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INTRODUCTION

Laboratory bioassays have been used to describe or quantitate the ability of crude and refined oils to kill marine life. The principal objective of earlier studies was to estimate oil concentrations that were definitely lethal to marine animals, and in some cases there have been attempts to predict environmental effects on the basis of these studies. More recently, two objectives of laboratory toxicity testing have emerged. First, acute toxicity studies have been used to determine and compare the toxicities of different crude and refined oils to marine life, and the toxicities of individual components of oils. In these studies, the quantity of oil or oil component necessary to kill 50% of the standard test species exposed under a constant set of conditions (median lethal concentration, LC50) is equated to the toxicity of the oil or oil component. Secondly, acute toxicity data have been used to determine and compare the sensitivities of different species or the different life stages of a given species. The goal here has been to identify species or life stages that are especially sensitive or vulnerable to oil. Here the LC50 has been used as an index of species sensitivity.

There has been a wide variety of results from these studies, and some apparent contradictions, mainly because of the absence of standard exposure and analytical methods but also because of difficulties involving quantitation of the oil used and its instability in the test waters. The amount of oil used to prepare a test solution is invariably much more than the amount of oil that enters into the test water and the composition of oil in the test water is invariably different from the composition of the parent oil. The composition and amount of oil that enters the water column is a function of many factors, such as mixing energy and duration, temperature, and salinity. Once in the water, oil components are subject to evaporation and microbial oxidation, which can appreciably alter the composition and concentration of oil constituents with time, even at low temperatures. Analytical methods for measuring the amount of oil in the test water have only recently been widely available, and all of these methods are less than ideal.

The scope of this review is limited to studies dealing with the ability of crude and refined oils to kill marine animals. Emphasis is given to the more recent quantitative studies that were not available to earlier reviewers (Evans and Rice 1973; Moore and Dryer 1974; National Academy of Science 1975). This review covers (1) the behavior of oil in water; (2) the methodology problems associated with bioassays; (3) the comparative toxicity of oil-water mixtures, oils, and components of oils; and (4) the comparative sensitivity of different life stages and species.

BEHAVIOR OF OIL IN WATER

Bioassays involving crude or refined oils are fundamentally different from bioassays involving other substances (such as heavy metals) in one major respect. Oil bioassays attempt to evaluate the effects of a mixture of many different toxic compounds together rather than a single pure compound. There is a wide spectrum of physical and biological properties associated with these various compounds which affects the rate and amount of these compounds transported into water. Once in the water, the persistence of some of the toxic compounds in oil is influenced by a variety of processes such as biodegradation, evaporation, etc.

A basic knowledge of physical processes that affect both the transport of oil into an aqueous phase and the persistence of oil transported is essential to evaluate results of bioassay tests. In this section, we discuss some of the factors that transport oil into and out of the water column. All of these processes occur simultaneously, and the relative

importance of these processes varies from one compound or class of compound to another. Because of the complexities involved, simple bioassay methods do not work well for investigating toxic effects of oil.

Factors that Affect the Quantity of Oil Transported into Water

Oils can become associated with an aqueous phase in a variety of different ways, such as emulsion, dispersion, or accommodation (Peake and Hodgson 1966); or some of the constituent compounds of an oil may dissolve, forming a true solution. The solubility of oil compounds in water varies considerably with the class of compound and is an important factor that determines the toxicity of oil-water solutions. Some hetero compounds such as pyridine are completely miscible with water. Benzene is the most soluble aromatic hydrocarbon at about 1,800 ppm in water. The solubility of other aromatic hydrocarbons decreases with increasing degree of alkyl substitution and number of aromatic rings. Aliphatic hydrocarbons are among the least soluble hydrocarbons, with solubility decreasing sharply with increasing carbon number (McAuliffe 1966, 69).

The amount of the soluble fraction of oil that enters the water phase is mainly determined by mixing energy, mixing duration, and the viscosity of the oil. Turbulence (or mixing energy) was found to have a pronounced effect on the amount of both particulate and sub-particulate oil going into the water phase (Gordon et al 1973). In similar studies at our laboratory we have been unable to detect concentrations of 10 ppb in water 1 cm beneath a slick that is gently layered on the water surface (Taylor and Karinen, This symposium). Gentle mixing of oil in seawater for 20 hr will generate water-soluble fractions with oil concentrations in seawater from about 1 to 10 ppm (Anderson et al 1974; Rice et al 1976a). Violent mixing can produce oil concentrations in seawater in the hundreds of parts per million, with much of the oil present as dispersed droplets.

The amount of time that oil and water are mixed is as important as mixing energy in determining the quantity of oil that enters the water phase. Using gentle mixing, the amount of oil that enters the water phase steadily increases for over 30 hr (Gordon et al 1973, Anderson et al 1974, Percy and Mullin 1975, Rice et al 1976a).

The viscosity of the oil also affects the amount of oil that enters the water phase, because more mixing energy is required to mix thick, viscous crude oil. We have observed that the relatively viscous Prudhoe Bay crude oil yields WSF's that are about half the concentration of those from Cock Inlet crude oil when mixed under identical conditions (Rice et al 1976a).

There are several other less well-studied factors that affect the amount of oil entering the water phase. There is evidence that polar hydrocarbon derivatives are generated from oil by photo-oxidation (Lysyj and Russell 1974). These polar hydrocarbons tend to dissolve into solution from an oil slick, which raises the total concentration of oil-derived hydrocarbons with time. In addition, pH (Kauss et al 1973) and salinity (Rice et al 1975) affect the amount of oil entering the water phase.

Changes in temperature influence the transport of oil into water because changes in temperature change the viscosity of the oil, the solubility of oil components, and the stability of emulsified or suspended oil. The viscosity of oil increases as temperature decreases, thus at low temperatures more mixing energy or time is required to transport oil into the aqueous phase. As temperature decreases, the solubility of the non-volatile oil components decreases, but the solubility of the volatile components increases. Finally, emulsions and suspensions are more stable at lower temperatures. These conflicting effects make it difficult to predict the overall effect of temperature on the amount of oil transported into the aqueous phase.

Factors Affecting the Composition of Oil in Water

The composition of oil transported into the aqueous phase is also strongly dependent on compound solubilities, mixing energy, mixing duration, and oil viscosity. The composition of oil in water may or may not be similar to the composition of the parent oil, depending on how the oil is associated with the water. When oil is mixed violently with water, many dispersed droplets having a composition similar to that of the parent oil are formed. When oil is mixed slowly, the bulk of the hydrocarbons transported into water is composed of the more soluble hydrocarbons, unlike the composition of the parent oil. For example, Bean et al (1974) found water-soluble fractions to have compositions quite unlike the parent oil. They found increases in IR absorption at 3,000 to 3,100 cm^{-1} for WSF's, indicating significant increases in the relative concentration of aromatic hydrocarbons in the WSF.

The AEF is the ratio of the concentration of aromatic compounds to n-paraffins in the oil-water mixture, divided by the ratio of the concentration of aromatic compounds to the n-paraffins in the parent oil. A dispersion from a turbulent mix will result in an AEF of 1-3, indicating that the composition of oil in water is about the same as the parent oil. In contrast, the AEF will be higher in oil-water solutions prepared with less turbulence. Aromatic enrichment factors of 10-125 and similar magnitude have been reported for WSF's prepared with slow, gentle mixing of Kuwait, Prudhoe Bay, and Cook Inlet crude oils (Anderson et al 1974, Short et al 1975, Rice et al 1976a).

In addition to solubility, mixing energy, mixing duration, and oil viscosity, there are undoubtedly other factors that affect the composition of oil in water. For example, the solubility of many compounds is influenced by pH (Kauss et al 1973), salinity, and temperature. Removal of selected hydrocarbons from solution by biodegradation, evaporation, photochemical oxidation, etc. will change the composition of oil in water.

The fact that the amount and composition of oil that is transported into distilled water or seawater is strongly dependent on the method used to prepare the oil-water mixture, emphasizes the need for analytically determining the amount of oil actually transported into the aqueous phase. There have been many studies of static bioassays that report only the volume of oil used to prepare the oil-water test mixture. The concentrations of oil that the test species were actually exposed to in these studies are almost completely unrelated to the amount of oil used to prepare the test solutions, so that these studies are of limited value.

Factors Affecting the Persistence of Oil in Water

After the oil has been transported into the water, several factors cause hydrocarbons to be lost, resulting in changes in both concentration and composition of the oil solution or dispersion. Cheatham et al (In prep.) demonstrated that the losses of total aromatics from WSF's of crude oil were significant, and that the rate of loss was less at low temperatures (Fig. 1).

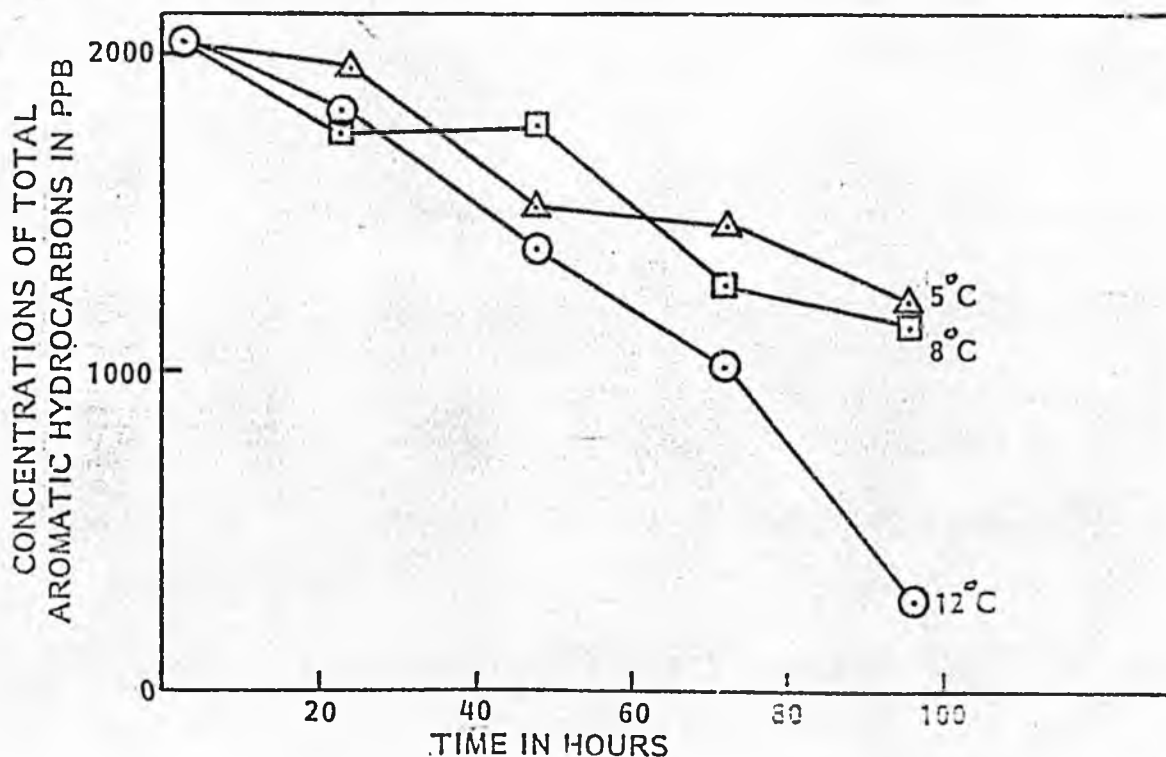


Fig. 1. Concentration of total aromatic hydrocarbons from water-soluble fractions of Cook Inlet crude oil measured by gas chromatography at 24-hr intervals. Solutions were non-aerated, and held at either 5°, 8°, or 12° C (from Cheatham et al In press).

Both paraffinic and aromatic hydrocarbons are susceptible to microbial oxidation, although several studies (Kator et al 1971) have indicated that paraffins are oxidized by microbes more easily than aromatic hydrocarbons. Further, dinuclear aromatic hydrocarbons are lost from solutions primarily by biodegradation, while mononuclear aromatics are lost primarily by evaporation; both processes occur at faster rates at higher temperatures (Cheatham et al In prep.).

Chemical and photo-oxidation can also change the concentration and composition of oil in water. Aromatic hydrocarbons in particular are susceptible to photo-oxidation. Finally, significant quantities of oil can be separated from the water when dispersed droplets coalesce into larger droplets and form a layer of oil at the surface.

METHODOLOGY PROBLEMS ASSOCIATED WITH BIOASSAYS

Since oil-water solutions are so complex and difficult to work with, researchers have had to develop various modifications of standard bioassay methods. The following discusses the general problems encountered in oil toxicity testing, and specific problems associated with comparisons of oil toxicity.

Preparation of Oil-water Mixtures

Different investigators have used a wide range of methods to prepare oil-water mixtures. For example, mixing methods range from virtually no mixing (layering oil on top of water) to very turbulent mixing for prolonged periods. As noted previously, the mixing turbulence and duration strongly affects the amount and composition of oil entering the water phase. Direct comparisons between results of different investigators cannot be made when different mixing methods have been used.

Individual investigators have tended to justify their choice of mixing methods on the basis of analogy to probable environmental situations. This strikes us as a fundamentally faulty approach. Several different mixing regimes may occur in the environment, so that it is impractical to try to simulate all of them. Acute bioassays cannot provide more than a hazy idea of what is likely to occur in the environment, nor were they intended to do so.

Methods of Exposing Animals to Oil

There are numerous practical problems in oil toxicity tests that have led to a variety of specific exposure techniques. For example, sometimes the test animals are passed directly through a surface film of oil into the oil-water mixture, and sometimes the animals are added to oil-water dispersions or WSF's. Some investigators have aerated the oil-water mixtures, which will affect results since aeration accelerates the evaporation of the toxic light aromatics. Many different designs of exposure containers have been used because the requirements of various animals differ. For example, most intertidal animals have to be trapped in the exposure container to prevent them from crawling out (controls included).

Death versus Moribundity

Since animals have a variety of structures and activity levels, dead and moribund animals may be difficult to distinguish from live animals. Usually, the lack of visible motion is used to define death, but with some slow-moving animals, death may not be distinguishable until some necrosis occurs, and this requires extending the time of the observation period. Some animals depress their metabolism and respiratory activities when stressed, and will appear dead, but can recover when placed in clean water. On the other hand, delayed mortality (deaths occurring after the exposure period has terminated), may occur in some species, such as molluscs (Swedmark 1973).

Delayed mortality leads to the problem of identifying moribund animals in contrast to dead animals. Moribund animals are severely affected and destined to die, usually within a few days. Moribund animals will die a physiological death and are not to be confused with affected animals that can live but would be "ecologically dead" because of increased vulnerability to predation or environmental stresses. If moribund animals are counted as alive at the end of the exposure (e.g. 96 hr), the calculated median lethal concentrations will be high, implying a level of tolerance that is erroneous. Crustacean larvae have been observed to be particularly slow in dying, and will exist in a moribund state for up to several days before the larvae become necrotic (Brodersen et al In press).

Each species that is tested must be observed carefully and with attention to its biology. Although there can be no universal guidelines for observing all animals in toxicity tests, post-exposure observations should be made to determine if affected animals are moribund or if they will recover.

Effects of Temperature on Oil Toxicity Tests

Temperature can affect the results of oil toxicity tests in a complex manner. Percy and Mullin (1975) found that the amphipod *Onisimus affinis* was consistently more sensitive to oil when exposed at 8°C than at 0°C, but that oil-water solutions prepared at 0°C were consistently more toxic than solutions prepared at 22°C. Korn et al (In prep.) attempted bioassays at different temperatures with pink salmon fry and shrimp exposed to crude oil WSF's, naphthalene, and toluene and found opposing results. Survival of pink salmon fry was decreased when exposed at low temperatures, whereas shrimp survival was decreased when exposed at higher temperatures. Temperature can affect toxicity measurements in two basic ways: (1) by affecting oil toxicity, and (2) by affecting animal sensitivity. Oil toxicity is increased at lower temperatures, since toxic aromatics persist longer at lower temperatures (Percy and Mullin 1975, Cheatham et al In prep.). Animal sensitivities will be affected by changes in temperature since rates of hydrocarbon uptake, metabolism, and excretion will be altered. Because the effects of temperature on oil toxicity and animal sensitivity may oppose each other, it is conceivable that the order of potency of different oils could vary with temperature. Indeed, Ottway (1971) found that the relative toxicities of 10 different crude oils changed in a complicated way between 4°C and 26°C.

Chemical Analysis

Until the 1970's, comparison of results between experimenters has been especially difficult because most experimenters have not measured the concentrations of oil present in their test solutions. In these cases, results have usually been reported as the volume of oil used to prepare the test solution. Reporting oil concentrations as the "volume added" will underestimate the toxicity of oil, because most of the oil exists as a separate phase above the water phase. As previously discussed, the amount of oil that actually enters the water phase depends strongly on the method of mixing, and the volume of oil used to prepare the test solution is an inadequate measure of the amount of oil actually in the test solution.

Several chemical methods have recently been used to determine the concentration of oil in the water phase. Infrared spectrophotometry (Anderson et al 1974, Bean et al 1974, Rice et al 1975, 1976a, 1976b), ultraviolet spectrophotometry (Brenniman et al 1976, Rice et al 1976a, Caldwell et al This symposium), fluorescence spectroscopy (Gordon et al 1973, Percy and Mullin 1975, Wells and Sprague 1976), and gas chromatography (Bean et al 1974, Benville and Korn In press, Cheatham et al In prep., Korn et al In prep.) have been used as analytical methods. These analytical methods all have the advantage of providing a measure of the amount of oil actually in the test water, as opposed to the amount of oil used to prepare the test water. Each of these analytical methods also has different practical advantages.

