/L$ fluoranthene and UV (UVA: 49 $\mu W/cm^2$; UVB: 4.2 $\mu W/cm^2$) resulted in respiratory stress due to decreased oxygen diffusion capacity of the gill caused by tissue damage (Weinstein et al., 1997). Non-phototoxic PAHs appear to cause acute toxicity through a narcosis mechanism (e.g., anesthesia) rather than tissue damage (Barron et al., 1997).

Oil and components of oil appear to act through a photosensitization mechanism, rather than through structural modification of chemical in the water column. Research on specific PAH components of oil (e.g., anthracene) has demonstrated that accumulated chemical residues may have no toxicity in the absence of UV, but exposure to sunlight can cause 100% mortality (Landrum et al., 1987). For example, fish exposed to anthracene in the dark exhibited 100% mortality at 36 hours of sunlight exposure. Fish allowed to depurate (loss of chemical residues) anthracene for 48 hour in the dark exhibited 50% mortality following sunlight exposure, and fish allowed to depurate in the dark for 144 hours showed no mortality following sunlight exposure. These data indicate that the photoenhanced toxicity of anthracene was directly related to the tissue levels of anthracene in the fish, although tissue residues were not reported (Landrum et al., 1987).

Additional support for a photosensitization mechanism of PAH photoenhanced toxicity in aquatic organisms includes the study of Ankley et al. (1997) investigating the relationship between UV exposure and chemical concentrations in the aquatic worm *Lumbriculus.* Worms were exposed to aqueous concentrations of anthracene, pyrene, or fluoranthene for 96 hours, followed by a 96 hour holding period in clean water at three UV intensities. The lethality of each PAH was a function of the product of UV intensity and accumulated tissue residues of each PAH (Ankley et al., 1997).

Figure 1. Schematic representation of the mechanisms of photoenhanced toxicity in aquatic organisms: photosensitization and photomodification.



Little et al. (2000) have reported experimental data for an environmentally weathered oil (middle distillate low in total PAHs; Barron et al., 1999) showing that the photoenhanced toxicity of oil occurs through a photosensitization mechanism (Fig. 2). Fish exposed to water accommodated fractions (WAF) (1.5 mg/L TPH) for 48 hours in the dark exhibited no mortality, but 36% mortality occurred with 48 hours of exposure to simulated sunlight (UVA: 250 $\mu W/cm^2$; UVB: 4 $\mu W/cm^2$) (Fig. 2, top panel). There was only 3% mortality of fish exposed to oil without UV. UV exposed oil was not toxic to fish tested under fluorescent light (Fig. 2, bottom panel), indicating photomodification was not responsible for toxicity. Although tissue residues were not measured, these data show that photoenhanced toxicity was observed in fish that were allowed to first bioaccumulate oil, and then received a UV exposure (photosensitization mechanism). Thus in this report, the discussion of photoenhanced toxicity is focused on a photosensitization mechanism.

The phototoxic components of oil appear to be restricted to specific polycyclic aromatic compounds with three to five benzene or substituted benzene rings (Mekenyan et al., 1994; Kosian et al., 1998). Photoenhanced toxicity of other classes of chemicals can occur, but this appears to be photomodification of chemicals in water (i.e., formation of more toxic and reactive chemicals) rather than activation of tissue residues of photosensitizing chemicals. Figure 3 shows the photoenhanced toxicity of several PAHs and heterocycles that are components of oil. Heterocycles are PAHs that contain an oxygen, sulfur, or nitrogen in place of a carbon (e.g., acridine in Fig. 3). A value of one in Figure 3 indicates no increase in toxicity of the chemical following UV exposure, whereas a value of 100 indicates a 100 fold increase in toxicity.

Research has only recently begun identifying the phototoxic components of oil. For example, dibenzothiophenes, important components of petroleum, have only recently been indicated as phototoxic (Kosian et al, 1998). Sediment pore water contaminated with oil refinery discharges was phototoxic to *Lumbriculus* in laboratory bioassays (18 hr pore water exposure in the dark, followed by a 4.5 hr UV exposure in clean water; UVA: 832 $\mu W/cm^2$; UVB: 127 $\mu W/cm^2$). The pore water was then separated into fractions containing different chemical constituents, and each fraction was tested for photoenhanced toxicity. Dibenzothiophenes were tentatively identified as a phototoxic constituent of pore water fractions exhibiting photoenhanced toxicity based on mass spectral identification and electronic features that confer phototoxicity (Kosian et al., 1998).

Aromatic ring conjugation is an important determinant of the phototoxicity of a polycyclic aromatic compounds. For example, the three ring aromatic compound anthracene exhibits a several order of magnitude increase in toxicity in the presence of UV, while its three ring homolog phenanthrene does not (Mekenyan et al., 1994).

Figure 2. Results of photosensitization and photomodification tests of an environmentally weathered oil. Top panel: fish were first allowed to bioaccumulate oil in the dark, then exposed to UV light in clean water; Bottom panel: oil was first irradiated with UV, then fish were exposed to oil without UV. Source: Little et al. (2000)