The infrared (IR) method of Gruenfeld (1973) is relatively simple, but is much more sensitive to paraffinic hydrocarbons than to aromatics. In addition, standards for this method are necessarily arbitrary because the composition of oil in the water phase is not the same as the parent oil. Although the IR method quantitates paraffins that presumably are not toxic, their concentrations are usually proportional to concentrations of toxic compounds.

Ultraviolet (UV) spectrophotometry and fluorescence spectroscopy are especially sensitive methods for detecting aromatic hydrocarbons, which are presumed to be the most toxic fraction of crude and fuel oils. However, the UV and fluorescence methods are completely insensitive to paraffinic hydrocarbons, and measure different aromatics with different sensitivities, making standards somewhat arbitrary.

Analysis by gas chromatography (GC) or high pressure liquid chromatography (HPLC) is more complicated than the UV and fluorescence methods, but it also provides more detailed results. Both GC and HPLC separate individual compounds from mixtures, can measure paraffins and aromatics with nearly equal sensitivity, and have standards that are not arbitrary. However, because most individual compounds are separated by these methods, the amount of data produced by a single analysis can be formidable. Analyses by GC and HPLC are relatively expensive since fewer samples can be analyzed per day.

Gas liquid chromatography (GLC) and high pressure liquid chromatography (HPLC) are probably the most appropriate analytical methods for oil toxicity investigations. Both GLC and HPLC are able to separate and measure the concentrations of individual aromatic hydrocarbons present in oil-water mixtures. As discussed later, there is general agreement that

aromatic hydrocarbons are responsible for most (if not all) of the toxicity of oil-water mixtures. Thus, using GLC or HPLC, one can attempt to correlate changes in the toxicity of oil-water mixtures with changes in chemical (i.e. aromatic) composition in order to assess the relative toxicity of each of the aromatics present (Bean et al 1974).

All of the previously described methods involve extraction of oil components into a low polarity solvent, such as hexane or methylene chloride. Since a low polarity solvent may not efficiently extract polar components of oil in the WSF, polar compounds may not be measured effectively. These polar compounds may form a significant proportion of oil-derived material in the water phase, especially if the water has been in contact with oil in the presence of light for a prolonged period (Lysyj and Russell 1974). Detailed measurement of these polar compounds is a formidable analytical problem, and the contribution made by these polar derivatives to toxicity has not been evaluated.

Statistical Analysis

Statistical analysis of bioassay data is required for meaningful comparisons between tests. Unfortunately, only a few investigators have determined 95% fiducial intervals so that statistically significant differences could be determined.

Probit analysis (Finney 1976) is the most appropriate statistical method for analyzing bioassay data because (1) it provides an accurate determination of the concentration of oil necessary to kill 50% of the test animals (LC50, LD50, or TLM), (2) it provides a measure of how much the toxicity of oil increases with increasing oil concentration, and (3) 95% fiducial intervals for LC50's can be calculated.

Other methods for calculating LC50's have been used which do not require as much data as the probit method, but the certainty of the calculated LC50 is reduced. The most commonly used of these methods is that of Doudoroff et al (1951) where the percent mortality is plotted against the log of the oil concentration, and a line is fitted to the plot. The antilog of the oil concentration corresponding to 50% mortality is the estimate of the LC50. This method will work even when exposure to oil concentrations results in only zero and complete mortality. However, the accuracy of the method in such cases can be very poor, and there is no way to estimate confidence in the LC50. In addition, an underlying assumption of Doudoroff's method is that percent mortality is a linear function of the log of the oil concentration. A detailed plot of percent mortality versus the log of the oil concentration will usually yield a sigmoid curve, if enough observations are made at oil concentrations resulting in less than 100% mortality but more than zero mortality in the test animals.

A comparatively simple method that does not have the drawbacks of Doudoroff et al's (1951) method is that of Spearman and Kärber (Finney 1976). This method requires only that oil concentrations resulting in zero and 100% mortality in the test animals be known, although it also makes efficient use of data where some concentrations have some survivors. Confidence intervals can be calculated for LC50's determined by this method, and the applicability of the method is not affected by assumptions regarding the tolerance distribution of test animals.

Neither probit analysis nor the method of Spearman and Kärber determine variability in toxicity test results from any source other than the test organisms. Specifically, we have found that variation in the toxicity of water-soluble fractions or dispersions of a given crude or refined oil mixed under identical conditions significantly exceeds the variation expected from the test organisms. Therefore, in order to obtain statistically reliable data when other sources of variation are involved, such as different WSF preparations, a bioassay should be conducted on each WSF preparation and the LC50's analyzed using Student's t-test. This procedure would then include all sources of variation.

Specific Methodology Requirements Associated with Comparing Oil Toxicities or Animal Sensitivities

At least three specific methodology requirements must be met when comparing the toxicities of different oils or comparing the sensitivities of different species.

First, a reference toxicant is needed when comparing oil toxicities. Different investigators have compared oil toxicities, but the validity of comparing results without a reference toxicant is questionable when the tests are not conducted simultaneously with the same population of test animals under identical conditions. Use of a reference toxicant will permit valid comparisons by verifying that different sub-populations of animals are equivalent in sensitivity, or that minor modifications of procedures between different researchers are insignificant.

The toxicant DSS (dodecyl sodium sulfate), suggested as a standard reference toxicant by LaRoche et al (1970), has been used in some tests; however, it is evidently not a satisfactory standard for use over a wide range of temperatures. Rice et al (1976a) were unable to generate reliable data on DSS toxicity to some shrimp because a precipitate involving DSS forms readily above 30 ppm at 30 ‰ salinity and temperatures less than 15°C. They concluded that DSS is a poor reference toxicant.

Although the bioassay method is appropriate for comparisons of toxicity, few investigators have used the same test species, exposure conditions, or appropriate reference toxicant. It is therefore impossible to directly compare results between investigators. However, conclusions made by each investigator are usually valid, and one can compare conclusions formed by different investigators.

The second requirement when comparing oil toxicities is a standard test species. Comparisons of toxicity differences between several oils require that the sub-populations of animals used in the tests be of equal sensitivity. This is presumably assured when the same test species is used, although "reference toxicant" tests will verify that the sensitivities of the sub-populations have not been affected by other factors, such as sex, health, etc. Several investigators have used more than one species, and found the relative toxicities of different oils (or different methods of mixing a given oil) are generally not significantly affected by the choice of test species. Anderson et al (1974) tested four different oils each mixed with water in two different ways (WSF's and dispersions), and determined the toxicity of each oil-water mixture to six different marine animal species. Within each mixing method, the order of toxicity was not significantly different for the six animals. Rice et al (1976a) conducted a similar study and found that the LC50's of the eight species studied were generally within the same order of magnitude regardless of the kind of oil or mixing method used. Percy and Mullin (1975) found that the order of potency of four different crude oils was different for each of the three marine invertebrates they tested. However, they did not determine the concentrations of oil in the doses they used to determine the toxicity of these oils, so that these results may be due to variation in potency between preparations of their test solutions. When different investigators use different species to compare oil toxicities, the relative toxicities of different oils can be compared, but absolute values from one study cannot be compared to values from another study unless the same species is used. For the purpose of comparing toxicities of different oils, the use of a standard test species is most desirable.

Third, accurate comparisons of sensitivities of different species require that oil concentrations remain stable during the exposure. However, in static tests the oil concentrations usually decline with time, since aeration, biodegradation, temperature, and other factors affect the persistence of petroleum hydrocarbons in seawater. Nevertheless, most toxicity studies to date have used static exposures rather than flowthrough exposures because static exposures are relatively easy and inexpensive, and the technology for flowthrough bioassays with oil has been complicated, costly, and slow to develop.

There are two reasons why declining oil concentrations during static exposure cause difficulties in comparing the sensitivities of different species. First, the effective exposure period is short, possibly only a few hours, depending on the rate the oil concentration declines. Consequently, estimates of toxic oil concentrations are likely to be high. Since the rate of decline is likely to vary between tests, the effective period of exposure is also likely to vary between static tests. Second, some species accumulate the toxicants at faster rates than others, and are likely to be the species that will die faster than others. Animals that slowly approach equilibrium with toxic solutions may not be as tolerant as they seem because lethal concentrations may decline to sublethal concentrations before the animals have achieved equilibrium. Sensitivities of fast- and slow-dying species can be compared if they are tested long enough in flowthrough exposures where the toxicant concentrations are held constant during exposure. The value of flowthrough tests depends on constant exposure to stable concentrations, which must be verified by chemical analyses.

COMPARATIVE TOXICITY OF OIL-WATER MIXTURES, OILS, AND COMPONENTS OF OILS

Historically, bioassays were developed to determine the biological potency of substances such as drugs and insecticides. For these purposes, the bioassay method requires that a standard set of exposure conditions be maintained and that standard test species be used whose sensitivity does not change between tests. If the standard species sensitivity does change between tests, then reference bioassays may be conducted with a standard reference toxicant to identify and correct for this change in sensitivity. If the above requirements are met, then the bioassay method will yield valid comparisons of toxicity. The bioassay method is thus appropriate for determining relative toxicity differences.

Toxicity Differences Between Water-soluble Fractions of Oil and Oil-water Dispersions

The toxicity of oil-water mixtures depends in part upon the way the oil is associated with the water. Experiments comparing the toxicity of water-soluble fractions and dispersions of oil suggest that the toxicity of an oil is due to the soluble compounds contained in that oil, and not due to compounds in dispersed droplets. The chemical composition of the droplets is probably very similar to that of the parent oil, except that the droplets probably contain slightly lower concentrations of soluble compounds because of losses of these compounds due to equilibration with the water. If oil dispersions are less than or equally as toxic as WSF's (which contain far fewer dispersed droplets of oil), the toxicity must be due to the soluble fractions of oil.

Oil dispersions are slightly less toxic than WSF's of a given oil when oil concentrations are analyzed by IR. Rice et al (1976a) tested five species with dispersions and WSF's of two crude oils and found that toxicities of dispersions were always less than, but within an order of magnitude of, the toxicities for corresponding WSF's. Anderson et al (1974) did not analytically determine oil concentrations in the doses they used to determine the toxicity of dispersions. However, they did provide data relating the amount of oil used to prepare a dispersion to the amount of oil found in a typical dispersion (as determined by IR). After calculating the amount of oil found in toxic dispersions, we estimate that the toxicity differences between the dispersions and WSF's studied by Anderson et al ranged from nil to about an order of magnitude, with the dispersions being consistently less toxic.

Dispersions and WSF's seem to be equally toxic when oil concentrations are measured by UV. Rice et al (1976a) compared toxicities of dispersions and WSF's of two crude oils to each of 6 test species. They found significant differences in only 4 of 12 cases when oil concentrations were determined by UV. In 3 of these 4 cases, the dispersions were slightly more toxic than the corresponding WSF. The lack of significant differences in toxicity suggests that the toxicity of oils is due to the water-soluble compounds.

In conclusion, all these results suggest that oil toxicity is due to chemical toxicity of soluble aromatics, rather than physical toxicity of dispersed droplets.

Toxicity Differences Between Different Oils

Toxicities vary between oils, which is to be expected because the concentration and composition of individual hydrocarbons within the oil vary. The refined oils are generally considered to be more toxic than crude oils, since smaller volumes of refined oils are needed to kill 50% of the test animals in a given length of time. The increased toxicity of refined oils, as measured on a volume added basis, is primarily caused by two factors. Refined oils often have higher concentrations of aromatic hydrocarbons, and refined oils are usually less viscous than crude oils thus requiring less mixing energy for toxic concentrations to be mixed into the water.

We believe that direct toxicity comparisons between different oils is a simplistic approach, because so many factors other than composition differences between oils can affect the observed results. We now know that the toxicity of a given oil is influenced by mixing method, temperature, salinity, etc.

We believe that any oil, whether crude or refined, is best considered as a source of toxic compounds. Based on a knowledge of the toxicity of all the compounds contained in an oil and the concentration of these compounds, one should be able to predict the toxicity of any oil-water mixture. (This may not be so formidable a task, since the solubility of most toxic compounds in oil is so low that they probably do not make significant contributions to toxicity.) With this approach, the toxicity of an oil would be evaluated in terms of (1) the concentrations of toxic compounds it contained, and (2) the physical characteristics of the oil (such as viscosity) that would promote the transfer of these toxic compounds into solution when a standard oil-water preparation method is used.

Comparative Toxicity of Different Oil Components

Although there is general agreement that aromatic hydrocarbons are responsible for the toxic effects of crude oils and refined products, the relative importance of various aromatic hydrocarbons to toxicity is not clearly defined. Therefore, there has been an increasing interest in determining the degree of contribution of individual aromatics to the toxicity of oil-water solutions.

There have been several studies on the toxicity of individual hydrocarbons (summarized in Table 1). Studies by Neff et al (1976), Benville and Korn (In press), and Caldwell et al

(This symposium) compare the toxicities of mono- and di-aromatic compounds to four marine species. Even though the species tested are quite different, and include tests with larvae, the following three results are quite consistent: mono-aromatics are the least toxic, acute toxicity increases with increasing molecular size until the 4- and 5-ring compounds are reached, and alkylation of the aromatic nucleus seems to increase the toxicity of the parent compound. Thus, in both the benzene and naphthalene series, toxicity increases with increasing degrees of alkylation. The toxicities reported for m-, o-, and p-xylene for shrimp, bass, and crab larvae suggest that the position of alkyl substitution on the aromatic ring may influence toxicity (Benville and Korn In press, Caldwell et al This symposium). The most toxic hydrocarbon evaluated by Neff et al (1976) was 1-methylphenanthrene. Considering the toxicity of the compounds they studied and the concentrations of these compounds in toxic WSP's, Neff et al (1976) concluded that much of the toxicity of most crude and refined oils was due to the mono- and dinuclear aromatics.

TABLE 1

Comparative toxicity of different aromatic hydrocarbons, expressed in 96-hr LC50's with concentrations in ppm. Asterisk (*) indicates that toxic concentrations were above solubility limits.

AROMATIC H.C.	96-hr LC50's in ppm					
	POLYCHAETE ¹	SHRIMP ²	CRAB LARVAE ³	SHRIMP ⁴	BASS ⁵	GOLDFISH ⁶
Benzene	----	27	108	20	5.8-10.9	----
Toluene	----	9.5	28	4.3	7.3	22.8
Ethyl benzene	----	----	13	0.5	4.3	----
Tri-methyl benzene	----	5.4	5.1	----	----	12.5
Xylene	----	7.4	----	----	----	16.9
m-	----	----	12	3.7	9.2	----
o-	----	----	6	1.3	11.0	----
p-	----	----	----	2.0	2.0	----
Naphthalene	3.8	2.4	> 2			
Methyl naphthalene	----	1.1	1.6			
Di-methyl naphthalene	2.6	0.7	0.60			
Tri-methyl naphthalene	2	----	----			
Phenanthrene	0.6	----	----			
Methyl phenanthrene	0.3	----	----			
Fluorene	1	----	----			
Fluoranthrene	0.5	----	----			
Chrysene	*	----	----			
Benzo(a)pyrene	*	----	----			
1,2,5,6-Dibenzanthracene	*	----	----			

¹Neff et al (1976). *Neanthes arenaceodentata*

²Neff et al (1976). *Palaemonetes pugio*

³Caldwell et al (This symposium). *Cancer magister*, Stage I larvae

⁴Benville and Korn (In press). *Crago franciscorum*

⁵Benville and Korn (In press). *Morone saxatilis*

⁶Erannizan et al (1975). *Carassius auratus*

The solubility of individual aromatic hydrocarbons decreases with increasing methyl substitution and number of rings, and this is reflected in most WSP's (Caldwell et al This symposium). The concentrations of mono-aromatics, such as benzene, are usually greater in WSP's than naphthalene and other larger aromatics. Since the toxicity of aromatic hydrocarbons increases as the concentrations in the WSP decrease, it is difficult to identify which specific compounds are most responsible for the toxicity of WSP's. The toxicity of oil-water solutions is probably due to contributions from both the more soluble, less toxic mono-nuclear aromatics and the less soluble, more toxic dinuclear aromatics. The relative importance of the two classes will likely depend on several factors, such as their relative concentrations in the parent oil, temperature, mixing characteristics, etc.

Since the solubilities of the larger polynuclear aromatic hydrocarbons are so low, these compounds probably contribute little to the acute toxicity of oil-water solutions. Neff et al (1976) found several polynuclear aromatics that were not acutely toxic in four days at the maximum possible concentrations (solutions were 100% saturated). However, these

compounds may contribute to long-term damage if they accumulate to significant concentrations in the tissues after lengthy exposures.

Several investigators have given evidence suggesting that mono- and dinuclear aromatic hydrocarbons account for much of the toxicity of oil-water solutions, and there have been correlations of adverse effects with tissue concentrations of aromatic hydrocarbons. However, it is premature to conclude that the aromatic hydrocarbons are solely responsible for the acute toxicity of oil-water solutions. The presence and toxicity of polar hydrocarbon derivatives or polar oxidation products of oil hydrocarbons have generally been ignored, because they are more difficult to identify and measure.

COMPARATIVE SENSITIVITY OF DIFFERENT LIFE STAGES AND SPECIES

When assessing the potential impact of spilled oil, it is crucial to know if some species or life stages are more sensitive than others to oil toxicity. As previously discussed, comparisons of animal sensitivities to oil are most valid when tested with flowthrough exposures during which the oil concentrations remain constant. Unfortunately, most studies have used static exposures, in which the oil concentrations decline with time, and slowly dying animals may appear more tolerant than they actually are to four-day exposures. In spite of these limitations with static exposures, we attempt to compare sensitivities of different life stages and species to oil.

Comparing Sensitivities of Different Life Stages

Survival of a species is dependent on the survival of each developmental stage. In general, eggs are assumed to be more tolerant than other life stages to oil exposures because of the protection given by the surrounding chorion. In contrast, larvae have been assumed to be more sensitive to oil pollution, probably because of their high mortality in response to natural environmental stresses. In an earlier review, Moore and Dwyer (1974) concluded that larvae are approximately an order of magnitude more sensitive than adults to oil toxicity. Although their conclusions are consistent with general assumptions of larval sensitivity, support for their conclusions is based on literature where oil concentrations had to be "normalized" for comparative purposes.

Comparisons of results from early studies on the sensitivity of eggs to oil are severely limited since exposure concentrations are reported in quantities of oil added. The sensitivities of fish eggs and larvae of Black Sea turbot (Mironov 1967), herring eggs incubated under an oil film (Kühnhold 1970), and cod eggs incubated in the water-soluble fraction of crude oil (Kühnhold 1974) have been reported. In addition to mortality, effects on developmental rates and developmental abnormalities which would presumably cause death at a later stage have been observed. In more recent quantitative studies, Struhsaker et al (1974) found herring eggs to be more tolerant than larvae. Similarly, Rice et al (1975) found salmon eggs to be quite resistant to the crude oil WSF, and that sensitivity increased after hatching until yolk absorption was complete.

Some investigators have also attempted to document the sensitivity of larvae of various species to oil but the concentrations are given as "volume added" and comparisons are arbitrary. Crude oil concentrations as low as 1,000 ppm are reported to retard development of *Crassostrea* larvae (Renzoni 1975). Concentrations of 100 ppm of oil are fatal to larvae of lobster (Wells 1972), shrimp, and crab (Mironov 1968). In addition, 10 ppm or less produced a delayed mortality and resulted in marked changes in color and behavior in lobster larvae (Wells 1972). Chia (1973) exposed 14 species (5 phyla) of pelagic larvae to 0.5% diesel oil and found that larger larvae generally lived longer than smaller larvae. The large range in reported lethal concentrations (10 to 1,000 ppm) may reflect differences in the toxicities of crude oils or the sensitivities of species, but is more likely due to differences in preparation of the oil-water mixtures. As stated previously, the amount of oil in solution is particularly influenced by mixing energy and duration (Gordon et al 1973, Anderson et al 1974, Rice et al 1976a). All of the above studies on larvae were quantitated in terms of "volume of oil added" rather than by quantitative analytical measurements that allow comparisons.

Recent studies employing analytical measurement of oil concentrations in the test exposures have found the sensitivities of larvae and juveniles to vary considerably, with sensitivity depending on life stage and species (summarized in Table 2). Neff et al (1976) found larvae of grass shrimp to be more sensitive than adults, while post-larvae of brown shrimp were more resistant than early or late juveniles. They concluded that generalizations cannot be made about larvae versus adult sensitivity, and this is borne out by other studies. The early stages of a polychaete worm were found to be more resistant than later juvenile stages and adults to crude oil and No. 2 fuel oil (Rossi and Anderson 1976). Stage

I lobster larvae are more sensitive (4-day LC50 of 0.86 mg oil/liter) than stages III and IV (LC50 of 4.8 mg/liter) (Wells and Sprague 1976). Brodersen et al (In press) determined sensitivities of stage I larvae of four species of shrimp, two species of crab, and of six larval stages of coonstripe shrimp. Sensitivities differed considerably, but stage III and VI larvae of coonstripe shrimp were the most sensitive (4-day LC50's of 0.35 and 0.24 ppm). Brodersen et al concluded that the stage I larvae tested were all slightly more sensitive than adults (larvae were 1.2-4.9 times more sensitive). They concluded that larvae are slow to die, and that comparisons of sensitivities should be based on oil concentrations that cause moribundity rather than mortality during a four-day test.

The animals most sensitive to oil appear to be crustacean larvae but this may only be apparent because of test methodology differences, or because these larvae have been studied more than any other phylum. Brodersen et al (In press) report a 96-hr LC50 for moribundity of 0.24 ppm of crude oil for stage VI larvae of coonstripe shrimp; Wells and Sprague (1976) report an LC50 of 0.14 ppm of oil after exposing lobster larvae for 30 days; and Sanborn and Malins (1977) report an LC50 of less than 8-12 ppb of naphthalene after 36-hr exposures for larvae of spot shrimp and Dungeness crab. The test by Wells and Sprague was quite long (30 days) compared to most larval bioassays. The observations on shrimp larvae by Brodersen et al were of moribundity rather than death during the exposure. Although the observations of Wells and Sprague (1976) during the 30-day test with lobster were of death, they may be analogous to the previous observations on moribundity in shrimp larvae (Brodersen et al In press) since there was ample time during the 30-day tests for moribund lobster larvae to die. The tests by Sanborn and Malins (1976) were short in duration (36 hr) but were conducted with a pure and highly toxic compound under continuous-flow conditions.

TABLE 2

Comparison of larval sensitivities to crude oil and No. 2 fuel oil. The reported LC50's are from 96-hr static tests, except the 30-day test by Wells and Sprague, and the flowthrough, 36-hr tests by Sanborn and Malins.

Study and Species	Stage	Range of LC50 (ppm)	
		Crude oils	No. 2 fuel oil
Rossi and Anderson (1976)			
Polychaete	Juveniles	15-19.8	4-8.4
	Adults	12.5-17.6	2-4.2
Neff et al (1976)			
Grass shrimp	Larvae	--	1.2
3 sp. shrimp	Post larvae	--	1.4-6.6
3 sp. shrimp	Juveniles, adults	--	1.0-3.7
Wells and Sprague (1976)			
American lobster	Stage I-IV	0.8-4.9	--
	Stage I	0.14 (30-day LC50)	
Brodersen et al (In press)			
6 sp. shrimp and crab	Stage I	0.9-1.3	
Coonstripe shrimp	Stage I-VI	0.2-1.3	
Mecklenburg et al (This symposium)			
King crab	Stage I	2.0	
	Stage I <u>moltina</u>	1.3	
Coonstripe shrimp	Stage I and II	4-7.9	
	Stage I <u>moltina</u>	0.9	
Sanborn and Malins (1977)			
Spot shrimp	Stage I and V		Naphthalene < 12 ppb

Previous studies with crustacean larvae exposed to oil (Wells 1972, Katz 1973) have suggested that molting larvae were more sensitive, since mortalities often increased during molting. Mecklenburg et al. (This symposium) confirmed that molting larvae of coonstripe shrimp and king crab were more sensitive than non-molting larvae. Molting larvae of coonstripe shrimp were about five times more sensitive to oil than non-molting larvae. Similarly, adult tanner crabs exposed to oil just prior to molting died during the molting process (Karinen and Rice 1974). Emery (1970) also found molting larvae of two crustaceans to be more sensitive to creosol than non-molting larvae. Emery suggested that tolerance is reduced during molting because of the toxicant-laden fluid that is taken up rapidly prior to molting to create enough hydrostatic pressure to split the exoskeleton. Since all molting crustaceans are probably more sensitive to oil exposure than non-molting crustaceans, comparison of sensitivities will be more valid if sensitivities are determined for crustaceans when they are all in the same period of the molt cycle, such as intermolt. However, isolating intermolt larvae for toxicity tests can be a problem, especially in warmer climates where larvae of some species molt more frequently and the intermolt periods are shorter.

Juveniles of non-crustaceans, such as polychaetes, are apparently more tolerant than adults to oil (Rossi and Anderson 1976). Polychaete juveniles grow by adding additional segments, and do not molt like crustaceans. Since there is no molting process, polychaete juveniles do not have a rapid uptake of fluid associated with growth. These differences in growth and sensitivity suggest that molting animals are more vulnerable to oil, probably because of increased permeability during the molting process.

At this time, generalizations about greater sensitivity to oil during early life stages cannot be made, since a general pattern has not emerged from the literature. More life stages from several groups need to be tested, with the bulk of the early literature being of little value. It seems likely that sensitive stages will be those that are already under a certain amount of natural stress. Larvae in general would seem to qualify, since they typically have a high natural mortality. If larvae are more sensitive, then species from a colder climate may be particularly vulnerable because of the relatively long period spent as developing larvae.

Although some questions remain about the relative sensitivity of larvae and adults exposed to oil, larvae are probably more vulnerable than adults. Larvae that are weakened by oil, though not killed outright, may become easy prey while adults so affected are afforded some protection by their greater size and their better-developed exoskeletons. Furthermore, some adults and juveniles will probably avoid contaminated water, since they can detect the oil and have the motor ability to avoid the area (Rice 1973). Larvae may lack both of these abilities. Lack of avoidance behavior has been observed in larvae of three species of marine fish (Kühnhold 1970) and in herring and crab larvae (Rice et al 1976a). The interactions in the environment are complex, and while sensitivities to oil can be extrapolated from the laboratory to the ecosystem, vulnerability and survival of larvae exposed to oil in the natural environment are another matter.

Comparing Sensitivities of Different Species

The sensitivities of several marine species have been tested in quantitative static or flowthrough tests (summarized in Table 3). There have been differences between the studies, such as the oil used, mixing techniques, analytical methods, temperatures, and exposure methods (static or flowthrough). In spite of these differences, the ranges of LC50's overlap considerably between most studies, suggesting that sensitivities of most animals are fairly similar.

The study by Rice et al (1976b) tested 27 species of marine fish and invertebrates, and permits the best comparisons of species sensitivities since methods, temperature, etc., were all similar. Fish and shrimp were usually among the more sensitive species tested, while intertidal animals were generally more tolerant. Intertidal animals are probably more tolerant to static exposures because they can temporarily insulate themselves from the exposures, at least until the concentrations in the static exposures have declined to sublethal levels. The intertidal limpets and chitons were more sensitive than the other intertidal animals, but this may have been due to damage occurring when they were collected (pried off the substrate).

The sensitivities of cold-water fish and shrimp (Rice et al 1976b, Korn et al In prep.) appear greater than sensitivities of similar species from warmer climates (Anderson et al 1974, Neff et al 1976). The differences in sensitivity are consistent, but are not large. Rice et al (1976b) speculate that the cold-water species may appear more sensitive because lower temperatures increase the persistence of toxic aromatic hydrocarbons, even though there are differences in oils and species between the studies. This speculation is compatible with other studies. Cheatham et al (In prep.) have shown that water-soluble fractions

at lower temperatures have increased persistence of aromatic hydrocarbons because of decreased losses from evaporation and biodegradation. Korn et al (In prep.) tested the effect of temperature on the toxicity of toluene, naphthalene, and the water-soluble fraction of crude oil to shrimp and fish. They found that with increasing temperature, toxicity increased for shrimp but decreased for fish. They concluded that temperature affects complex interactions in three ways: (1) the persistence of aromatic hydrocarbons is increased at lower temperatures, (2) temperature affects an animal's sensitivity by changing the rates of hydrocarbon uptake, metabolism, and excretion, and (3) temperature may act as a synergistic stress at cold or warm extremes.

TABLE 3

Comparison of adult sensitivities to crude oils and No. 2 fuel oil. All tests were static, except the flowthrough tests (FT) by Battelle. Most of the LC50's reported for the Battelle studies are estimates that have been calculated from Battelle's raw data.

Study and species tested	Temperature range (°C)	96-hr LC50's (ppm)	
		Crude oils	No. 2 fuel oil
Battelle/Sequim 1973-76			
2 fish sp. (FT) ¹	8	15-65	--
Coonstripe shrimp ₁ (FT) ^{2,3}	10-11	6.6-24.9	0.8
Coonstripe shrimp ¹	8	1.3-4.9	--
Texas A&M 1974-76			
4 crustacean sp. ^{4,5}	18-22	6->19.8	1.3-4.9
3 fish sp. ⁶	18-22	5.5-19.8	3.9-6.3
3 polychaete sp. ⁶	20	9.5-12.5	2.3-2.7
Auke Bay Lab 1976 ⁷			
4 fish sp.	3.6-10	1.2-2.9	0.8-2.1
6 crustacean sp.	3.5-5.4	0.6-4.2	0.5-1.7
4 limpet and chiton sp	3.9-7	3.6-9.6	0.4-5.0
12 invertebrate sp.	3.6-10	>3.1-14.7	>0.9-5.6

¹Bean et al 1974

²Vaughan 1973

³Vanderhorst et al 1976

⁴Anderson et al 1974

⁵Neff et al 1976

⁶Rossi et al 1976

⁷Rice et al 1976

CONCLUSIONS

Methods

1. Toxicity tests with oil require some form of chemical analysis to determine what concentrations of oil are in the water. Gas chromatography or HPLC is preferable since these methods can measure concentrations of individual aromatic hydrocarbons.

2. Statistical analysis generating confidence intervals is more informative than graphic estimates of the LC50.

3. If oil toxicities are compared, reference toxicants and the same test species should be used.

4. If species sensitivities are compared, flowthrough tests with stable oil concentrations during exposure should be used.

Comparative Toxicity

1. Crude and refined oils are best considered as sources of toxic compounds, with toxicity depending on the concentration of toxic compounds in the oil and on physical factors, such as temperature and viscosity of the oil, which affect the transport of petroleum hydrocarbons into the water.
2. Refined oils are generally considered less toxic than crude oils on a volume added basis because refined oils often have higher concentrations of aromatic hydrocarbons and are usually less viscous than crude oils.
3. The toxicity of oils is apparently due to the soluble compounds in the water rather than dispersed droplets.
4. The toxicity of aromatic hydrocarbons increases with the number of rings and with the degree of alkyl substitution. The solubility of these compounds decreases with these factors, so that the relative importance of individual aromatic hydrocarbons to toxicity of water-soluble fractions is unknown. Mono- and dinuclear aromatics are probably the most important classes of compounds, accounting for most of the toxicity in water-soluble fractions.

Comparative Sensitivity

1. No conclusions can be made concerning egg sensitivities because not enough quantitative data exist.
2. Crustacean larvae appear more sensitive than most adults. Molting animals are more sensitive than non-molting animals.
3. Sensitivity data for larvae or juveniles from other groups are generally lacking, except for polychaete juveniles, which are more tolerant than adults.
4. Adult sensitivities are fairly similar when data from static tests are compared. Intertidal animals appear more tolerant in short-term, static exposures, probably because of their ability to temporarily insulate themselves from the environment.
5. Cold-water species probably have sensitivities that are equivalent to the sensitivities of species from warmer water, but cold-water species may be more vulnerable to oil toxicity because toxic aromatic hydrocarbons persist longer at colder temperatures.
6. The preceding conclusions are based on static exposures. Because of the deficiencies of the static exposure method (declining dose), conclusions about sensitivity differences may be altered considerably when tests with flowthrough exposures have been conducted.

Recommendations for Future Research

1. A high standard of quantitative methodology (flowthrough tests, chemical analyses, and statistical analyses) should be used in future toxicity tests.
2. There is little need to test the toxicity differences between different oils. Future research should: (a) determine if aromatics account for most, if not all, of the toxicity of WSP, and (b) determine the relative importance of individual mono- and dinuclear aromatics to the acute toxicity under various conditions.
3. Additional sensitivity data are needed for eggs, larvae, and juveniles of several groups of animals such as molluscs, echinoderms, etc. since virtually no information on these animals is available.
4. Several species need to be retested with flowthrough exposures, so that (a) better estimates of sensitivity can be measured, and (b) sensitivities can be compared between species.
5. A few selected species should be tested for longer periods of time, so that the relationship between oil concentrations that are toxic for short and long exposures can be determined.

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MOLTING AND SURVIVAL OF KING CRAB (*PARALITHODES CAMTSCHATICA*)
AND COONSTRIPE SHRIMP (*PANDALUS HYP SINOTUS*) LARVAE
EXPOSED TO COOK INLET CRUDE OIL WATER-SOLUBLE FRACTION

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ABSTRACT

Larvae of coonstripe shrimp and king crab were exposed to solutions of the water-soluble fraction (WSF) of Cook Inlet crude oil in a series of bioassays on intermolt stages I and II and the molt period from stage I to stage II. Molting larvae were more sensitive than intermolt larvae to the WSF, and molting coonstripe shrimp larvae were more sensitive than molting king crab larvae. When molting larvae were exposed to high concentrations of the WSF (1.15-1.87 ppm total hydrocarbons) for as little as 6 hr, molting success was reduced by 10-30% and some deaths occurred. When larvae were exposed to these high concentrations for 24 hr or longer, molting declined 90-100% and the larvae usually died. The lowest concentrations tested (0.15-0.55 ppm total hydrocarbons) did not inhibit molting at any length of exposure, but many larvae died after molting. Median lethal concentrations (LC50's) based on 144 hr of observation for molting coonstripe shrimp and 120 hr for molting king crab were much lower than the 96-hr LC50's, showing that the standard 96-hr LC50 is not always sufficient for determining acute oil toxicity. Although our LC50's for intermolt larvae are higher than levels of petroleum hydrocarbons reported for chronic and spill situations, some of our LC50's for molting larvae exposed 24 hr and longer are similar to or below these environmental levels. Comparisons of sensitivity to oil between different crustacean species or life stages should be based on animals tested in the same stage of the molt cycle, such as intermolt.