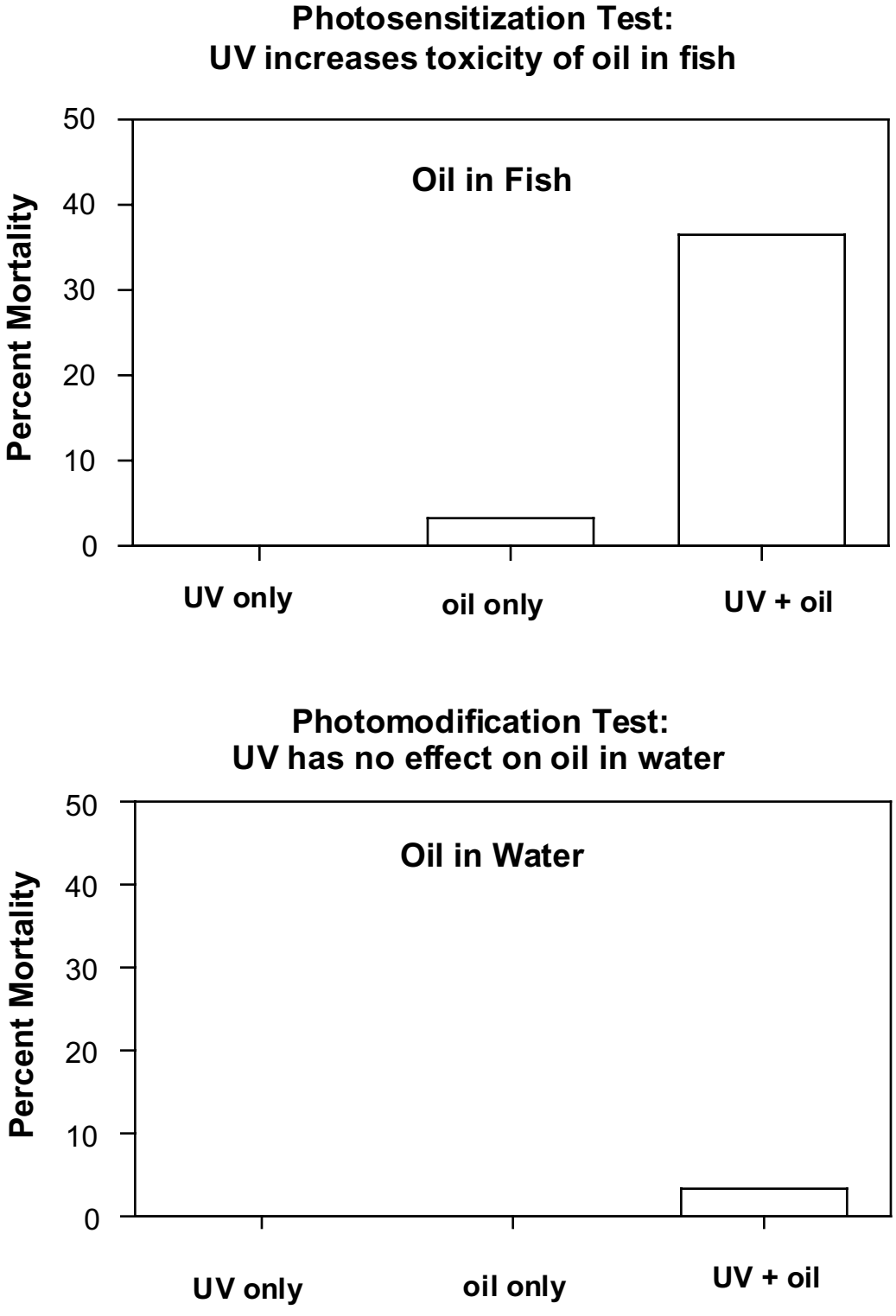
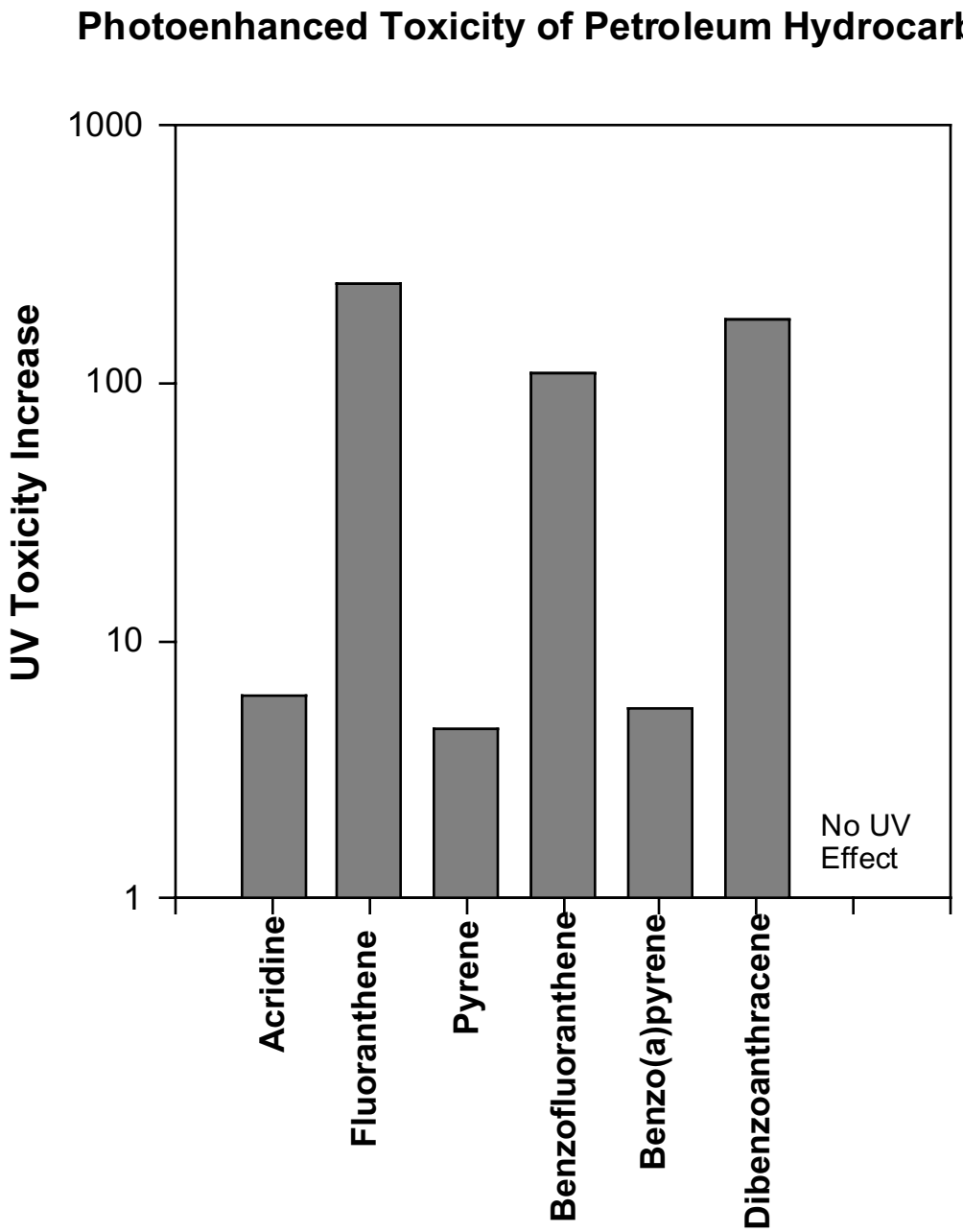


Figure 3. Photoenhanced toxicity of polycyclic aromatic compounds that occur in petroleum. *Daphnia magna* were exposed for 24 hours to individual PAHs , followed by a 2 hour exposure to UV (370 µW/cm^2, UVA +UVB). Source: Wernersson and Dave (1997).



QSAR modeling of the effect of substituents on the electronic structure of PAHs indicates that alkylation (i.e., addition of carbon groups) will have little effect on the photoenhanced toxicity of PAHs (Veith et al., 1995). This indicates that the more abundant alkyl PAHs present in petroleum will have similar photoenhanced toxicity as the parent chemical (non-alkylated homolog).