Key Words: Molting, crustaceans, larvae, *Paralithodes camtschatica*, *Pandalus hypsinotus*, crude oil, Alaska.

INTRODUCTION

The breeding and larval stages of marine invertebrates are considered to be the most sensitive to natural environmental stresses (Thorson 1950). In crustacean larvae this sensitivity is compounded during molting. Factors contributing to a high natural mortality in molting crustaceans include increased permeability and altered ionic regulation, the mechanical process of casting off the old exoskeleton, and increased predation while the cuticle is still soft and locomotion is slowed down (Lockwood 1967, Hagerman 1973). Manmade stresses such as pollution from offshore oil production and transoceanic transport of crude and refined oils could impose an additional burden. Pollution by petroleum hydrocarbons in Alaskan surface waters would be particularly damaging to crustacean larvae in the early spring when the larvae are released from the females and undergo several rather closely spaced molts. Cold temperature is another factor to consider in arctic and subarctic waters because aromatic hydrocarbons persist longer (Atlas and Bartha 1972, Cheatham et al. 1976) and some animals retain accumulated hydrocarbons for longer periods of time (Short and Rice in prep.) than would be the case in warmer waters.

The sensitivity of molting crustacean larvae to oil pollution has been reported for only a few species. Oil was more toxic to molting larvae than intermolt larvae in tests with a lobster, *Homarus americanus* (Wells 1972, Wells and Sprague 1976), and the crabs *Neopanope packardiana* (Katz 1973) and *Rhithropanopeus harrisi* (Neff et al. 1976). Sublethal effects on these crustaceans included both delayed and stimulated molting.

For this study we chose two crustaceans that are important Alaskan fishery resources, the king crab, *Paralithodes camtschatica*, and the coonstripe shrimp, *Pandalus hypsinotus*, and exposed their larvae to various concentrations of the water-soluble fraction of Cook Inlet crude oil. Separate bioassays were conducted on larvae during intermolt stages I and II and during the molting period from stage I to stage II. We wished to determine sensitivity differences between the intermolt and molt stages and the effects of various concentrations

and lengths of exposure to the WSF on molting success and survival.

METHODS

Mixing and Analysis of Oil Solutions

Solutions of the water-soluble fraction of Cook Inlet crude oil were prepared at ambient seawater temperatures (3°-5°C) and salinity (29 ‰) by the methods of Anderson et al (1974) as modified by Rice et al (In press). Samples of the test solutions were taken at the beginning of each exposure and whenever a solution was replaced with a fresh solution. The samples were measured by infrared spectrophotometry (IR) at a wavelength of 2930 cm⁻¹ (Gruenfeld 1973). At this wavelength IR measures paraffinic hydrocarbons, but not aromatics. Optical densities were converted to ppm total hydrocarbons by comparing the optical densities to prepared standards. Gas chromatographic characterization of a typical WSF of Cook Inlet crude oil has been given by Rice et al (1976).

Collection and Rearing of Larvae

Ovigerous coonstripe shrimp and king crab were caught in pots or collected by divers and held in flowing seawater tanks at the Kasitsna Bay and Auke Bay laboratories. Individual females were isolated in rearing containers just before hatching and release of the larvae. When the larvae were released the females were removed from the containers. The water in the rearing containers was aerated continuously and changed daily. Larvae were fed daily on detritus and laboratory-reared *Artemia*.

Bioassays

The bioassays were conducted in sealed 200-ml and 500-ml glass jars submerged in a waterbath chilled to ambient seawater temperature. There were replicate jars for each of 7 to 12 concentrations of the WSF. There were 10-20 larvae in each jar and the tissue weight/volume ratio in the test jars never exceeded 1 g/liter. Oxygen concentrations measured at the end of the exposure periods were above 70% saturation.

Observations of death and molting success were made without removing the larvae from the test jars. This eliminated handling of the larvae, which could be detrimental. We observed the larvae in each test jar by holding the jar horizontally above a 24-in convex magnifying mirror. Larvae were recorded as dead when there was no visible motion.

Intermolt stage I king crab and coonstripe shrimp larvae were bioassayed within 24 hr of release from the females. The first intermolt stage lasted about 10 days, so we could be sure that molting would not begin during these bioassays. Bioassays on molting larvae were begun 1 or 2 days before the larvae started to molt to stage II. Intermolt stage II coonstripe shrimp larvae were bioassayed 3 days after molting was completed. The second intermolt stage also lasted 10 days. No bioassays were conducted on intermolt stage II king crab larvae.

For intermolt larvae we conducted static 96-hr bioassays (single-dose with declining concentration). Three separate bioassays were conducted on coonstripe shrimp intermolt stage I larvae, and one bioassay each for coonstripe shrimp intermolt stage II and king crab intermolt stage I.

We conducted several bioassays on molting larvae, and each bioassay had a different exposure period: 6, 24, 48, and 96 hr for coonstripe shrimp, and 6, 12, 24, 48, and 72 hr for king crab. In exposures longer than 24 hr the old WSF solution was replaced with fresh solution every 24 hr. After the exposure periods the larvae were returned to uncontaminated seawater for further observations. The total length of time for each test, including the exposure period and the period in clean seawater, was 120 hr for king crab larvae and 144 hr for coonstripe shrimp larvae.

Statistical Analysis

The acute toxicity of the Cook Inlet crude oil WSF to intermolt and molting larvae was expressed as the median lethal concentration (LC50), which is the concentration of WSF causing death in 50% of exposed larvae in a given amount of time. For intermolt larvae we give the standard 96-hr LC50. For molting larvae we give 96-hr LC50's which, although they are each based on 96 hr of observation, involve different exposure times and periods in clean seawater. When comparing acute toxicity to molt and intermolt larvae, for molting

larvae we use the 96-hr LC50 based on 24 hr of exposure and 72 hr in clean seawater.

The LC50's and 95% fiducial limits were calculated by probit analysis (Finney 1971) where possible. Where the data were insufficient for probit analysis we determined LC50's by the methods of Spearman-Kärber (Finney 1971) and Dorfneroff et al (1951). Abbott's formula (Finney 1971) was used to compensate for control deaths.

RESULTS

Molting larvae of both coonstripe shrimp and king crab were significantly more sensitive to the Cook Inlet crude oil WSP than were intermolt larvae of either species (LC50's with nonoverlapping fiducial limits, Table 1). Molting coonstripe shrimp larvae were four to eight times more sensitive to the WSP than were intermolt stages I and II. The tolerance of king crab larvae during molting was not decreased to the extreme observed in coonstripe shrimp larvae.

TABLE 1

The 96-hr LC50's for molting and intermolt larvae of coonstripe shrimp, *Pandalus hypsinotus*, and king crab, *Paralithodes camtschatica*, exposed to the WSP of Cook Inlet crude oil. The 95% fiducial limits are given in parentheses.

Larval stage	96-hr LC50's in ppm of total hydrocarbons for--	
	Coonstripe shrimp	King crab
Intermolt larvae:		
Stage I	7.94* (6.05-9.50)	2.00 (1.60-2.60)
Stage II	4.06 (3.22-5.11)	--- ---
Molting larvae:		
Stage I-Stage II	0.95 (0.87-1.03)	1.33 (1.23-1.45)

*Mean of three separate bioassays.

As the WSP concentrations and exposure periods increased, the number of coonstripe shrimp larvae that molted to stage II declined (Fig. 1). Control coonstripe shrimp larvae for each bioassay completed molting to stage II, with 95-100% molting success, by the sixth day or 144 hr after the beginning of each bioassay. At the lowest concentration tested, 0.25 ppm total hydrocarbons, there was little or no effect on molting success even when the larvae were exposed to replenished WSP solutions for as long as 96 hr. The next highest concentration, 0.63 ppm total hydrocarbons, did not affect molting success when the larvae were exposed only 6 hr, but was progressively more effective in inhibiting molting with each increased duration of exposure. The highest concentrations, 1.15 and 1.37 ppm, severely inhibited molting in exposures of 24 hr or longer. Molting success was only about 10% in these longer exposures to high concentrations and did not drop below 10% even when the WSP solutions were renewed daily.

Molting success in king crab larvae exposed to the WSP followed essentially the same pattern of decline (Fig. 2). Control groups for king crab larvae molting to stage II reached a maximum molting success of 37% by the end of 120 hr of observation. Exposure of king crab larvae to the two lowest concentrations, 0.15 and 0.55 ppm total hydrocarbons, for 6 and 12 hr resulted in some stimulation of molting compared to controls. Longer exposures to these low concentrations had little effect, even when the larvae were exposed for 72 hr to periodically-renewed solutions. The 1.20 ppm concentration had no effect on molting success at exposures less than 48 hr, but 48- and 72-hr exposures completely inhibited molting. The highest concentrations, 1.65 and 1.87 ppm, progressively inhibited molting with increased exposure time until the 48- and 72-hr exposures which again resulted in zero molt.

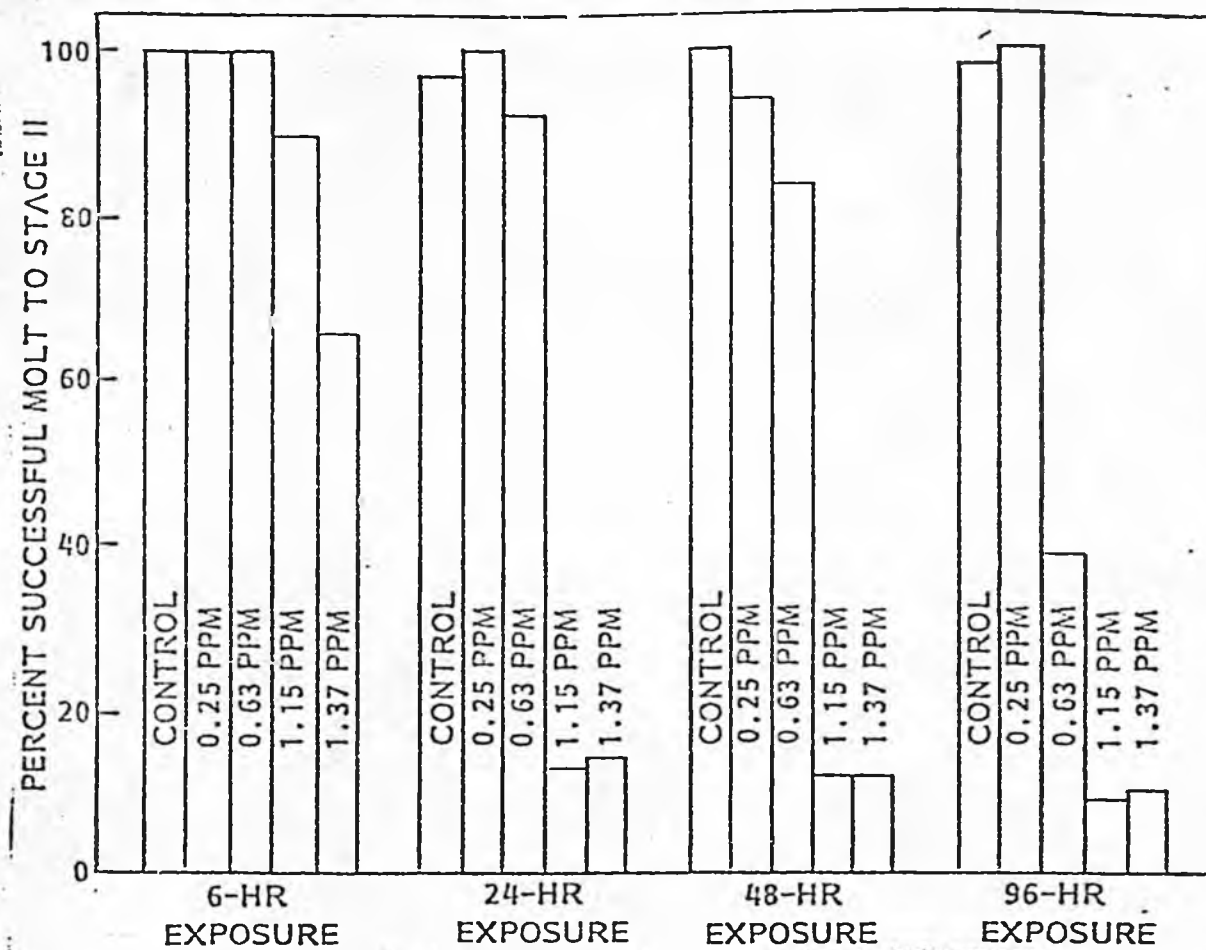


Fig. 1. Molting success of larvae of coonstripe shrimp, *Pandalus hypsinotus*, exposed for increased lengths of time to increased concentrations of the WSF of Cook Inlet crude oil. After the exposure time listed, the exposure water was replaced with clean seawater to make a total of 144 hr of observation for each test. Molting success at 144 hr = stage II larvae/initial total of stage I larvae.

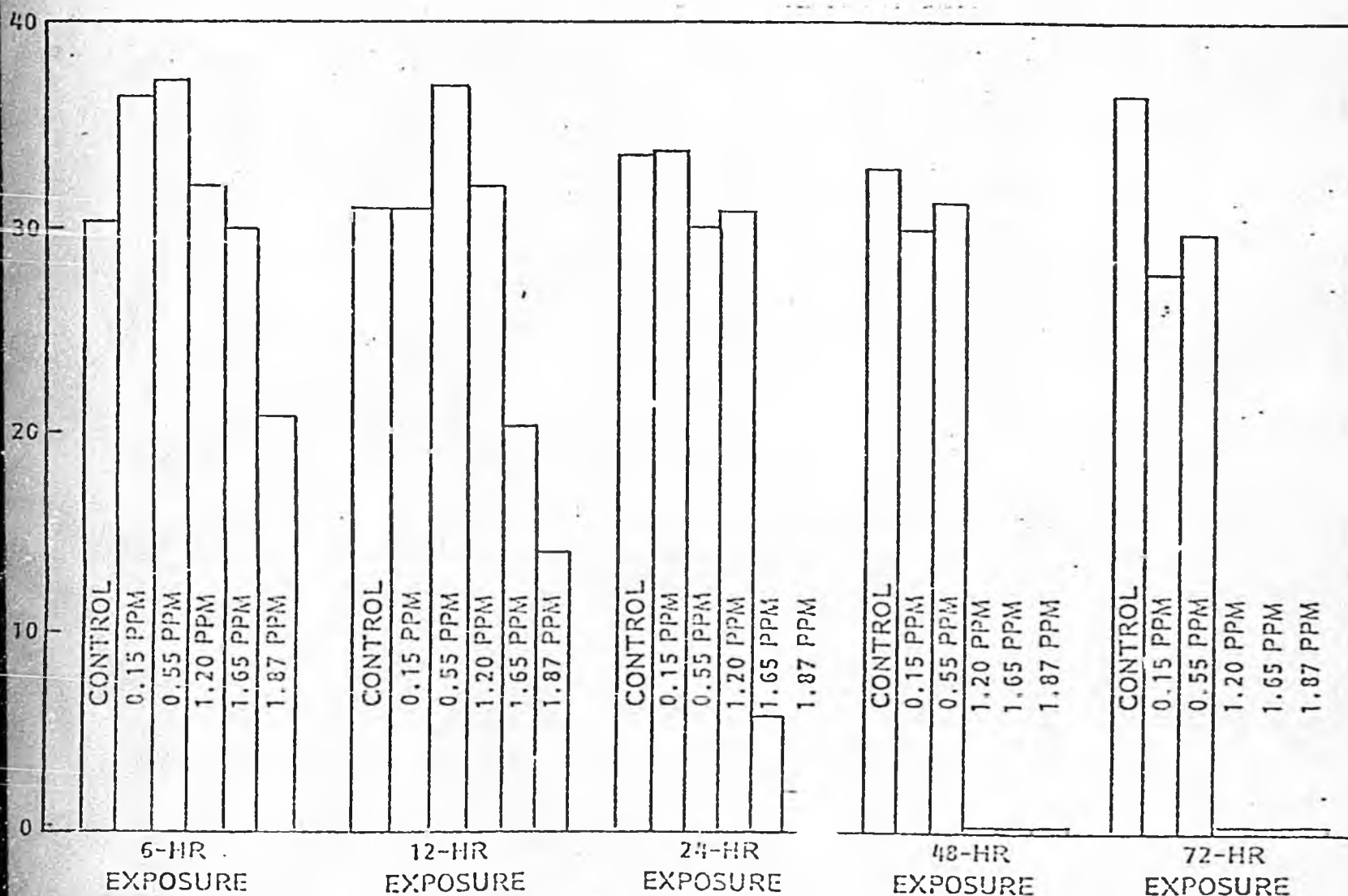


Fig. 2. Molting success of larvae of king crab, *Paralithodes camtschatica*, exposed for increased lengths of time to increased concentrations of the WSF of Cook Inlet crude oil. After the exposure time listed, the exposure water was replaced with clean seawater to make a total of 120 hr of observation for each test. Molting success at 120 hr = stage II larvae/initial total of stage I larvae.

rate in the tests. This is shown by comparing the LC50's for deaths occurring by 96 hr with the LC50's for deaths occurring by the end of the observation period (Table 2). There were no significant numbers of deaths by 96 hr when the larvae were exposed for only 6 hr, but deaths occurring within the next 48 hr yielded an LC50 of 2.24 ppm total hydrocarbons for coonstripe shrimp. (Observations were not continued as long for king crab.) For coonstripe shrimp larvae exposed 24 hr and longer the 96-hr LC50's were all similar, averaging 0.96 ppm, and also very similar in king crab which averaged about 1.40 ppm total hydrocarbons. The progressively more toxic effects of these longer exposures to periodically-renewed WSF solutions did not show up until sometime after 96 hr, when the larvae were in clean seawater. For example, for coonstripe shrimp larvae exposed for 96 hr, the LC50 dropped from 0.96 ppm at 96 hr to 0.24 ppm at 144 hr.

TABLE 2

The LC50's for larvae of coonstripe shrimp and king crab exposed to the WSF of Cook Inlet crude oil during the molting period from stage I to stage II. After the duration of exposure listed, the exposure water was replaced with clean seawater to make a total of 144 hr for each test on coonstripe shrimp and 120 hr for king crab. The 95% fiducial limits are given in parentheses; NS indicates insignificant number of deaths.

Duration of oil exposure	LC50's in ppm of total hydrocarbons for--			
	Coonstripe shrimp		King crab	
	96-hr LC50	144-hr LC50	96-hr LC50	120-hr LC50
6 hr	NS	2.24	NS	NS
12 hr	No bioassay		4.75	1.75 (1.72-1.78)
24 hr	0.95 (0.87-1.03)	0.62 (0.35-0.75)	1.33 (1.23-1.45)	1.38 (1.08-1.61)
48 hr	0.98 (0.83-1.15)	0.50 (0.42-0.62)	1.45 (1.35-1.50)	0.76 (0.66-0.88)
72 hr	No bioassay		1.37 (1.20-1.55)	0.93 --
96 hr	0.96 (0.80-1.15)	0.24 (0.15-0.37)	No bioassay	

DISCUSSION

Larvae of coonstripe shrimp and king crab were more sensitive to the WSF of Cook Inlet crude oil during the molting period from stage I to stage II than during intermolt stages I and II (Table 1), which agrees with previous studies on molting crustacean larvae exposed to different oils. In two of the earlier studies (Wells 1972, Katz 1973) the oil concentrations were not chemically quantified so the reported lethal concentrations cannot be compared to ours. Lethal concentrations for molting stage I-II larvae reported in the other two studies (Neff et al. 1976, Wells and Sprague 1976) are based on different oils and different types of bioassays than ours, but are useful to mention for the purpose of a broad comparison. The 96-hr LC50 for the first larval molt of the crab *Rhithropanopeus harrisi* was between 0.63 and 0.94 ppm (WSF of No. 2 fuel oil; Neff et al 1976) and for the lobster *Homarus americanus* it was 0.86 mg/liter (oil-water dispersion of crude oil; Wells and Sprague 1976). These concentrations are similar to our 96-hr LC50's of 0.95 ppm for molting coonstripe shrimp larvae and 1.33 ppm for molting king crab larvae in that all of the concentrations are lower than the 96-hr LC50's reported for nonmolting larvae, juveniles, and adults exposed to various oils (reviewed by Wells and Sprague 1976).

Molting coonstripe shrimp larvae were about four times more sensitive to the WSF than molting king crab larvae (Table 1), suggesting that as much difference in sensitivity to oil exists between crustacean species during molting as has been reported for species in intermolt stages. These differences in sensitivity to oil also occur intraspecifically at

larvae differ in their sensitivities to oil with, for example, stage III being more sensitive than stages I and II (Brodersen et al In press); this suggests that coonstripe shrimp larvae molting at the later stages could also be more sensitive than we observed for the first molt. However, larvae of *Neopanope texana* were more sensitive during the first molt than the later molts (Katz 1973). In *Homarus americanus* (Wells and Sprague 1976) and *Rhithropanopeus harrisi* (Neff et al 1976) most of the deaths also occurred during the first molt, but the tests on these species were long-term (30 or more days) and did not include separate tests for the different molt periods, so the sensitivities of later molts were not determined. The available data are insufficient to determine if the most sensitive molt period differs with species or is usually the first larval molt, and if any one group of crustaceans (such as shrimp) is more sensitive than another (such as crab) during molting.

The effects on molting success (Figs. 1 and 2) and mortality (Table 2) that occurred when larvae of coonstripe shrimp and king crab were exposed for increasing periods of time to increasing concentrations of the WSF of Cook Inlet crude oil were basically the same for each species and are summarized as follows. The lowest concentrations of the WSF (0.15-0.55 ppm total hydrocarbons) did not cause molting success to decline at any length of exposure, but did cause some stimulation of molting in king crab larvae after short exposures. Although molting success did not decline, at long exposures to the low concentrations many of the larvae died after molting, as reflected in the low 144-hr and 120-hr LC50's. Exposure of larvae to the highest concentrations of the WSF (1.15-1.87 ppm) for only 6 and 12 hr reduced molting success by 10-30% and resulted in some deaths of both molted and nonmolted larvae. When exposed to these high concentrations for 24 hr or longer, 90-100% of the larvae were inhibited from molting and usually died, yielding LC50's that were equal to or well below the high concentrations tested. The stimulation of molting we observed in king crab larvae has also been reported for larvae of the crab *Rhithropanopeus harrisi* when exposed to low concentrations of No. 2 fuel oil (Neff et al 1976). On the other hand, we did not observe any delays in molting relative to controls; long-term studies like those reported on crabs and lobster (Katz 1973, Neff et al 1976, Wells and Sprague 1976) would be needed to determine if the nonmolted larvae which survived our tests would have eventually molted to stage II or later stages.

In our bioassays on molting larvae, many larvae died after 96 hr, so the progressively more toxic effects of increased exposure times and increased concentrations of the Cook Inlet crude oil WSF were not as clear from the 96-hr LC50's as they were from the 144-hr and 120-hr LC50's (Table 2). Delayed mortality (i.e., deaths occurring after exposure) has also been noted for some other crustacean larvae exposed to toxic pollutants (Buchanan et al 1970, Brodersen et al In press). These data indicate that a bioassay lasting only 96 hr is not always sufficient to yield an accurate determination of acute toxicity.

Greater sensitivity to environmental stresses during molt than intermolt periods is a general phenomenon among crustaceans (Lockwood 1967) which should also be reflected in tests on crustaceans molting at any life stage, and in tests with pollutants other than oil. Exposures of tanner crab, *Chionoecetes bairdi*, to crude oil near the time of molting resulted in reduction in molting success and autotomizing of limbs (Karinen and Rice 1974). The greater sensitivity of molting crustacean larvae during exposures to insecticides (Buchanan et al 1970, Epifanio 1971) and creosol (Emery 1970) has also been reported.

Increased sensitivity to oil during molting is probably related to the physiological changes associated with the molting process. Rises in blood osmotic pressure and changes in permeability to seawater (Lockwood 1967, Hagerman 1973) could result in heightened concentrations of toxic hydrocarbons in the tissues. Depressed metabolism has been observed in molting adult blue crab, *Callinectes sapidus* (Lewis and Haefner 1976), and in oil-exposed king crab, *Paralithodes camtschatica* (Mecklenburg and Rice In prep.), and may impair the ability of molting larvae to metabolize and excrete the oil-derived hydrocarbons accumulated in the tissues.

Because molting has a significant influence on the sensitivity of crustaceans to oil, comparisons of sensitivities between life stages and species will be most valid if based on animals tested in the same stage of the molt cycle, such as intermolt. Testing the sensitivity of intermolt larvae to oil may be difficult for many crustacean species because the intermolt periods are often as short as 2 or 3 days. Thus, larvae would begin to molt during the standard 96-hr bioassay.

Measurements of hydrocarbons from chronically polluted areas or oil spills in Alaskan waters are not yet available. Levels of petroleum hydrocarbons reported for other areas, harbors and tanker routes as well as the open ocean (reviewed by Brown et al 1973, Wells and Sprague 1976), are very low compared to our LC50's for intermolt and molting larvae of king crab and coonstripe shrimp. However, the concentration of 0.8 mg/liter reported for a small oil spill in Nova Scotia (Gordon et al 1973) is higher than some of the LC50's we found for molting larvae exposed 24 hr and longer.

CONCLUSIONS

(1) For both coonstripe shrimp and king crab, larvae molting from stage I to stage II were more sensitive to the WSF of Cook Inlet crude oil than were larvae in intermolt stages I or II. From this we further conclude that comparisons of the sensitivities of crustaceans to oils should be based on tests on animals in the same stage of the molt cycle, such as intermolt.

(2) In tests on molting coonstripe shrimp and king crab larvae, as the concentrations and lengths of exposure to the WSF of Cook Inlet crude oil increased, molting success decreased and deaths increased. This pattern was not as clear from the 96-hr LC50's as it was from the 120-hr and 144-hr LC50's. From this we also conclude that the standard 96-hr bioassay is not always long enough for accurate determinations of the sensitivities of crustacean larvae to oils.

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EFFECTS OF OIL ON MARINE ECOSYSTEMS: A REVIEW FOR ADMINISTRATORS AND POLICY MAKERS

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ABSTRACT

A broad selection of recent literature on the effects of oil on marine ecosystems is reviewed. The focus is on studies on crude oil, and the results are discussed with the purpose of providing a summary of findings that will be a useful reference for administrators and policy makers involved in decisions concerning petroleum developments and related activities. The characteristics of crude oil and factors modifying its impact on the marine environment are discussed. Most research on the toxicity of oil has dealt with acute effects and data on long-term impacts at the community level are inconclusive. It is concluded that chronic low-level pollution is potentially more damaging to ecosystems than isolated catastrophic spills. Decision makers are forced to rely on interpretative judgments rather than conclusive data.

Much of the material in this report was gathered as background material for use in preparing the marine section of the final environmental impact statement on the proposed trans-Alaska pipeline system (U.S. Department of the Interior, 1972). Some of the statements are essentially unchanged from the way they were presented in the appendix to volume IV of the impact statement. The impact statement made it clear that not enough data are available to analyze conclusively all of the potential environmental impacts of operation of the pipeline marine terminal facilities at Port Valdez, Alaska, and the transshipment of crude oil by tankers to west coast ports. A conclusion that can be drawn, however, and a message of the impact statement, is that oil poses a significant hazard to marine ecosystems, and a good deal of intensive research is necessary if these hazards are to be quantified and fully understood.

Research on oil pollution published since the impact statement on the pipeline was issued reveals that scant progress has been made, particularly with regard to the effects of chronic low-level oil pollution. Current and projected demands for energy in the United States are prompting accelerated development of offshore petroleum reserves, expanded oil tanker traffic, and proposals for construction of deepwater port facilities to handle the increasing number of supertankers. These developments will not wait for conclusive

answers to questions on oil pollution. Recognizing this, we feel it is important that public administrators and policy makers be made aware of the inferences and trends evident in the research findings to date. These findings present a persuasive case that decisions regarding the handling of crude oil and petroleum products should be conservative and in favor of protecting the natural environment. While this report is by no means a complete review of the literature, it is sufficient to illustrate the potential danger of oil pollution to marine ecosystems and provide some guidance for policy decisions.

History is replete with examples of man's scientific and technological advances carrying him into situations he did not fully comprehend and with consequences he could not evaluate. Bella (1970) noted that "our ability to change this world is going to increase faster than our ability to predict what that change is going to be." He concludes that our management procedures must recognize the degree of ignorance we have about this world in which we live.

Pollution of the ocean by oil is a worldwide problem of growing concern to many nations. Spills like the *Torrey Canyon*, the *Arrow*, the Santa Barbara Channel blowout, and other spectacular incidents have helped stimulate international organizations of governments and industry to react to the problem. Viewed pragmatically, international response has been at least as adequate as domestic programs. Predicting the impact of an oil spill on the environment requires an understanding of the complex interactions involved. What

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appears to be universally lacking is the difficult research leading to an understanding of chronic and sublethal effects of oil at the biological community level. The following discussion outlines these complexities and points out how they make most generalizations invalid and the extrapolation of most data dangerous,

DESCRIPTION OF OIL

Crude oil is a complex mixture of many different specific hydrocarbons and a variety of compounds containing sulfur, oxygen, nitrogen, and some trace metals. Hydrocarbons make up the bulk of crude oil and can roughly be placed into one of three classes: paraffinic, naphthenic, and aromatic. From one area to another, crude oils vary in their composition and in density, volatility, and solubility. Their relative toxicity will vary (Ottway, 1971) but is roughly proportional to their aromatic content.

Paraffinic (or aliphatic) hydrocarbons are straight or branched carbon chains and are saturated (thus no carbon-carbon double bonds) with hydrogen or other groups. These hydrocarbons are the least toxic, although they may have an anesthetic or narcotic effect if concentrations are great enough.

Naphthenic compounds (cycloparaffins) contain at least one ring structure that is saturated. With this base, more rings or chains may be attached to form a variety of complex molecules.

Aromatic hydrocarbons also contain a ringed structure, but the ring is unsaturated with hydrogen and contains carbon-carbon double bonds (benzene ring). The simplest aromatic is benzene, which is very toxic and relatively water soluble in comparison to most hydrocarbons found in crude oil. Benzene and other low-boiling aromatics are the most toxic petroleum fractions. High-boiling aromatics act as slower poisons than low-boiling aromatics, but they are equally severe in their effect. In addition, some are known to induce cancer; 3,4-benzopyrene, 1,2-benzanthracene, and some alkylbenzanthracenes have been isolated from crude oil, and their carcinogenic effects on animals and man have been demonstrated (Blumer, 1970).³

³Blumer, M. 1970. Scientific aspects of the oil spill problem. Presented at NATO Conference, Brussels, 6 Nov. 1970, 21 p., Woods Hole Oceanogr. Inst., Woods Hole, Mass.

Olefinic hydrocarbons (paraffinlike but unsaturated and containing reactive carbon-carbon double bonds) are not generally found in crude oils but are plentiful in certain gasolines and other refined products. The fate of olefins in the marine environment is poorly understood, but this class of compounds may be quite reactive under certain conditions and may combine readily with hydrogen, oxygen, chlorine, sulfur, and other elements to produce toxic substances. Once incorporated into organisms, olefins may remain intact for surprisingly long times (Blumer, 1967). The full range of olefinic hydrocarbons probably interferes with the reception of chemical messengers, or odors, in the sea by certain marine organisms (Blumer, 1970, see footnote 3).

When crude oil is processed ("cracked"), olefins and other compounds for gasoline and fuel oils may be formed or separated. Fuel oils, commonly involved in spills, are rated from 1 to 6. Those rated 1 are the lightest, most volatile, and most toxic and have the greatest aromatic concentrations; those rated 6 are the least volatile, least soluble, and least toxic and are asphaltic (tarlike).

Hydrocarbons are not foreign to the marine environment; normal paraffins are synthesized by most, if not all, living organisms. Blumer, Guillard, and Chase (1971) characterized the natural hydrocarbon content of 22 species of phytoplankton and cited literature for zooplankton. There are certain characteristic differences, however, between hydrocarbons native to organisms and the hydrocarbons in petroleum, particularly in the relative distribution of the various hydrocarbons. Crude oils and certain petroleum products are complex mixtures that contain molecules of different sizes in ratios not found in any one species of organism. Certain specific paraffins, and some naphthenic and aromatic compounds, are rarely found in organisms not exposed to oil pollution. These characteristic differences have been the basis for several scientific papers (Blumer, Souza, and Sass, 1970; Ehrhardt, 1972; Clark and Finley, 1973; and others).

FACTORS INFLUENCING THE IMPACT OF OIL

The impact of oil on the marine environment is governed by several factors—physical, chemical, and biological—in addition to the inherent complexity of crude oil and refined products. The behavior, effects, and fate of an oil spill involve all of

these factors; and because they are interdependent, the reliability of our predictions concerning the impact of a spill is limited by our knowledge of the least understood variable.

Straughan (1972) noted our general inability to predict the environmental impact of a spill because of the complexity of the matter, and identified several factors that govern biological damage caused by a spill: 1) type of oil spilled, 2) dose of oil, 3) physiography of the area of the spill, 4) weather conditions at the time of the spill, 5) biota of the area, 6) season of the spill, 7) previous exposure of the area to oil, 8) exposure to other pollutants, and 9) treatment of the spill. Several of these factors are touched upon below.

Natural Physical Processes Affecting Oil in the Water Column

Once oil is spilled, it is dissipated by evaporation, dissolution, and mixing or dilution in the water column. The natural processes are speeded by wind action and by waves and currents that increase spreading and vertical mixing. Various fractions respond differently to these processes, and the weathered residue behaves differently than the material originally spilled. A contaminated bay may be flushed by freshets, tidal action, or longshore currents. Some oil sinks directly to the bottom, especially in fresh water, where some oil fractions have densities approaching that of fresh water, and in water with high sediment loads. Certain fractions may undergo autoxidation.

Conover (1971) reported that sedimentation of fecal-bound oil that had been ingested by zooplankton may have accounted for up to 20% of the spilled oil entering the water column at Chedabucto Bay, Nova Scotia. Oil can also be removed from the water column by absorption within organisms and accumulation within the food chain. Suspended sediments carried by runoff from a major flood entered the Santa Barbara Channel area immediately before and after the well blowout (Kolpack, 1971). Kolpack noted that adsorption of oil on the flocculated suspended particles followed by decomposition was a major factor in carrying much of the oil to the sea floor. Kinney et al. (1970) reported, however, that in Cook Inlet, Alaska, glacial silt from the inlet had no apparent effect on the emulsion properties or

the sinking of the type of crude oil found in that area.