Consideration of Photoenhanced Toxicity in Oil Spill Response

Photoenhanced toxicity should be considered in oil spill response because petroleum in water is phototoxic to aquatic organisms. Petroleum products and weathered oil exhibit a 2 to greater than 100 fold increase in toxicity under simulated natural sunlight (Pelletier et al., 1997; Calfee et al., 1999). Crude oils, fuel oil #2, and Bunker C were phototoxic to shellfish larvae (Pelletier et al., 1997). In the oil product studies, WAF was prepared at 0.1 g oil/L water and tested for 48 hours under UV (UVA: 397 $\mu W/cm^2$; UVB: 134 $\mu W/cm^2$) or fluorescent light (UVA: 9.7 $\mu W/cm^2$; UVB: 3.4 $\mu W/cm^2$) (Pelletier et al., 1997). An environmentally weathered oil low in known phototoxic PAHs was 2 to 12 times more toxic to zooplankton (*Ceriodaphnia*), larval shrimp (*Mysidopsis*), larval fish (*Menidia*), and amphibian tadpoles (*Rana*) (Calfee et al., 1999; Cleveland et al., 2000; Little et al., 1999; Little et al., 2000). In these weathered oil experiments, organisms were exposed to WAF for 7 days under simulated natural sunlight (e.g., UVA: 60 to 75 $\mu W/cm^2$, 14 hr photoperiod; UVB: 0.1 to 0.3 $\mu W/cm^2$, 4 hr photoperiod) or fluorescent lighting (e.g., 14 hr photoperiod of UVA: 3 $\mu W/cm^2$ and UVB: 0.002 $\mu W/cm^2$). Several petroleum concentrations were evaluated, ranging from 0 (control) to 3 mg/L of total petroleum hydrocarbons.

Estimates of the temporal and spatial extent of injury to aquatic organisms from an oil spill may be greater if photoenhanced toxicity is considered. Existing laboratory toxicity thresholds for oil have been derived under fluorescent lighting (minimal UV) (e.g., Markarian et al., 1995), which may substantially underestimate the toxicity of oil in the environment. For example, fluorescent lighting (e.g., UVA: 3 $\mu W/cm^2$; UVB: 0.002 $\mu W/cm^2$) has UV levels 25 to 50 times below the light intensities necessary to cause photoenhanced toxicity (e.g., UVA: 60 $\mu W/cm^2$; UVB: 0.1 $\mu W/cm^2$) (Little et al., 2000). Photoenhanced toxicity thresholds may be lower for both acute and chronic effects of phototoxic chemicals (Holst and Giesy, 1989).

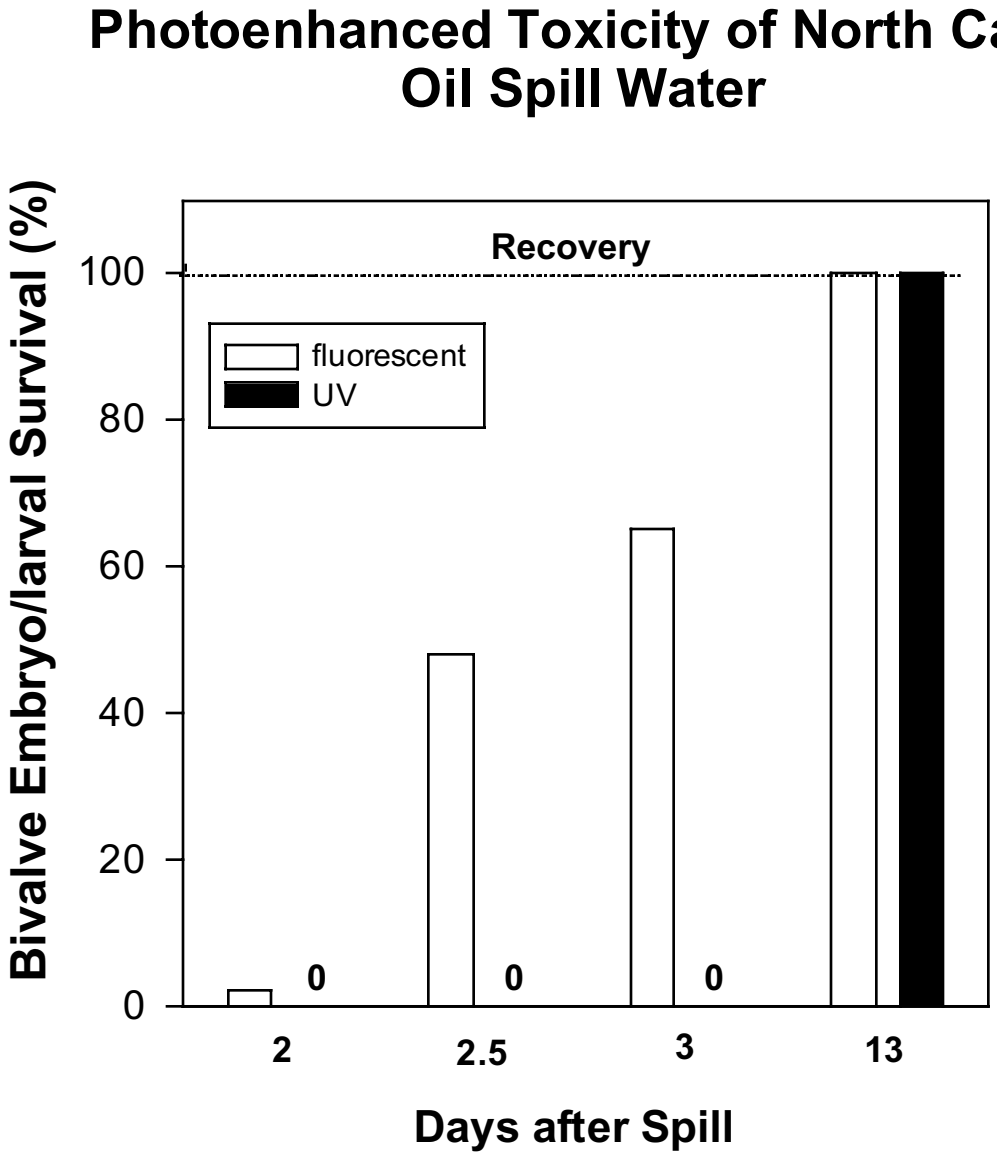
Ho et al. (1999) evaluated the photoenhanced toxicity of spill water collected between 2 and 13 days after the Rode Island North Cape spill of fuel oil #2. In these experiments, bivalve embryos were exposed to spill water for 48 hours under UV (UVA: 300 $\mu W/cm^2$; UVB: 110 $\mu W/cm^2$, 16 hr photoperiod) or fluorescent lighting (UVA: 8 $\mu W/cm^2$; UVB: 1.9 $\mu W/cm^2$). Under fluorescent lighting, there was 60% survival of

shellfish embryos exposed to spill water collected three days after the spill, indicating partial recovery (loss of toxicity) of the water column (Fig. 4) (Ho et al., 1999). In contrast, there was no survival of embryos that were exposed under UV, indicating that the spill water remained acutely toxic three days after the spill (Fig. 4). Samples collected 13 days after the spill were not toxic to embryos in either light treatment. These data suggest that estimates of the extent of toxic concentrations of petroleum in spill water based on tests performed under fluorescent lighting may underestimate actual injuries to aquatic organisms.

Oiled and PAH contaminated sediments may also exhibit photoenhanced toxicity (Kosian et al., 1998). For example, sediments containing 16 ppm total PAHs caused over 40% mortality of amphipods under UV, and less than 10% mortality without UV (Jones et al., 1999). In these experiments, amphipods were exposed to contaminated sediments for 10 days under UV (UVA: 535 $\mu W/cm^2$; UVB: 71 $\mu W/cm^2$) or fluorescent lighting. At low PAH concentrations (0.5 ppm), there was less than 10% mortality of amphipods in either light treatment. These data indicate that the persistence of oil and PAH toxicity in the environment may be underestimated if photoenhanced toxicity is not considered.