Forrester (1971) noted the extensive distribution of oil particles stirred into the water by wave action after a bunker C oil spill in Chedabucto Bay. Oil particles were found to a depth of 80 m inside the bay and to depths of 45 m at a distance of 65 km outside the bay. Near-surface distribution of particles extended 250 km southwest along Nova Scotia in a band extending up to 25 km offshore. Berridge, Thew, and Loriston-Clark (1969) indicated that the stabilization of emulsions like those observed at Chedabucto Bay and elsewhere was caused by complex chemical components in the nonvolatile residues and not by bacterial activity, marine organisms, or suspended solid matter.

Environmental Differences

The fate and effects of oil spilled in the marine environment are difficult to generalize because several types of environments may be involved. Some extreme comparisons are tropics versus arctic, open ocean versus estuaries, and the differences between the intertidal and subtidal zones.

Within these environments are several diverse physical conditions such as temperature, salinity, oxygen, and nutrient concentrations, as well as biological differences such as species composition, diversity and density, and community metabolic rate. The prediction or assessment of pollution effects on the basis of observations extrapolated from one environment to another is seldom supported by adequate data. Unfortunately, however, few data on pollution effects exist for most areas and species, which has led to the use of information from areas that may be dissimilar in critical respects.

There are arguments as to which environment is the most stable and capable of withstanding attacks by additional pollution stresses. Copeland (1970), discussing the response of ecological systems to stress, suggested the principle that "...those systems already subjected to energy-requiring stresses are more likely to resist the changes than those (such as tropical systems) adapted to relatively constant environments." He concluded that estuarine ecosystems composed of organisms capable of wide adaptations and generalizations, such as north temperate systems, would be relatively unaffected by the same magnitude of disturbance that would drastically alter

a tropical system. Odum (1970) noted, however, that many estuarine species are living near the limit of their tolerance range and that any alteration in the environment, such as additional stresses caused by low levels of pollution, could exclude these animals permanently from the estuary.

All healthy balanced ecosystems are generally functioning at or near some critical tolerance limit. In an ecosystem with a variable environment, such as a north temperate estuary, responses to additional stress might not always be the same. For example, even though factors surrounding an oil pollution incident might be outwardly similar in most respects to another spill in a comparable area, the biological impacts may differ. The ability of the local community to absorb the additional stress will be influenced by the coincidence of seasonal variability of natural stresses, the differences in vulnerability of stages in an organism's life cycle, and many other dynamic features of the ecosystem.

Biological Differences

The effects of oil pollution on many different organisms in various habitats may vary from no effect to responses of avoidance and decreased activity, to nonadaptive responses of panic and physiological stress. What kills one species may have little or no effect on another. Affected organisms vary from single cells, to sedentary clams, to highly mobile predators, each of which has different behavioral and physiological interactions with the environment.

Just as different species are affected differently, so may individuals within a species be affected differently. In particular, different life stages such as eggs, hatched larvae, and newly molted individuals may have different sensitivity to the same level of pollution. Mironov (1968), for example, reported that prelarval stages of barnacle, *Balanus* sp., were 100 times more sensitive to oil pollution than the adult form. This contrasts with the relative lack of sensitivity to crude oil by pink salmon eggs and sac fry, which were 10 times more tolerant than older fry (Stanley D. Rice and Adam Moles, Auke Bay Fisheries Laboratory, National Marine Fisheries Service (NMFS), NOAA, Auke Bay, AK 99821, pers. commun.).

Renzoni (1973) conducted a series of experiments on the toxicity of several crude oils and petroleum products to the sperm, eggs, and larvae of the oysters *Crassostrea angulata* and *C. gigas* and the mussel *Mytilus galloprovincialis*. He

found a relatively high degree of tolerance by eggs and larvae but reported that the fertilizing capacity of sperm was markedly affected by similar exposures.

Biodegradation

Quantitative data describing the biodegradation of various components of crude oil, especially in arctic and subarctic areas, are limited.

ZoBell (1973a) briefly reviewed the current understanding of microbial degradation of oil, including interactions, limiting factors, problems, and perspectives. Ahearn (1973) stated that research on microbial utilization of hydrocarbons for treatment of oily pollutants in the environment, though more intensive in recent times, is still in an early stage of development. It is known that microorganisms can degrade much of a crude oil, particularly the less toxic paraffinic compounds. No single species can degrade all the compounds, but many different species together can metabolize a large number of the compounds, if not all. The rate of microbial degradation, which is principally aerobic, decreases with a decrease in temperature. Large quantities of oxygen are needed. It has been estimated, for instance, that complete oxidation of 1 gallon of crude oil would require all of the dissolved oxygen in 320,000 gallons of water. This comparison may be unrealistic because most oil is at the surface of water in contact with air and only the outer surfaces of oil can be attacked at any one time. It is reasonable to assume, however, that an oxygen-deficient environment may well occur under some oil slicks and in oil-contaminated sediments.

Glaeser and Vance (1971) studied the behavior of Prudhoe Bay crude oil in controlled spills in the Chukchi Sea but were not able to isolate any microorganisms which could degrade hydrocarbons at the ambient temperatures of the Arctic, although some emulsification of the crude oil was observed. However, ZoBell and Agosti (1972) collected oil-oxidizing bacteria near natural oil seeps from the Alaska North Slope and observed oxidation rates of mineral oil at -1°C and above. They noted that the solid surfaces of the ice crystals appeared to facilitate bacterial growth, because the rate at -1°C was substantial and near the 4°C rate.

The apparent contradiction between the studies is probably best explained by ZoBell's (1973b) continued observations with North Slope bacteria. He

found that the nine different crude oils were not degraded as rapidly as purified mineral oil. Glaeser and Vance's studies were with microorganisms from the surface water of the Chukchi where small numbers of bacteria may have been present. Furthermore, the observations of Stroughan (1971), who noted the apparent lack of biological damage by the Santa Barbara blowout, may apply here. She discussed the possibility that the fauna had an unusually high tolerance for oil, probably because of adaptation from chronic low-level oil exposures from local natural seepages. The observations of ZoBell and Agosti (1972) on the oxidation rates of oil at -1°C may be an example of similar adaptive response by the North Slope bacteria collected near natural seeps. These oxidation rates and other adaptive responses might not occur from organisms that have not been preacclimated to chronic low-level exposures of oil and may explain why Glaeser and Vance obtained reports of negligible oxidation rates at 0°C from microorganisms from surface water of the Chukchi Sea. Robertson et al. (1973) estimated hydrocarbon-oxidizing bacteria populations were in the order of 1/ml in Cook Inlet and Port Valdez, but less in the Arctic Ocean. Numbers decreased with salinity in Cook Inlet and with depth in Port Valdez.

ZoBell (1963) reported that oil is readily adsorbed by clay and silt and suggests that although adsorption of oil by solids renders the oil more susceptible to autotial and microbial oxidation, almost no bacterial decomposition occurs after burial in the bottom sediments, probably because the environment is anaerobic. Blumer and Sass (1972) found that some paraffinic hydrocarbons remained in bottom sediments 2 yr after the West Falmouth oil spill and aromatic hydrocarbons were prominent, which suggests that these more toxic compounds are utilized by bacteria to a minimum degree.

Oil in Sediments

The effect of oil in sediments is poorly understood, although several authors have quantitated oil concentrations and noted its persistence. Scurratt and Zitko (1972) observed little diminution of bunker C oil concentration from soft sediments 26 mo after the wreck of the tanker *Arrow*. The oil reached maximum concentrations in coarse sediments 1 yr after the spill, but the concentrations reduced thereafter. Chemical degradation can

occur but is normally restricted to the surface layer of the bottom penetrated by ultraviolet light. Blumer and Sass (1972) noted that "The preservation of hydrocarbons in marine sediments for geologically long time spans is one of the accepted key facts in current thought on petroleum formation." However, in spite of the stability of hydrocarbons in marine sediments, there are characteristic differences between the hydrocarbons in polluted and unpolluted areas. Tissier and Oudin (1973) found that hydrocarbons in polluted sediments differed from those of unpolluted sediments by having lower percentages of heavy components, by not having an odd carbon dominance in the n-alkanes, and by having polycyclic aromatic hydrocarbons with alkyl chains.

Oil residues were observed on sandy beaches by ZoBell (1963) and in marshes and in sediments of the deepest area (15.3 m) near the West Falmouth spill by Blumer, Sass, Souza, Sanders, Grassle, and Hampson (1970).⁴ About 2 wk after fuel oil was spilled at Resolute Bay, Northwest Territory, in August 1970, casual sampling revealed that oil penetrated into beach material to a depth of about 3 inches (7.6 cm) (Barber, 1971). Oil may be buried and stay intact for a considerable time, even at the higher temperature of the California coast (ZoBell, 1963). During laboratory experiments, Johnston (1970) determined oil decay rates in sand columns contaminated with various concentrations of oil. Ten percent of the oil was oxidized over a period of several months; the remaining 90% decayed much slower.

The West Falmouth spill provided a unique opportunity for a study of the immediate and long-term effects of an oil spill on an area where the previously existing environmental base was well known (Blumer, Sanders, Grassle, and Hampson, 1971). One effect of the oil was to reduce the cohesion of bottom sediments of tidal marshes and the estuary by killing the benthic plants and animals (Blumer, Sass, Souza, Sanders, Grassle, and Hampson, 1970, see footnote 4). The resulting erosion spread hydrocarbons to new areas, where the process was repeated. Because of the stability and persistence of the hydrocarbons in marine bottom sediments, Blumer, Souza, and Sass (1970) noted that hydrocarbons may be returned to the biosphere by organisms living and feeding in the sediments. This redistribution of hydrocarbons can be

⁴Blumer, M., J. Sass, G. Souza, H. Sanders, F. Grassle, and G. Hampson. 1970. The West Falmouth oil spill. Unpubl. manuscr. Woods Hole Oceanogr. Inst., Ref. No. 70-44, 32 p.

the source of a chronic pollution problem near that spill).

It is quite possible that normal functions of sediments will be disrupted when contaminated by oil. Changes in the sediments that are subtle and difficult to detect, such as decreased nutrient recycling and community metabolism, could result in the loss of significant contributions to the productivity and stability of an area. Although oil in sediments has been monitored and measured after several spills, other aspects of the oil-sediment relation have yet to be studied.

BIOLOGICAL EFFECTS OF OIL POLLUTION

Blumer (1970, see footnote 3) summarizes the potential damage to organisms from pollution by crude oil and oil fractions as follows:

1. Direct kill of organisms through coating and asphyxiation.

2. Direct kill through contact poisoning of organisms.

3. Direct kill through exposure to the water-soluble toxic components of oil at some distance in space and time from the accident.

4. Destruction of the generally more sensitive juvenile forms of organisms.

5. Destruction of the food sources of higher species.

6. Incorporation of sublethal amounts of oil and oil products into organisms (resulting in reduced resistance to infection and other stresses—the principal cause of death in birds surviving immediate exposure to oil).

7. Incorporation of carcinogenic and potentially mutagenic chemicals into marine organisms.

8. Low-level effects that may interrupt any of numerous events (such as prey location, predator avoidance, mate location or other sexual stimuli, and homing behavior) necessary for the propagation of marine species and for the survival of those species higher in the marine food web.

Some of the potential effects described by Blumer may be obvious, such as the direct deaths from acute exposures. Less obvious indirect deaths may occur from effects at either the individual or population level. Individual organisms subjected to sublethal exposures may undergo an "ecological death" if they are less capable of adjusting to and responding to natural changes (stresses) in their physical and biological environments. For example, postmolt Tanner (snow

crab, *Chionoecetes bairdi*, lost legs during short exposures to crude oil (Karinen and Rice, in press). Even though the crabs lived through the exposure, they probably could not have survived in the natural environment because some of them lost as many as seven legs, including both chelae. Moreover, crabs or other adversely but sublethally affected organisms would be more likely to be eliminated by natural selection.

Effects from chronic exposure may be adverse to a population over a period of time if exposed but normal-appearing adults have their ability to reproduce seriously impaired. This loss may be due to physiological changes such as reduced fecundity and delayed ovary development or to impaired behavioral mechanisms which could prevent mate location and identification or homing and timing of spawning. Although the effects at this level might not result in death of the adult, they could induce a trend of decreasing numbers that might eventually eliminate the population or race.

Hydrocarbons in the Marine Food Web

Blumer (1967, 1969) and Blumer, Guillard, and Chase (1971) studied the fate of organic compounds in the marine food web. They found that certain hydrocarbons, even high, unsaturated ones, are stable once they are incorporated into a particular marine organism and that they may pass through many members of the marine food web without alteration and may actually be concentrated in tissue. Most hydrocarbons are lipid soluble and thus may accumulate in food webs to the point where toxic levels are reached. This pathway is illustrated by the well-documented chlorinated hydrocarbon group of pesticides.

The entrance of oil-derived hydrocarbons into marine food webs has been observed several times at several trophic levels. Conover (1971) reported that 10% of the bunker C oil in the water column after the Chedabucto Bay spill was combined with zooplankton and that their feces contained up to 7% oil. Mironov (1968) also noted the ability of some zooplankters to accumulate hydrocarbons. The incorporation of hydrocarbons into the food web at these primary levels assures exposure at all higher trophic levels.

Blumer, Souza, and Sass (1970) and Ehrhardt (1972) reported pollution-derived hydrocarbons in shellfish. Uptake and retention of labeled hydrocarbons of several classes by a marine mussel,

Mytilus edulis, was noted by Lee, Sauerheber, and Benson (1972). Smith (1968) reported the presence of oil and benzene-ring compounds in the feces of limpets browsing on an oily deposit, and in top shells, *Monodonta*, and limpets, *Patella*, living on oiled rocks. He reported that analysis of the gut indicated "the proportion of oil in material ingested by these animals was estimated as about 20-30 percent in *Patella* and 5-50 percent in *Monodonta*."

Organisms at the highest trophic levels may be affected directly by the oil itself or indirectly by hydrocarbons that have reached them through the food web. Horn, Teal, and Backus (1970) found large amounts of tar in the stomachs of three saury, *Scomberesox saurus*, from a sample of ten in the Mediterranean Sea near Gibraltar. Although saury are generally considered to be carnivorous, the occurrence of tar and also of "vegetable debris" in one of the stomachs examined by Horn et al. (1970) suggests that the species is not a very discriminate feeder. Although all ingested oil was obviously not incorporated into the tissues (some oil was found in feces), such feeding behavior does describe a pathway for hydrocarbons to be directly taken up into the tissues of the organism. Thus, oil ingested, absorbed, and even adsorbed may enter the food chain when contaminated organisms are eaten.

Carcinogenicity

Some doubt may remain as to the direct carcinogenicity to man of crude oil and crude oil residues in marine organisms (Blumer, 1969), but evidence pointing toward this is accumulating (Blumer, 1970, see footnote 3; 1972). A literature search and evaluation conducted for the U.S. Coast Guard by Battelle Memorial Institute (1967) noted that shellfish, although alive, may have been unfit for consumption because of the carcinogenic hydrocarbon 3,4-benzopyrene in their bodies. Oysters that were heavily polluted and contaminated with ship fuel oil were reported to contain 3,4-benzopyrene. The Battelle review also reported that barnacles attached to creosoted poles contained the same carcinogenic hydrocarbon (3,4-benzopyrene). Sarcomas were elicited when extracts from the barnacles were injected into mice. The endemic occurrence of papillary tumors around the rectal opening of soft-shell clam, *Mya arenaria*, was reported, but the author (Battelle Memorial Institute, 1967) did not feel

these were due to oil pollution, even though the clams were taken from waters adjacent to areas highly polluted by ship fuel oil. Hyperplasia in reproductive cells of a bryozoan in response to coal tar derivatives was observed by Powell, Sayce, and Tufts (1970). They noted that similar abnormalities may also have occurred in coastal faunas exposed to spills such as the *Torrey Canyon* and the Santa Barbara blowout. However, most observations on these spills were concerned with gross mortality and may not have detected the sublethal effects.

ZoBell (1971) reported the natural synthesis and metabolism of carcinogenic hydrocarbons by several marine organisms. Thus, oil pollution is certainly not the only source for carcinogenic hydrocarbon introduction into marine food webs. Suess (1972) recognized that carcinogens were in seafoods but concluded that they would probably not be dangerous unless the foods contained an excess amount of polynuclear aromatic hydrocarbon carcinogens. Carcinogenesis from oil-contaminated marine organisms has not been proved, but Ehrhardt (1972) expressed a need for carcinogenic testing of hydrocarbon fractions extracted from marine organisms contaminated by exposure to oil.

Observed Toxic Effects

A study of the available information on potential toxic effects of oil pollution reveals more unknowns than proven conclusions. Only a decade ago, ZoBell (1963) reviewed the literature on the effects of oil on bacteria and higher organisms and concluded that oil pollution had no great adverse impact on fishery resources in general. He did point out, however, a few reports of toxic effects, tainting of flesh, and damage to vessels and fishing gear.

The quantity of literature on effects of oil spills has increased since the *Torrey Canyon* incident of 1967. Most of the recent work has depended on onsite visual surveys after occurrence of an oil spill rather than on experiments and detailed study. The surveys have been limited mostly to the effects of oil and of cleaning or dispersing agents on primarily adult intertidal organisms and populations. These observations on a restricted segment of the affected ecosystem include only a few of the factors that influence the total impact of oil. Wilson, Cowell, and Beynon (1973) noted that the absence of results from studies at the commu-

nity level make the interpretation, extrapolation, and use of many observations very difficult. Further, the differences between various crude oils and between the hundreds of petroleum products in their physical and biological effects must always be kept in mind. Comparative data generally are far too few to permit attaching any relative significance to production area or product formulation in this review.