Arfsten et al. (1996) concluded that photoenhanced toxicity should be considered in assessing the ecological risks of phototoxic PAHs. This concern for photoenhanced toxicity is also applicable to spilled oil because oil and specific components of oil can be phototoxic (Pelletier et al., 1997; Little et al., 2000). As discussed above, specific 3 to 5 ring PAHs and heterocyclic aromatics are likely the phototoxic components of oil, but research in this area is still ongoing. Anthracene and fluoranthene are highly phototoxic, whereas naphthalene and phenanthrene are not. Photoenhanced toxicity should be considered in the selection of spill counter measures and oil recovery operations. Oil spill responses will influence the temporal and spatial extent of petroleum exposure to aquatic organisms, and the subsequent bioaccumulation of phototoxic components of oil. Also, as discussed below, the choice of spill counter measures may also influence the UV exposure to aquatic organisms.

Figure 4. Toxicity of oil spill water to aquatic organisms determined under UV and fluorescent lighting. Water was collected 2 to 13 days following the North Cape Rode Island spill of fuel oil #2 and tested in 48 hr laboratory bioassays. Source: Ho et al. (1999)



Determinants of Photoenhanced Toxicity

Photoenhanced toxicity is determined by both chemical and light exposure, which can be expressed as a reciprocity relationship:

Phototoxicity = f (chemical dose x light dose)

This is an idealized model that describes the degree of photoenhanced toxicity as a function (f) of both the concentration of phototoxic chemical in an aquatic organism (chemical dose) and the UV exposure of the organisms (light dose). The relationship shows that photoenhanced toxicity may occur at high tissue levels of phototoxic chemicals and low UV exposure, or low tissue levels and high UV exposure. Evidence of this reciprocity relationship includes the time-dependent mortality of an aquatic worm (*Lumbriculus*) exposed to three phototoxic PAHs. PAH toxicity was dependent on both UV intensity and PAH concentrations in tissue (Ankley et al., 1997). In these experiments, worms were exposed to aqueous concentrations of anthracene, pyrene, or fluoranthene for 96 hours without UV, followed by a 96 hour holding period in clean water at three UV light intensities. The lethality of each PAH was a function of the product of UV light intensity and accumulated tissue residues of each PAH (Ankley et al., 1997).

The degree of photoenhanced toxicity will be dependent on the potency and concentrations of accumulated phototoxic chemicals, and the intensity and spectra of UV radiation reaching the tissue bound chemical in the aquatic organism. To date, photoenhanced toxicity has primarily been demonstrated in small translucent organisms, such as early life stages (e.g., embryos and larvae) of shellfish, crustaceans, and fish. These organisms may lack pigment and have an epidermis of only a few layers, allowing UV to penetrate deeply (Hunter et al., 1980). Photoenhanced toxicity has been shown to occur at the UV wavelengths and intensities that occur in the water column of aquatic environments. For example, Calfee et al. (1999), Cleveland (2000), and Little et al. (2000) have shown that photoenhanced toxicity can occur at only 1% of the surface level of UV (e.g., UVA: 60 to 75 $\mu W/cm^2$; UVB: 0.1 to 0.3 $\mu W/cm^2$) (Barron et al., 2000). Laboratory studies demonstrating photoenhanced toxicity have used simultaneous UVB (280 to 320 nm) and UVA (320 to 400 nm) exposures, thus the specific wavelengths of UV that cause photoenhanced toxicity have not been identified.

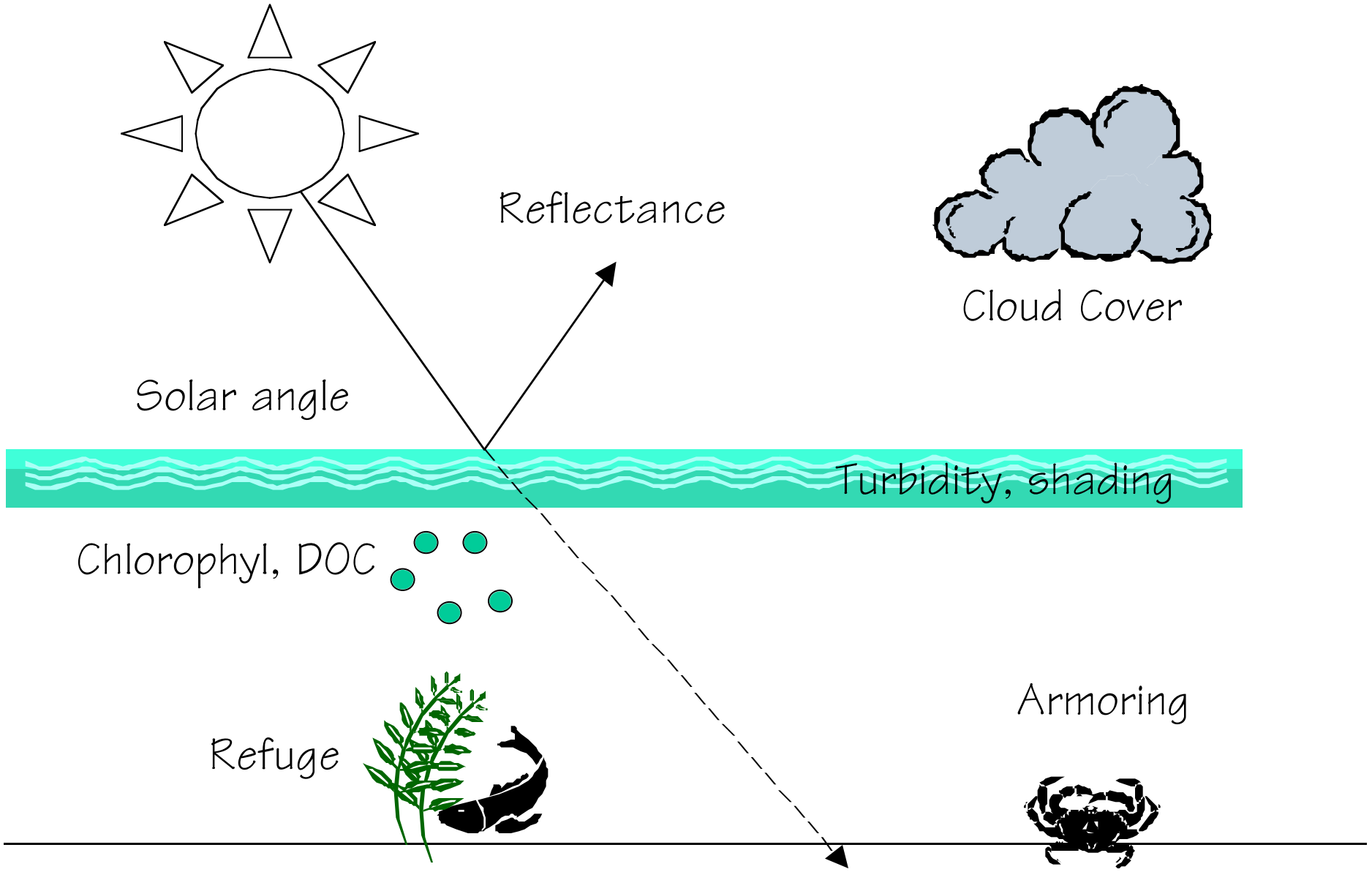
An important determinant of photoenhanced toxicity is the bioaccumulation of phototoxic PAHs. Factors affecting chemical exposure to aquatic organisms will include the composition and environmental fate of the spilled oil, the rate and extent of bioaccumulation of the phototoxic components of oil, and their biological persistence. PAHs can rapidly accumulate in aquatic organisms, but are eliminated from tissues relatively quickly once exposure ceases (Barron, 1995). Chemical uptake in aquatic