Field Investigations

The utility of many "after-the-fact" studies is limited because of the lack of knowledge of pre-spill conditions. Data are often collected without proper controls for comparison, and knowledge of natural local fluctuations and species composition of animal populations is usually quite limited. For these reasons conclusions about the impact of a particular spill may vary.

Ehrsam (1972) reported substantial immediate kills of marine life from a fuel oil spill at Anacortes, Wash., and concluded that if larval and juvenile forms of certain organisms were killed, the full impact of the spill may not be known for some time. Katz (1972) observed intertidal transects of the same affected area and concluded that the effects were minor and long-term effects would be unlikely. Webber (1972) pointed out, however, that these after-the-fact studies observed only a small wedge of the total biota. Knowledge of subtidal and benthic organisms as well as larvae and juveniles was lacking.

Other large spills have been studied in greater detail and have contributed significantly to our understanding of the gross effects of oil. Yet, they have been unable to answer many important questions on the effect of pollutant hydrocarbons in the marine environment, and generalizations learned from one spill may not apply to another because each is different.

Field observations of behavior and effects of oil in Arctic ice environments are few. The U.S. Coast Guard investigations in the Arctic have primarily been directed toward gaining knowledge to improve cleanup methods (Glaeser and Vance, 1971; McMinn and Golden, 1973). Campbell and Martin (1973) discussed possible large-scale movements and persistence of oil spilled in the Beaufort Sea. They suggested that the surface waters of the Arctic Ocean and the winter waters of Chedabucto Bay, Nova Scotia, might be comparable, particularly with regard to the physical

behavior of oil. Chedabucto Bay is the site of the grounding of the tanker *Arrow* in February 1970 with 2.8 million gallons of bunker C oil aboard. Campbell and Martin (1973) found that highly stable oil-water emulsions formed to a depth of 50 m throughout Chedabucto Bay. They described conditions by which oil reaching the edge of the pack ice could be distributed under the ice.

Thomas (1973) also suggested that results of the studies at Chedabucto Bay might in some respects be applicable to spills in the Arctic. He observed remobilization of oil from beneath the weathered surface of deposits during the summers following the *Arrow* spill and the subsequent re-oiling of some intertidal areas, adding a chronic pollution aspect to the spill. Extensive mortalities of soft-shell clams and salt marsh cord grass, *Spartina alterniflora*, resulted where this occurred. In other areas, clams were visibly contaminated with oil and clam fishing was closed, at least through the summer of 1972 (Thomas, 1973).

When the *Torrey Canyon* broke up near the southwest coast of England in 1967, 15 million gallons of Kuwait crude oil with a high aromatic content were released. Efforts to cope with this first super disaster depended principally upon 2 million gallons of toxic dispersant, which probably caused more damage than the oil, most of which had weathered at sea for a week or more before reaching the shores. Many techniques for oil containment and control on the seas were attempted during the time oil leaked from the tanker; the fact that they all failed reveals the inadequacies of our technology and preparedness for such emergencies.

Extensive investigations of the West Falmouth spill by Blumer and his associates at Woods Hole provide one of the best documentaries of an oil spill. A total of 185,000 gallons of no. 2 fuel oil (41% aromatic content) were spilled in 1969 from a ruptured barge. Intertidal and subtidal benthic organisms of all phyla were killed during the first few days (Blumer and Sass, 1972). Blumer, Souza, and Sass (1970) showed that the uptake of fuel oil hydrocarbons by shellfish left them unfit for human consumption. Later, Blumer and Sass (1972) reported the continued persistence of fuel oil hydrocarbons in the sediments after 2 yr. Although there had been some degradation, the boiling range and composition of the hydrocarbon mixture was basically unchanged.

The 1969 Santa Barbara blowout released an estimated 5,000 barrels of crude oil per day ini-

tially (Foster, Charters, and Neushul, 1971), yet biological damage was not reported widespread and the area has started to recover. Foster, Neushul, and Zingmark (1971) observed that much of the damage to intertidal areas corresponded to sand movement, probably from storm damage. Cimberg, Mann, and Straughan (1973) concluded that the blowout had less effect on intertidal marine organisms than did sand movement and substrate stability. Straughan (1971), reporting on investigations at Santa Barbara, noted factors unique to that accident: 1) the long history of natural oil seepage in the Santa Barbara Channel and 2) the unusually heavy winter runoff at the time of the spill, which reduced salinities, increased sedimentation, and possibly increased pesticides in the channel. R. L. Kolpack (pers. commun. cited by Baker, Straughan, and Jessee (1971)) noted that Santa Barbara crude oil is relatively insoluble in water and contains a very low percentage of toxic aromatic compounds. Thus, information gathered on the effect of the Santa Barbara spill or any other is of limited utility in predicting the ecological effects of crude oil spills or of other oils in other areas.

Several studies have provided encouraging reports of varying degrees of recovery after some of the recent larger spills. Investigations about 1½ yr after the *Torrey Canyon* spill revealed that at least the affected shoreline areas were recolonizing and recovering, although recovery was not yet complete at that time (Spooner, 1969). The areas affected by the 1969 Santa Barbara blowout were recently reported to be recovering (Cimberg et al., 1973), as was a reef affected by bunker C oil spilled from a tanker collision in San Francisco Bay in January 1971 (Chan, 1973).

Too few of the controlled field investigations have been designed to bridge the gap between field surveys after spills and simultaneous laboratory experiments. Perkins (1970) exposed periwinkles and other intertidal organisms to the oil dispersant BP1002 in the laboratory and then released marked individuals in the natural environment. After recapture of the individuals exposed, he found that survival from doses as low as one three-thousandth of the 24 h LC_{50}^a was lower than among the recaptured controls. Crapp (1971a) conducted field experiments by applying crude oil and oil emulsifiers to the intertidal zone.

^a24 h LC_{50} equals that dose of toxicant that resulted in 50% survival after 24 h exposure.

Physical damage by the oil was observed, but toxicity damage was not great because the oil had previously been exposed to air; in contrast, the oil-emulsifier mixtures were toxic. Baker (1970) applied a crude oil to salt-marsh plots at different times of the year and monitored the effects on plants. Summer applications of oil severely affected annuals but not perennials.

Laboratory Studies

Experiments in the laboratory also do not provide all the answers about how an oil spill will affect a marine organism or its environment. Laboratory research has demonstrated the toxicity of various crude oils and petroleum products on several forms of marine life. Much of this research has focused on the planktonic life history stages of pelagic and benthic animals. Many of these planktonic larvae are phototactic at their earliest stages and concentrate in the surface layer of the sea. This community of the surface 5 cm, the neuston, is the first affected by most oil entering the water. Thus, many organisms are most sensitive to oil pollution at the time of their greatest likelihood of exposure.

Studies by Mironov (1968) on the development of fertilized eggs of the plaice, *Rhombus macoticus*, showed extreme sensitivity of the eggs to the influence of the oil products in seawater. He noted that injury to the eggs occurred at concentrations of 10^{-4} to 10^{-5} ml/liter (0.1 to 0.01 ppm). In these concentrations of oil products, 40 to 100% of the hatched prelarvae showed some signs of degeneration during development and perished. Mironov (1969a) also demonstrated that 0.001 ml of crude oil per liter was toxic to the eggs of anchovy, scorpionfish, and sea parrots from the Black Sea.

Newly set spat of *Elinius modestus*, an Australian barnacle introduced to Europe, were tolerant of 100 ppm crude oil but showed reduced cirral activity and retarded shell growth (Corner, Southward, and Southward, 1968). Adults of this species also showed reduced activity at 100 ppm (Corner et al., 1968).

Mironov (1969b) tested crude oil on several copepods and a cladoceran, and found that 0.001 ml/liter accelerated death in all forms and that 0.1 ml/liter caused death in less than 1 day. *Acartia* and *Calanus* died at 0.01 ml/liter oil in seawater in 72 to 96 h (Mironov, 1968). Larvae of crab and shrimp died at 1 ppm (Mironov, 1969c).

Little is known of the mechanisms of various

toxic effects. Damage to cell membranes and the cellular contents of planktonic larvae may occur. Goldacre (1968) demonstrated such cytological damage and death to the freshwater protozoan, *Amoeba proteus*, exposed to crude oil fractions. Brocksen and Bailey (1973) measured increased respiratory response of striped bass and chinook salmon to sublethal concentrations of benzene. The fish recovered to normal activity when they returned to noncontaminated water for several days. Rice and Short were unable to demonstrate changes in the enzyme activity of cholinesterase or Na-K stimulated ATPase in juvenile pink salmon, *Oncorhynchus gorbusha*, after in vivo and in vitro exposures to Prudhoe Bay crude oil (Stanley D. Rice and Jeffrey Short, Auke Bay Fisheries Laboratory, NMFS, NOAA, Auke Bay, AK 99821, pers. commun.). This is somewhat surprising because various hydrocarbon pesticides have been shown to affect both enzymes.

Cellular membranes of phytoplankton are also damaged by the penetration of hydrocarbon molecules: the cellular contents are extruded, and oil penetrates into the cell. Detergents administered in a concentrated solution also penetrate the plant cells and cause the dissolution of cellular membranes and the extrusion of cellular fluid (Ruivo, 1972). The effects of oils on plant respiration are variable, but an increase of respiration is frequently observed, probably because of an alteration of the mitochondria. This could result in an uncoupling of the oxidative phosphorylation enzymes from the electron transport enzymes, and the energy release would be lost as heat.

All marine animals ultimately depend on the photosynthetic activity of phytoplankton and algae for the production of biomass. Baker (1971), reviewing the literature, noted that weathered *Torrey Canyon* oil had no apparent effect on the photosynthetic activity of green algae. He did find, however, that green algae treated with fresh crude oil died and that photosynthesis in kelp, *Macrocystis* sp., was reduced when the kelp was exposed to various petroleum products. Kauss et al. (1973) determined the effects of crude oil on several species of freshwater algae in both field and laboratory experiments. In their field studies, response of the algae to a spill varied from suppression of growth to its stimulation. In their laboratory studies, they noted depressed photosynthetic rates in one algal species after it had been exposed to aqueous crude oil and other selected aromatics.

Growth of phytoplankton from axenic cultures and mixed cultures of natural populations was inhibited by water-soluble extracts from no. 2 fuel oil in a laboratory study by Nuzzi (1973). Mironov and Lauskaya (1968) demonstrated that marine phytoplankton vary several orders of magnitude in sensitivity to crude oils and kerosene in oil concentrations ranging from 0.1 to 1,090 ppm. Of the 20 species tested, a diatom, *Ditylum brightwellii*, was the most sensitive. The wide variation in susceptibility may account for the statements in other reviews of low toxicity of crude oils to phytoplankton (Føyn, 1965; Nelson-Smith, 1970) and supports the premise that biological response will differ among species.

Sublethal and Chronic Effects of Oil Pollution

While data are scarce in some of the areas previously discussed, information on the ecological effects of chronic sublethal oil pollution is essentially nonexistent. Observing these effects is difficult because they are not dramatic and may pass unnoticed by the casual observer. A full description would require observations extending over a long period of time.

Lewis (1972), commenting on approaches to the study of chronic pollution, contends "... that without a massive expansion of ecological and reproductive data by simultaneous multidisciplinary studies not only will we be unable to detect the significant long-term changes, but we will even remain unaware of the most suitable or important species and methods to build into a monitoring program."

A few studies concerning sublethal effects on organisms have appeared in the literature. Wells (1972) reported deaths of lobster larvae to exposures of 0.1 ml of Venezuelan crude oil per liter, while larvae exposed to 0.01 ml/liter had poor survival rates and were unable to molt to the fourth stage. Decreased limb (cirral) activity of marine larvae exposed to oil has been reported (Smith, 1968). Kuhnhold (1972), while observing toxic effects of crude oils to eggs of cod and to larvae of cod, plaice, and herring noted that the larvae exposed to oil-contaminated water were unable to avoid well-defined milky clouds of toxic oil dispersions. Blanton and Robinson (1973) observed damage to the gills of specimens of seven species of fish that had apparently been exposed to an oil spill off the Louisiana coast.

Crapp (1971b) observed that fucoid algae replaced barnacle and limpet populations near an outfall where the effluent contained about 20-25 ppm oil from treated ballast water of tankers unloading at Milford Haven. Although the relative oil content was low, the cumulative volume discharged was large (20,000 gallons of oil per year), a situation similar to that which may occur at Port Valdez, Alaska, when the trans-Alaska pipeline is completed.

Blumer (1972) discussed how low-level chronic effects of oil may damage marine organisms because of their dependence on natural organic chemical clues for a variety of functions. Salmon and other fishes utilize organic chemical clues in migrations; predators are attracted to prey by organic compounds at the parts-per-billion level (Whittle and Blumer, 1970); and other organisms may use chemical clues for predator avoidance, selection of habitat, and sex attraction. Blumer (1972) discussed the fears that oil pollution may interfere with these fundamental biological processes by masking or blocking, or by mimicking natural stimuli (resulting in false responses). He cited literature discussing the attraction of lobsters to kerosene and to purified hydrocarbon fractions derived from kerosene and noted that many dead lobsters were washed ashore after the West Falmouth spill. Blumer's fears about interference with chemoreception are further substantiated by the observations of Takahashi and Kittredge (1973) on crab behavior. Crabs, *Pachygrapsus crassipes*, exposed to water-soluble extracts of crude oil failed to exhibit feeding behavior or mating behavior responses when given appropriate chemical stimuli. Inhibition of chemoreception of some motile marine bacteria by a crude oil and several other hydrocarbons has been demonstrated by Walsh and Mitchell (1973).

Rice (1973) performed laboratory tests of avoidance of pink salmon fry to Prudhoe Bay crude oil and observed avoidance of oil at concentrations as low as 1.6 mg/liter. He concluded that salmon fry had the capability of detecting sublethal concentrations of oil and that they might avoid areas contaminated with sublethal levels of oil, which would result in confused and nonadaptive migratory behavior. The effect of chronic low-level pollution in areas such as Port Valdez, the terminus of the trans-Alaska oil pipeline, could be as severe as the total loss of all salmon runs in the local area because of altered behavioral responses to sublethal oil pollution.

CONCLUSIONS

Although crude oil generally should be considered toxic to marine organisms and harmful to their environment, most ecosystems can tolerate some pollution because oil can be dissipated or removed by processes like evaporation, autoxidation, dilution, and biodegradation. However, each organism and environment has a limit to how much oil can be absorbed and metabolized. Catastrophic spills are obviously pollution at a level that ecosystems cannot tolerate without damage. However, if the spills are not continued, the oil will slowly be removed and recovery of the area, at least to some degree, will likely occur. There is some evidence for recovery of some affected individuals.

Assessments of the impact of oil pollution cannot depend solely on evaluation of immediate kills of organisms from acute exposures. Chronic low-level oil pollution can cause subtle changes in organisms and is potentially more dangerous to the ecosystem than dramatic catastrophic spills. For this reason, the effects of chronic pollution warrant intensive study so that they will not be underestimated. The cumulative impact of "ecological death" of individuals which have impaired functions may be quite significant, yet difficult to assess because the death is not tied directly to an acute oil exposure. Equally as dangerous is the potential impact on populations where reproductive processes, adversely affected through physiological or behavioral mechanisms, result in fewer progeny. Chronic pollution may eliminate a species from an area entirely, and once eliminated that species may remain suppressed and may not repopulate the area because of continuing pollution or because its niche has been filled by a more tolerant, possibly less desirable, species.

The adverse effects of oil on animal populations has been of wide concern when stocks of special interest, such as those providing the basis of a sport or commercial fishery, have been involved. It should be remembered that changes in populations of lesser apparent significance will also cause changes in the community because each species population interacts with and is dependent on the rest of the community.

The foregoing review of information does little to simplify or ease the problems of policy makers concerned with marine production and transportation of oil and petroleum products. The weight of

the evidence leaves little doubt that oil poses a serious hazard to living marine resources, that spills and chronic pollution have happened and will continue to occur, and that the interests of the marine environment are best preserved if marine transportation of oil and petroleum products is minimized. The continuing need for new sources and increased amounts of energy, however, limits many of the conservative and prudent alternatives to these hazards. Until research has provided conclusive data, policy makers must continue to rely on these interpretative judgments for much of their guidance in making decisions that can profoundly affect the well being of marine ecosystems.

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HB 712

March 30, 1978

ALASKA LEGISLATION - 1978
S.B. 557
OIL SPILL PENALTIES

This bill, which amends H.B. 137, Chapter 129 of the 1977 Session, should be further amended as follows:

1. Section 46.03.758(a)(2) is amended to read:

"the exact nature and extent of oil pollution can be neither documented with certainty nor precisely quantified on a spill-by-spill basis; however, in light of the magnitude of harm which may be caused by oil discharges, and the vital importance of commercial, sport and subsistence fishing, tourism, and Alaska's natural abundance and beauty to the economic future of the state, and its quality of life, it is the judgment of the legislature that substantial civil penalties should be imposed for the discharge of oil, in order to provide a meaningful incentive for the safe handling of oil and to insure that the public does not bear substantial losses from oil pollution for which, because of its subtle, long-term or unquantifiable nature, compensation would not otherwise be received; and however, it is not the intent of this section to impose a civil penalty for discharge of oil where there is no demonstrable damage to the environment; and"

This amendment is the most important of the four. It deletes the reference to unquantifiable damage, a thoroughly illogical concept which no one has ever been able to explain, and it adds language to require a demonstration that environmental damage has occurred before penalties under this law can be imposed.