organisms can occur from both dietary intake (e.g., contaminated food items) or absorption across the gills and other surfaces (Barron, 1995). PAHs and other components can have high affinity to particulates and dissolved organic carbon, which lowers their bioavailability and accumulation in aquatic organisms (Barron, 1990). Photoenhanced toxicity of PAHs can also be reduced in the presence of high turbidity, due to decreased UV penetration and reduced bioavailability of chemicals bound to particulates (Ireland et al., 1996).

Determinants of UV exposure to aquatic organisms are depicted in Figure 5, and include factors affecting light exposure at the water surface such as photoperiod, sun angle, reflectance, cloud cover, and ozone and aerosol thickness (Baker et al., 1980). Decreasing light penetration in the water column is termed attenuation, and is affected by turbidity and shading (e.g., surface foam), phytoplankton concentrations (e.g., chlorophyl-a levels), and dissolved organic carbon (DOC). For example, phytoplankton can absorb and scatter UV (Yentsch and Yentsch, 1980). Biological factors affecting UV exposure include refuges (e.g., reefs, vegetation, sediments) and armoring (e.g., exoskeleton of crustaceans). Organisms may also exhibit avoidance or attraction to light that will influence their exposure to UV (Barcelo, 1980). Organism exposure to UV may also be influenced by the organisms vertical distribution in the water column, which can be affected by ocean currents and mixing (Hunter et al., 1980; Kullenberg, 1980). Oil slicks and dispersed oil may also alter UV exposure to aquatic organisms by preventing UV penetration into the water column (e.g., below an oil slick) or by refracting or reflecting light (e.g., attenuation of light through dispersed oil).

The degree of photoenhanced toxicity to aquatic organisms will be directly related to both the intensity and the spectral distribution of incident light within the water column, rather than at the water surface of aquatic habitats (Landrum et al., 1987; Ankley et al., 1995; Arfsten et al., 1996). In general, penetration of the water column by shorter wavelengths (e.g., UVB, 280-320 nm) is much lower than penetration by longer wavelengths (UVA, 320-400 nm; visible, 400-700 nm) (Lean, 1998). The decrease in light intensity and the alteration of the spectral distribution of light with increasing water depth are determined by the attenuating characteristics of aquatic habitats (Kirk, 1994a,b). For example, the depth of water required to remove 99% of UVB ranges from 20 m in clear and colorous ocean water, to less than 3 meters in most coastal waters, to a few cm in brown humic lakes and rivers (Kullenberg, 1980; Kirk, 1994b). One percent of the surface UVB penetrated to 10 meters in waters off Iceland (Calkins and Thordardottir, 1980).

Figure 5. Factors affecting UV exposure to aquatic organisms.



Water quality and physical conditions that reduce light penetration can reduce photoenhanced toxicity in aquatic habitats (Ireland et al., 1996). For example, UV intensity at the surface can be reduced 50 to 90% even at shallow depths (i.e., 10 cm). At a depth of 1 meter, UV at the water surface can be reduced greater than 99%. However even at these levels of attenuation, UV can be sufficient to induce photoenhanced toxicity (Little et al., 2000).

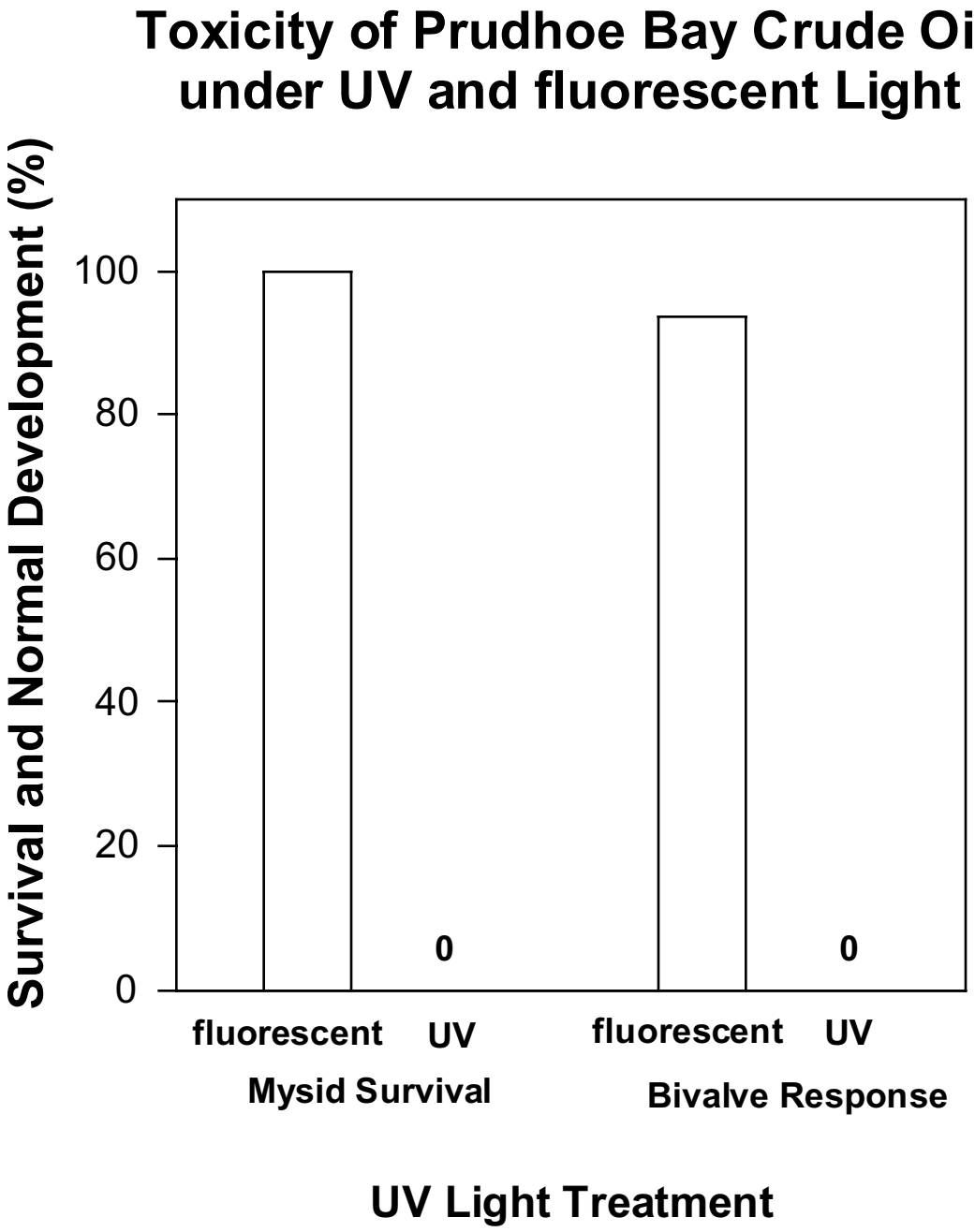
Potential for Photoenhanced Toxicity in Prince William Sound and Gulf of Alaska Waters