The existing law, which does not require evidence of environmental damage before imposing penalties, must be considered punitive. Yet, subsection (a)(3) states this should not be the case.* Thus, this amendment would render the penalty remedial rather than punitive.

2. Section 46.03.758(b)(1) is amended to read:

"Subject to subsection (a)(2) and to ~~(3)~~ (2) of this subsection, the penalties for the following categories of receiving environments may not exceed"

Making the penalties subject to subsection (a)(2) is a precaution to ensure that penalties will not be imposed unless it is demonstrated that environmental damage occurred.

*46.03.758 (a)(3) "in order to provide an incentive which is effective, but not punitive."

The change from (3) to (2) is a technical change. There is no (3) in subsection (b).

3. Section 46.03.758(d) is amended to read:

"The schedule shall vary according to the toxicity, degradability and dispersal characteristics of the oil. The schedule shall also vary according to the sensitivity and productivity of the receiving environment. And, the schedule shall take into account seasonal changes. Variations under this subsection may be by subcategories of receiving environments, specific receiving environments, or both. The maximum penalties established in (b) of this section shall apply to discharges in the most sensitive and productive of receiving environments within each category of receiving environment, and the penalty shall decrease for less productive or less sensitive receiving environments."

This is consistent with past arguments that seasonality is an important factor in determining potential environmental damage. Also, it reinforces our first amendment. "Less" is added before "sensitive" in the interest of good construction.

4. Section 46.03.758(g) is amended to read:

"Except as provided in (f) and (j) of this section, the entire penalty specified in the regulations shall be imposed, except that a person who discharges oil into a receiving environment may demonstrate, by a preponderance of evidence, that mitigating circumstances relating to the effects of the discharge would make imposition of the full penalty inappropriate. In determining whether mitigating circumstances exist, the court shall recognize that scientific knowledge pertaining to oil spills is very limited and if there is insufficient knowledge either to predict a base case or to show mitigating circumstances varying from that base case, the administratively established schedule of penalties shall apply. Only when no such mitigating circumstances exist shall the schedule of full penalties apply."

The language deleted describes the body of scientific knowledge pertaining to oil spills as "very limited." This is simply not true. The scientific community, academicians, government, and industry have produced volumes of data on the fate and effects of oil spills (an abbreviated bibliography is attached), and their work is continuing. Therefore, the deleted sentence cannot be justified.

Alvin

Sponsor Analysis of HB 137

1. Need for the legislation

Oil pollution can and does cause a wide range of harm to renewable resources, and the environment in general. Many types of harm, which can have devastating long-term effects on important marine resources, cannot be quantified. Included within this category are mortality to fish and shellfish larvae, food chain contamination, carcinogenic effects, and chronic toxicity. (See "Effects of Oil on Marine Ecosystems: A Review for Administrators and Policy Makers," Evans and Rice (1974), copy of which is attached to this analysis.)

Much of the damage caused by oil pollution can at least partially be seen -- oil-smothered beaches and intertidal areas, dead birds, marine mammals and the like. Yet even here, under existing state and federal law, the public, and the industries which depend upon the productivity of the sea, must bear these damages without compensation. As impressive as oil spill liability statutes may seem on their face, the fact of the matter is that traditional concepts of burden of proof will normally pose an insurmountable barrier to effective redress for the great bulk of harm caused by oil pollution.

In Alaska, the primary remedy for recovering damages for oil pollution is AS 46.03.822, which provides a strict liability remedy for those damaged by oil discharges. Yet it must be remembered that, to receive any compensation under the statute, the plaintiff must prove actual loss, the precise economic value of the loss, and causation. It is naive to believe that in any but the most basic cases can that burden be met. For example, let us suppose that, two years after a major oil spill, the salmon run in the area declines by 30%. Is that decline attributable to the oil spill? Natural fluctuation? Hard winters? Overfishing? A little of each? An even greater problem, of course, arises with regard to the subsistence user, who must prove not only causation, but must also meet his burden of establishing an exact dollar figure for lost subsistence opportunities. As the Court of Appeals for the Ninth Circuit warned in Union Oil Co. v. Oppen, 501 F. 2nd 559 (1974), after holding that commercial fishermen could sue for lost profits due to oil pollution:

"To [recover any damages] it must be shown [by the fishermen] that the oil spill did in fact diminish aquatic life, and that this diminution reduced the profits the plaintiffs would have realized from their commercial fishing in the absence of the spill. The reduction of profits must be established with certainty and must not be remote, speculative or conjectural. . . . These are not small burdens, nor can they be eased by our abhorrence of massive oil spills. All that we do here is to permit plaintiffs to attempt to prove their case. . ." Id at 570.

The problem, of course, reaches unmanageable proportions when the state, on behalf of the public, seeks redress for harm caused to its natural resources. Here we deal not with lost profits, but with the diminution of the quality of the state -- lost recreational opportunities, decreased tourism, loss of heritage and the like.

The State of Maine considered it a major victory when a federal district court ruled in Maine v. M/V Tamano, 357 F. Supp. 1097 (D.Me. 1973) that the state could sue for these kinds of damages. The difficulty, of course, came afterwards -- in translating the harm from the spill into a dollar figure. Maine was fortunate in this instance. The primary visible harm was the direct coating of commercially valuable clams. The state actually conducted an on-site survey, and counted the dead clams. Yet even here, the inability of the state to put a dollar figure on the value of "in place" clams forced an out-of-court settlement of \$750,000.

In most cases, however, the burden on the state will be far more formidable. While dead clams can be more or less effectively surveyed, most species cannot. And what of the indirect damages to commercial species -- such as food chain contamination? And the problem of valuation of non-commercial species? What is the "in place" value of a trumpeter swan, or a whale?

Alaska's answer to this problem has been the modest civil assessment provision of AS 46.03.760, which provides for an assessment of not less than \$500, nor more than \$100,000 for oil spills, based on the toxicity of the substance discharged, and the receiving environment. The statute is intended to provide "liquidated damages" for harm caused by oil pollution.

The most glaring problem with the statute is the \$100,000 upper limit. In light of the fact that in many situations, the only recovery the state will make will be under sec. 760, the \$100,000 figure seems ludicrous when applied to a Torrey Canyon or Santa Barbara.

The \$100,000 figure is unreasonably low for another reason. Any civil penalty scheme should be regulatory as well as remedial. The penalties should be sufficiently high to constitute a meaningful stimulus to comply with the laws which they enforce. A potential maximum exposure of \$100,000 no doubt fails to even register in the cost-benefit analysis of oil handling operations. An illustrative anecdote might be provided here. Last summer, Exxon Corporation willingly paid the U. S. Environmental Protection Agency \$100,000 in civil penalties for a 200-400 gallon discharge of oil into the Beaufort Sea, to protect a "tight hole" from potential exposure in litigation. With regard to oil spills, sec. 760 fails its regulatory, as well as its compensatory role.

Sec. 760 is primarily addressed to continuing acts of pollution -- by depriving the discharger of the economic incentive of continuing to violate state law. It is not an appropriate enforcement mechanism for single-act oil spills. Even with the presence of the toxicity and receiving environment criteria, it is still exceptionally difficult to apply to any given oil spill. A person simply has no means of assessing his potential liability under the law. A trial to set the amount of the assessment remains largely potluck, and, as a result, there have been none. Cases under the law languish for years, while the private and public bar argue across the negotiating table as to whether the spill in question is a \$500, or \$50,000 discharge. There are more efficient, as well as more effective ways of protecting the public interest.

2. Major issues regarding HB 137

A. What is a civil penalty?

Although called a "penalty," the primary purpose of a civil penalty is to compensate for public harm, and to effectively encourage the regulated industry to comply with the laws which it enforces. See U.S. v. Mar.-Tee Contractors, _____ F. Supp. _____, 8 ERC 1925 (D.N.J. 1976). In both intent and operation, HB 137 is remedial and regulatory, rather than punitive in nature.

B. Why dollars per gallon?

Civil penalty schemes are usually open-ended -- like current sec. 760 -- with a broad range between the lower and upper limits. This inevitably leads to the problems the state has faced with sec. 760.

The consequences of oil pollution should be definitively and unequivocally established from the outset. The dollars per gallon approach objectively grades the penalty according to the degree of public harm caused. The approach also furthers the regulatory purposes of the bill, by increasing the incentives for safe operation according to the amount of oil handled.

C. Why should an administrative agency establish the dollars per gallon schedule?

In the bill, the legislature establishes both the amount of the penalty, and the criteria for its application. What remains is a ranking of the relative sensitivity of receiving environments, and the relative toxicity of various types of oil. That is a technical effort which is properly vested in an administrative agency.

It might be argued that the establishment of a particular dollars per gallon figure should be established on a spill-by-spill basis by the courts. That, of course, would lead to precisely the same problem faced in utilizing current sec. 760. Moreover, courts are simply not equipped to make these kinds of relative judgments.

D. Why \$5 to \$50 per gallon?

We believe that \$5-\$50 per gallon is reasonably related to the gravity of the harm caused by oil pollution. In arriving at the figure, we of course recognize that it is impossible to quantify the environmental harm caused by oil pollution. But this bill is not an attempt to rigidly pre-establish damages. It is a penalty scheme in lieu of damages for which the public will never recover. It is also, of course, intended to serve as a meaningful incentive for safe operations.

On this point, it should be noted that a significantly lower range would result in unreasonably low assessments at the low end of the scale. For example, at a maximum range of \$1 per gallon, the discharge of 100 gallons of oil to a salmon stream would result in a penalty of \$100 -- a sum hardly commensurate with the gravity of that kind of incident.

E. Shouldn't there be an upper limit on liability?

Under HB 137, an upper limit does exist -- \$50.00 per gallon. The total limit of liability thus depends upon the total amount of oil spilled.

To place a dollar limit on the total amount that may be assessed would represent a policy judgment that, at a given point, the harm caused by oil pollution should be

borne by the public, rather than the discharger. The administration is not prepared to make that judgment. In this regard, it should be noted that Alaska currently imposes unlimited strict liability for actual damages -- as do Maine and Florida. See AS 46.03.822.

Admittedly, the recoveries which might be had under HB 137 are conceptually very high. So, it should be added, is the level of impact which an oil spill catastrophe can cause, as well as the figures which enter into industry's calculations in determining what level of safety is economically justifiable.

F. Why is it necessary to establish vicarious liability?

HB 137 does extend liability for the penalty to certain persons who are in a position to control the integrity of operations from which the discharge occurred. For example, oil terminals are made vicariously liable for spills caused by vessels which load at their facilities. Similarly, the lessee of an offshore platform is made vicariously liable for discharges caused from his platform, even though the actual cause of the discharge may be due to the acts of an independent contractor.

Focusing on the question of vicarious liability for terminals, it should be stressed that the owners of the transported oil are the ones who choose the carriers which

handle their products. Because of the terminal's ability to insure that only the safest vessels, and most experienced crews enter Alaska waters, it simply makes sense, as a matter of equity, to hold the terminal responsible for the vessels' actions, while they are in state waters.

It is this philosophy which motivated §204 of the Trans-Alaska Pipeline Authorization Act (43 USC §1653(c)) which makes the owners of the TAPS oil, through the Trans-Alaska Pipeline Liability Fund, vicariously liable for damages (above \$14 million) caused by oil spills from vessels which service the terminal. It is this same philosophy which led the State of Maine, in its Oil Discharge Prevention and Pollution Control Act of 1970, to make oil terminals vicariously liable for oil spills caused by "vessels destined for the licensee's facilities." 38 M.R.S.A. §552. This vicarious liability provision was upheld by the Maine Supreme Court in Portland Pipe Line Corporation vs. Environmental Improvement Commission, 307 A 2nd 1 (1973).

There are very real practical reasons for creating vicarious liability, aside from the amount of control which the terminal exercises over the quality of the vessel and its crew. First, the "independent contractor" defense -- which may be available if vicarious liability were not created -- has generally caused severe problems in oil spill

enforcement. For example, the dogged insistence of Alyeska Pipeline Service Company that it is not responsible for oil spills caused by independent contractors engaged in Trans-Alaska Pipeline work has frustrated state enforcement efforts in that regard.

Litigation against the owner of the vessel will often be fruitless. The vessel's owner or charterer will often be protected under the federal Limited Liability Act (46 USC §183, 186), which provides that the liability for damages of the vessel's owner or bareboat charterer as a result of a maritime incident will often be limited to the value of the vessel and freight on board after the incident. After a tanker grounding, of course, that value will be rather small. Of course, since HB 137 imposes liability for penalties, and not damages, it will be the state's position that the Limited Liability Act is inapplicable. It is, however, far from certain how the federal courts will treat this hybrid regulatory approach vis-a-vis that act.

The Torrey Canyon disaster provides a tragi-comic example of precisely this problem. On March 18, 1967, the Italian master of this Liberian registered flagship ran aground off the coast of England, spilling, over a period of days, most of her 119,000 tons of crude oil. On March 28, the vessel was sunk by RAF bombers.

The Torrey Canyon was owned by Barracuda Tanker Corp., a subsidiary of Union Oil Co. of California. Barracuda owned the boat pursuant to a sale-lease back arrangement with Union.

Barracuda immediately sued to limit its liability. The federal court obliged, limiting Barracuda's liability to \$50 -- the value of one lifeboat which survived the bombing. See In Re Barracuda Tanker Corp., 281 F. Supp. 228, 232 (S.D.N.Y. 1968).

The frustrated British and French governments finally resorted to a remedy unavailable to Alaska -- they seized the Torrey Canyon's sister ships in Singapore and Rotterdam and, in a very real sense, held them for ransom. Barracuda's insurers then paid the governments 3 million pounds sterling -- about one-sixth of the clean-up costs incurred as a result of the spill.

Vicarious liability is nothing new to hazardous undertakings -- particularly the handling of oil. Both as a matter of equity, and practical necessity, we believe it is imperative that those who induce, and profit from oil handling activities, and who in fact have the ability to control the integrity of those activities if they so desire, be impressed with a non-delegable duty to see that those operations are conducted in as safe as possible a manner.

Finally, it should be stressed that those upon whom vicarious liability is imposed are not without recourse if the penalty is assessed against them. As the Maine Supreme Court stressed in Portland Pipe Line Corp., supra, we are dealing here with a "mutually beneficial relationship [i.e. between terminal and vessels, and owners and operators where] there is, in the relationship, adequate opportunity to locate, among the business associates, the primary liability."

G. Will federal admiralty law let us do this?

The short answer is yes. Following the United States Supreme Court decision in Askew v. American Waterways Operators, 411 US 325 (1973), which upheld Florida's strict liability oil spill statute, it was argued by some that certain implications in that opinion might limit the state's ability to impose strict liability for sanctions or damages beyond the limits established in the Federal Water Pollution Control Act -- i.e. \$14 million dollars for vessels. In upholding Maine's unlimited strict liability oil spill law, the Maine Supreme Court, in Portland Pipe Line Cement, supra, specifically rejected that argument, and Congress has since made it clear that the Maine Supreme Court correctly interpreted its intent with regards to the state's power to enter the domain of admiralty, to seek redress for oil pollution. For example, in establishing the Trans-Alaska Pipeline Liability Fund, Congress stated with regard to vessels:

"This subsection shall not be interpreted to preempt the field of strict liability or preclude any state from imposing additional requirements." 43 USC § 1653(c)(9).

To the same effect is the following provision in the federal law creating the Deep Water Port Liability fund:

"This section shall not be interpreted to preempt the field of liability or to preclude any state for imposing additional requirements or liability for discharges of oil from a deep water port or a vessel within a safety zone." P.L. 93-697, § 18(k)(1).

Similar provisions are contained in comprehensive oil spill liability legislation now pending before Congress. It is clear that states have a "wide scope" in enacting laws pertaining to admiralty matters, as long as the law does not concern a matter requiring uniformity, or impair the harmony of the admiralty system. See Romero v. International Terminal Operating Co., 358 U. S. 354; Askew, supra. Both the courts and Congress have made it clear that the states' power to legislate in admiralty with regard to oil pollution is particularly broad.

H. Where does the money go?

The administration made a conscious decision, in submitting HB 137, not to specify how the penalties received should be allocated. If the bill is left untouched in this

regard, penalties collected would, pursuant to AS 30.25-.220(b), be deposited in the coastal protection fund. We believe it would serve no purpose to over-load the coastal protection fund with the substantial penalties which may be collected under this bill.

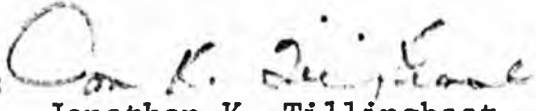
We would submit, for the legislature's consideration, that it would be more appropriate to return the penalties to the general fund, providing at the same time that the legislature may annually appropriate an amount equal to the penalties collected for the previous year for the purpose of financing renewable resource enhancement efforts in areas affected by oil pollution. These efforts, of course, could specifically include fish hatcheries. It is our belief that, in light of the extreme unlikelihood of effective recovery by fishermen for the full amount of damages actually caused to them as the result of oil pollution, that this mechanism would come as close as possible to, in fact, making the fishing industry whole after a catastrophe occurs. The department of law will be glad to assist the legislature in drafting a new subsection in this regard if that is the legislature's desire.

I. How does this bill affect private damages actions under AS 46.03.822?

This bill has no effect on the bringing of private damages actions under §822. It must be stressed that this section does not provide a damages remedy, but is rather a penalty law. It is true that the penalties are graded according to environmental and renewable resource impacts. However, again, the purpose of the bill is to provide a means of redress for harm caused by oil spills above those actual damages which might be proven in a given instance.

Respectfully submitted,

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