Currently there is no research evaluating the potential for photoenhanced toxicity of spilled oil in Prince William Sound and associated Gulf of Alaska waters. Based on the current understanding of photoenhanced toxicity, multiple factors are expected to influence photoenhanced toxicity in Alaska waters, including the extent and persistence of bioaccumulated components of oil that are phototoxic and the UV exposure to aquatic organisms.

North Slope crude oil has been shown to be phototoxic in laboratory tests with WAF of Prudhoe Bay crude oil (PBC) (Pelletier et al., 1997). WAF was prepared at 0.1 g oil/L water and tested for 48 hours under a 16 hr photoperiod with either UV (UVA: 397 $\mu W/cm^2$; UVB: 134 $\mu W/cm^2$) or fluorescent lighting (UVA: 9.7 $\mu W/cm^2$; UVB: 3.4 $\mu W/cm^2$). Figure 6 shows that the WAF of Prudhoe Bay crude oil (PBC) caused 100% mortality of larval shrimp and shellfish embryos with UV exposure, whereas no mortality occurred under fluorescent lighting.

Recent research has shown that water accommodated fractions of weathered oil can be phototoxic at total PAH concentrations less than 1 µg/L, even at low levels of UV (Barron et al., 2000; Little et al., 2000). For example, WAF prepared with an environmentally weathered oil (middle distillate oil low in known phototoxic PAHs; Barron et al., 1999) was 2 to 12 times more toxic to *Ceriodaphnia*, and larval mysid shrimp and fish (*Menidia berylina*) under UV (UVA: 60 to 75 u $\mu W/cm^2$; UVB: 0.1 to 0.3 $\mu W/cm^2$) than under fluorescent light (UVA: 3 $\mu W/cm^2$; UVB: 0.002 $\mu W/cm^2$). Photoenhanced toxicity occurred under low levels of simulated natural sunlight occurring in aquatic habitats (4 hours of UVB/day, 14 hours of UVA and visible light/day) at total PAH concentrations less than 1 µg/L (Calfee et al., 1999; Barron et al., 2000; Cleveland et al., 1999; Little et al., 2000). In comparison, water column concentrations of total PAHs following the Exxon Valdez spill ranged from less than 1 to 5 µg/l total PAHs (Neff and Stubblefield, 1995). The individual PAHs observed in Exxon Valdez spill water included phototoxic PAHs (Neff and Stubblefield, 1995). Pink salmon embryos exposed to Exxon Valdez oiled gravel have also been shown to accumulate phototoxic PAHs (Heintz et al., 1999). These data indicate that North Slope crude can be phototoxic and suggest that

Figure 6. Photoenhanced toxicity of Alaska North Slope crude. Mysid shrimp and bivalve embryos were exposed to WAF prepared at 0.1 g/L for 48 hours under a 16 hr photoperiod with UV or fluorescent lighting. Source: Pelletier et al. (1997)



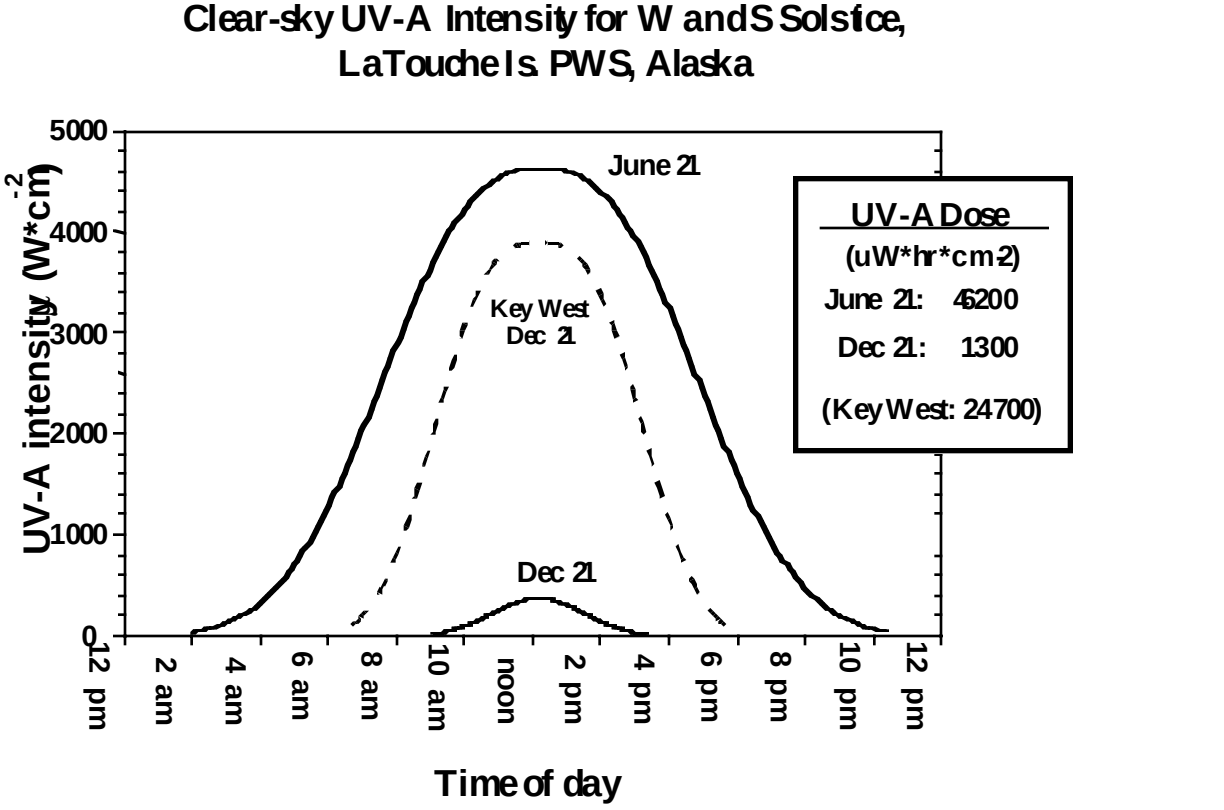
aquatic organisms may be exposed to phototoxic components of spilled oil in sediment and the water column in Prince William Sound.

UV exposure to aquatic organisms is expected to vary dramatically in Prince William Sound and associated Gulf of Alaska waters because of the diversity of organisms, varying cloud cover and water clarity, and extremes in day length. The UV irradiance data necessary to evaluate the potential photoenhanced toxicity in Prince William Sound were not available for this report. These data would include seasonal and annual estimates of UVA and/or UVB intensity or attenuation in the water column in both inshore and offshore areas. In the absence of site-specific data, UVA intensity at the water surface was estimated using the SBDART radiative transfer model under a scenario of a clear day with average ozone depth and aerosol composition (Ricchiazzi et al., 1999). Figure 7 shows estimated intensity of UVA in Prince William Sound at the winter and summer solstice, and compares these values to UVA estimates for Key West, Florida at the winter solstice (to allow comparison of UVA intensity at a lower latitude).

UV in the water column can be substantially lower than surface level intensity and will vary with environmental conditions. In clear water, UV can penetrate to over 8 meters (Landrum et al., 1987), or to only 0.5 meters in waters with high turbidity, DOC, or chlorophyl (Barron et al., 2000). Alexander and Chapman (1980) report that in mid-November to early February the water column is generally well mixed and clear in Port Valdez, and the one percent level of surface sunlight reaches to a 25 m depth. Around the summer solstice, heavy freshwater runoff carrying large quantities of suspended solids and phytoplankton blooms reduce the one percent penetration level to as shallow as 6 meters (Alexander and Chapman, 1980). This information indicates that UV penetration can be substantial in Prince William Sound and Gulf of Alaska waters, but will be dependent on local and seasonal conditions. Additionally, the extent of UV penetration will occur within the portion of the water column in contact with spilled oil. For example, Exxon Valdez oil was believed to be mixed to 5 meters. As discussed above, other factors that can influence UV exposure to aquatic organisms include the vertical distribution of susceptible life stages in the water column, and the attenuation or blockage of light by oil slicks or dispersed oil.

Research on the levels of UV necessary to photoactivate North Slope crude are not available (Pelletier et al., 1997, only evaluated one level of UV). Barron and coworkers (2000; Calfee et al., 1999; Cleveland et al., 2000; Little et al., 2000) have shown WAF of an environmentally weathered oil low in PAHs can exhibit photoenhanced toxicity at 0.3 $\mu W/cm^2$ UVB and 75 $\mu W/cm^2$ UVA.

Fig. 7. UVA intensity at the water surface for the winter and summer solstice, LaTouche Island, Prince William Sound. Data for the winter solstice in Key West, Florida is provided to allow comparison to UVA intensity at lower latitude. Source: SBDART radiative transfer model (Ricchiazzi et al., 1999).



In the absence of site-specific data, estimated UVA levels in Prince William Sound (Figure 7) were compared to a light intensity threshold (light level necessary for photoenhanced toxicity) determined for the environmentally weathered oil (Cleveland et al., 2000). Figure 8 compares the toxicity threshold to three scenarios for UVA at the summer solstice: (1) water surface (cloudless day), (2) 10% loss of UV, and (3) 90% loss of UV. Figure 8 shows that at the summer solstice, the light intensity threshold necessary for photoenhanced toxicity was exceeded even under a scenario of a 90% reduction in UVA. Figure 9 compares the UVA photoenhanced toxicity threshold to three scenarios of UVA at the winter solstice. Figure 9 shows that the light intensity threshold was only exceeded under a scenario of a 10% reduction in UVA. These results, although only modeled values, suggest that under some circumstances there may be sufficient UV irradiance of the water column to elicit photoenhanced toxicity of spilled oil in Prince William Sound. Additional research would be needed to define the locations

and seasons with the greatest potential for photoenhanced toxicity of spilled oil.

Potential for the Photoenhanced Toxicity of Chemically Dispersed Oil

Chemical dispersants break up free product oil into small droplets (e.g., 0.01 to 50 micron) which disperse in the water column. Dispersants generally increase the total concentrations of petroleum compounds (dissolved + particulate oil in the water column), but the relative environmental hazards of chemically dispersed and non-chemically dispersed oil are uncertain and may be spill-specific (DeCola, 1999). Laboratory studies conducted under fluorescent lighting (minimal UV) have shown that dispersants can either increase, decrease, or have no effect on the toxicity of oil (DeCola, 1999). Studies evaluating the photoenhanced toxicity of chemically dispersed oil have not been performed. Whether chemical dispersants increase the risk of photoenhanced toxicity would depend on the enhancement of the bioaccumulation of the phototoxic components of oil. Chemical dispersants may also influence the UV exposure of aquatic organisms, but research comparing UV light penetration of chemically dispersed and non-chemically dispersed oil is not available. Dispersants may increase the potential UV exposure of aquatic organisms by allowing greater light penetration through the oil slick, or may reduce UV exposure via light attenuation of the dispersed oil droplets into the water column.

Wolfe et al. (1999) evaluated the bioavailability and trophic transfer of phenanthrene in WAF of PBC and chemically dispersed PBC. Phenanthrene is a three ring PAH that is not phototoxic, but has nearly identical structure as the highly phototoxic oil component anthracene. Phenanthrene was used as a model chemical to evaluate mid-range PAHs present in oil. PBC was dispersed with Corexit^{R} 9527, which is the primary dispersant stockpiled for oil spill response in Prince William Sound. Corexit^{R} 9527 contains nonionic and anionic surfactants and a solvent (ethylene glycol monobutyl ether) (Wolfe et al., 1999). Corexit^{R} 9527 elevated the concentrations of phenanthrene in both the aqueous phase of PBC and in algae exposed to dispersed PBC. In contrast, there was no substantial differences in phenanthrene concentrations in a predatory rotifer species exposed to chemically dispersed or non-chemically dispersed PBC. Wolfe et al. (1999) speculated that trophic transfer of phenanthrene from rotifers to higher trophic levels would not likely be enhanced by the use of dispersants.

Figure 8. Comparison of a UVA photoenhanced toxicity threshold to three scenarios for UVA at the

summer solstice in Prince William Sound: water surface (cloudless day); 10% loss of UV; 90% loss of UV. UVA at the surface was determined from Figure 7 and the UVA toxicity threshold was determined from Cleveland et al. (2000). See text for details.

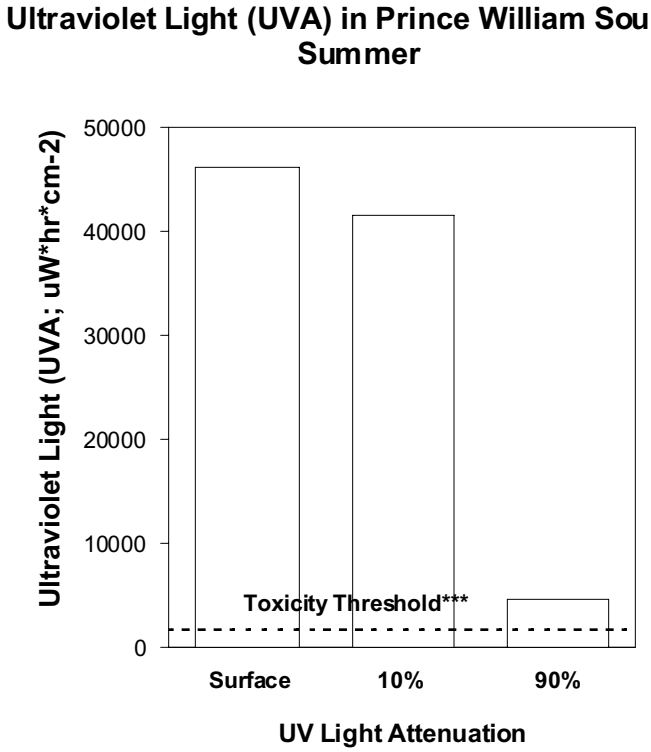
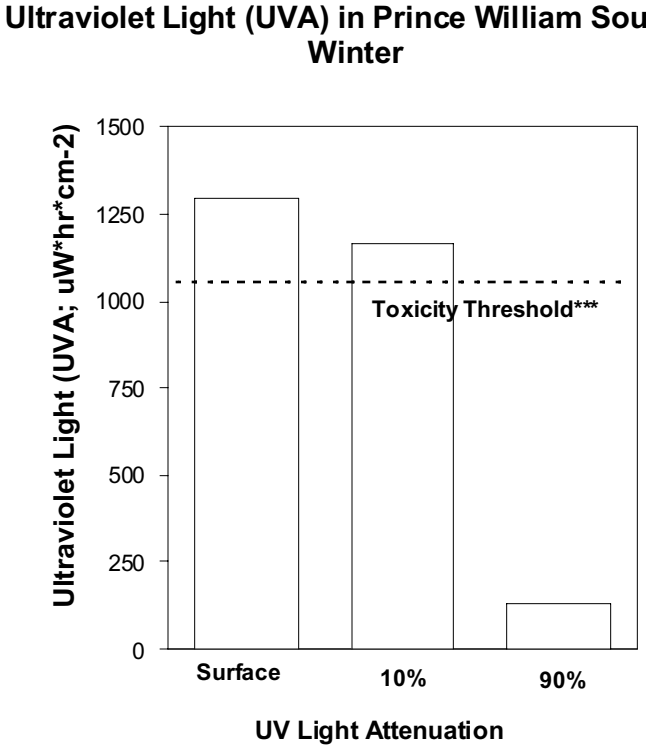


Figure 9. Comparison of a UVA photoenhanced toxicity threshold to three scenarios for UVA at the winter solstice in Prince William Sound: water surface (cloudless day); 10% loss of UV; 90% loss of UV. UVA at the surface was determined from Figure 7 and the UVA toxicity threshold was determined from Cleveland et al. (2000). See text for details.



Shrimp exposed to chemically dispersed PBC had substantially higher tissue

concentrations of saturated hydrocarbons (alkanes), but similar concentrations of naphthalenes and phenanthrenes as shrimp exposed to non-chemically dispersed PBC (Anderson, 1985). Singer et al. (1999) reported higher concentrations of larger PAHs (including phototoxic compounds) in chemical dispersions of PBC compared to WAF prepared from PBC. However, particulate and dissolved compounds could not be distinguished, and the accumulation in aquatic organisms was not evaluated (Singer et al., 1999). These studies indicate that chemically dispersed oil is likely to exhibit photoenhanced toxicity because phototoxic PAHs are dispersed in the water column and are likely to be bioavailable to aquatic organisms. However, it is unknown if chemically dispersed oil would be more or less phototoxic than non-chemically dispersed oil. Research is needed to evaluate the relative hazards of chemically and non-chemically dispersed oil, and the ecological risks of dispersed oil may be spill specific.

Conclusions and Research Needs

Overall, this evaluation indicates there is potential for photoenhanced toxicity of spilled oil in Prince William Sound and associated Gulf of Alaska waters. North Slope crude is known to be phototoxic, phototoxic PAHs were present in Exxon Valdez spill water, and phototoxic PAHs accumulate in aquatic organisms exposed to weathered Exxon Valdez oil. Additionally, under some circumstances sufficient UV radiation may penetrate the water column to elicit photoenhanced toxicity in aquatic organisms. The photoenhanced toxicity of spilled oil may pose the greatest hazard to translucent planktonic organisms and larval stages that inhabit the photic zone of the water column and intertidal areas. These organisms may lack pigment and have an epidermis of only a few layers, allowing UV to penetrate deeply (Hunter et al., 1980).

Chemically dispersed oil is likely to exhibit photoenhanced toxicity because phototoxic PAHs are dispersed in the water column and are likely to be bioavailable to aquatic organisms. However, no research has evaluated the photoenhanced toxicity of chemically dispersed oil, and it is unknown if chemically dispersed oil would be more or less phototoxic than non-chemically dispersed oil. Additional research is needed to evaluate the risks of photoenhanced toxicity of spilled oil and chemically dispersed oil. Existing field studies of oil spills and use of chemical dispersants could be reviewed to determine if the observed toxicity was substantially greater than was expected based on laboratory toxicity thresholds (performed under fluorescent lighting). This evaluation could indicate whether photoenhanced toxicity was responsible for higher than expected mortality to aquatic organisms.

Substantial uncertainties exist in the degree and extent of phototoxicity that may occur in Alaska waters. Research should be aimed at (1) quantifying the likelihood of photoenhanced toxicity of both chemically dispersed and non-chemically dispersed oil, (2) the locations and times of year where atmospheric and oceanic conditions may allow sufficient UV exposures and oil bioaccumulation in aquatic organisms, and (3) the

habitats, aquatic organisms, and life stages most at risk. Specific research objectives may include determining the following:

- **Oil and UV thresholds for photoenhanced toxicity**. Determine these thresholds using laboratory exposures of selected species of aquatic organisms to WAF prepared from North Slope crude. Test multiple concentrations of North Slope Crude and UV to establish levels necessary for photoenhanced toxicity. Measure the concentrations of North Slope crude in water and possibly tissues, and determine the wavelengths important in photoenhanced toxicity (e.g., UVA vs UVB).

- **Comparative photoenhanced toxicity of chemically dispersed and non-chemically dispersed oil**. Determine the photoenhanced toxicity of oil dispersed with Corexit^{R} 9527 in laboratory bioassays, and compare the results to toxicity thresholds established for WAF of North Slope crude tested under UV and fluorescent lighting.

- **Locations, depths, and times of year with sufficient UV exposure**. Compile existing data if available or perform measurements of UV at various depths in Prince William Sound and associated Gulf of Alaska waters. Develop seasonal maps of both inshore and offshore areas that have UV exposures above photoenhanced toxicity thresholds. Compute the depths in these locations that exceed UV thresholds.

- **Species and life stages at greatest risks**. Evaluate the vertical distribution and seasonal occurrence of planktonic and intertidal organisms and life stages that exist in the photic zone of Prince William Sound and associated Gulf of Alaska waters. Determine the species most a risk because of their occurrence in habitats with UV levels above phototoxicity thresholds.

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